A Cross Sectional Study on the Type of Patients and Treatment Intervention in a Neurosurgery Department of a Tertiary Level Hospital in Bangladesh.

Submitted By

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Dissertation Submitted To

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In partial fulfillment for the requirements of the degree of Master of Pharmacy in Clinical Pharmacy and Molecular Pharmacology

Under the guidance of

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November, 2015

Certificate by the Supervisor

This is to certify that the research paper work on **"A Cross Sectional Study on The Type of Patients and Treatment Intervention in a Neurosurgery Department of a Tertiary Level Hospital in Bangladesh",** is a genuine research work done by **Rejoana Haque** under my guidance and supervision and this research paper is submitted to the Department of Pharmacy, East West University, in partial fulfillment of the requirements for degree of Master of Pharmacy. This is also to certify that all the resources of the information in this connection are duly acknowledged.

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Certificate by the Chairperson

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I, Rejoana Haque (ID # 2014-1-79-015), hereby declare that, the research entitled **"A Cross Sectional Study on The Type of Patients and Treatment Intervention In a Neurosurgery Department of a Tertiary Level Hospital in Bangladesh",** submitted by me to the Department of Pharmacy, East West University, Aftabnagar, Dhaka, Bangladesh in partial fulfillment for the requirements of the degree of Master of Pharmacy in Clinical Pharmacy and Molecular Pharmacology is an authentic record of original project work carried out by me during 2015 under the supervision and guidance of **Dr. Repon Kumer Saha**, Assistant Professor, Department of Pharmacy, East West University, Aftabnagar, Dhaka, Bangladesh.

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TABLES OF CONTENTS

Contents		Page
Abstract		1
Chapter One	Introduction	2
1.1	What is Hydrocephalus?	3
1.1.1	Causes	3
1.1.2	Pathology	3-4
1.1.3	Pathophysiology	4
1.1.4	Complications	5
1.1.5	Treatments & drugs	5
1.1.6	Toxicities	5
1.2	What Is Spinal Stenosis?	6
1.2.1	Causes	6
1.2.2	Pathology	7
1.2.3	Pathophysiology	7-8
1.2.4	Complications	8
1.2.5	Treatments & drugs	8-9
1.2.6	Toxicities	9
1.3	What Is Acromegaly?	9
1.3.1	Causes	9
1.3.2	Pathology	10
1.3.3	Pathophysiology	10
1.3.4	Complications	11
1.3.5	Treatments & drugs	11
1.3.6	Toxicities	12
1.4	What Is Cervical Myelopathy?	12
1.4.1	Causes	13
1.4.2	Pathology	13
1.4.3	Pathophysiology	13

1.4.4	**Complications**	**14**
1.4.5	**Treatments & drugs**	**14**
1.4.6	**Toxicities**	**14**
1.5	**What Is Brain Arteriovenous Malformation?**	**15**
1.5.1	**Causes**	**15**
1.5.2	**Pathology**	**16**
1.5.3	**Pathophysiology**	**16**
1.5.4	**Complications**	**17**
1.5.5	**Treatments & drugs**	**17**
1.5.6	**Toxicities**	**17**
1.6	**What Is Subdural Hematoma?**	**17**
1.6.1	**Causes**	**18**
1.6.2	**Pathology**	**18**
1.6.3	**Pathophysiology**	**19**
1.6.4	**Complications**	**19**
1.6.5	**Treatments & drugs**	**20**
1.6.6	**Toxicities**	**20**
1.7	**What Is Osteophytes?**	**20**
1.7.1	**Causes**	**21**
1.7.2	**Pathology**	**21**
1.7.3	**Pathophysiology**	**21-22**
1.7.4	**Complications**	**22**
1.7.5	**Treatments & drugs**	**22**
1.7.6	**Toxicities**	**22**
1.8	**What Is Spondylolisthesis?**	**23**
1.8.1	**Causes**	**23**
1.8.2	**Pathology**	**23-24**
1.8.3	**Pathophysiology**	**24**
1.8.4	**Complications**	**24**
1.8.5	**Treatments & drugs**	**25**

1.8.6	**Toxicities**	**25**
1.9	**What Is Cervical Spondylosis?**	**25**
1.9.1	**Causes**	**25-26**
1.9.2	**Pathology**	**26**
1.9.3	**Pathophysiology**	**26-27**
1.9.4	**Complications**	**27**
1.9.5	**Treatments & drugs**	**27**
1.9.6	**Toxicities**	**28**
1.10	**What Is Lumbar Spondylosis?**	**28**
1.10.1	**Causes**	**28**
1.10.2	**Pathology**	**28**
1.10.3	**Pathophysiology**	**29**
1.10.4	**Complications**	**29**
1.10.5	**Treatments & drugs**	**29**
1.10.6	**Toxicities**	**29**
1.11	**What Is Tuberculous Spondylitis**	**29**
1.11.1	**Causes**	**30**
1.11.2	**Pathology**	**30**
1.11.3	**Pathophysiology**	**30**
1.11.4	**Complications**	**31**
1.11.5	**Treatments & drugs**	**31**
1.11.6	**Toxicities**	**31**
1.12	**What Is Intracerebral Hemorrhage?**	**31**
1.12.1	**Causes**	**32**
1.12.2	**Pathology**	**32-33**
1.12.3	**Pathophysiology**	**33**
1.12.4	**Complications**	**33-34**
1.12.5	**Treatments & drugs**	**34**
1.12.6	**Toxicities**	**34**
1.13	**What Is Cerebral Aneurysm?**	**34**

1.13.1	**Causes**	**35**
1.13.2	**Pathology**	**35**
1.13.3	**Pathophysiology**	**35-36**
1.13.4	**Complications**	**36-37**
1.13.5	**Treatments & drugs**	**37**
1.13.6	**Toxicities**	**37**
1.14	**What Is Chiari Malformation?**	**37-38**
1.14.1	**Causes**	**38**
1.14.2	**Pathology**	**38**
1.14.3	**Pathophysiology**	**39**
1.14.4	**Complications**	**39**
1.14.5	**Treatments & drugs**	**40**
1.14.6	**Toxicities**	**40**
1.15	**What Is Multiple Sclerosis?**	**40**
1.15.1	**Causes**	**41**
1.15.2	**Pathology**	**41**
1.15.3	**Pathophysiology**	**41-42**
1.15.4	**Complications**	**42**
1.15.5	**Treatments & drugs**	**42**
1.15.6	**Toxicities**	**42**
1.16	**What Is Meningioma?**	**43**
1.16.1	**Causes**	**43**
1.16.2	**Pathology**	**44**
1.16.3	**Pathophysiology**	**44**
1.16.4	**Complications**	**45**
1.16.5	**Treatments & drugs**	**45**
1.16.6	**Toxicities**	**45**
1.17	**What Is Schwannoma?**	**46**
1.17.1	**Causes**	**46**
1.17.2	**Pathology**	**46-47**

1.17.3	**Pathophysiology**	**47-48**
1.17.4	**Complications**	**48**
1.17.5	**Treatments & drugs**	**48**
1.17.6	**Toxicities**	**49**
1.18	**What Is Glioma?**	**49**
1.18.1	**Causes**	**49**
1.18.2	**Pathology**	**50-**
1.18.3	**Pathophysiology**	**50-51**
1.18.4	**Complications**	**51**
1.18.5	**Treatments & drugs**	**51**
1.18.6	**Toxicities**	**52**
1.19	**What Is Medulloblastoma?**	**52**
1.19.1	**Causes**	**52**
1.19.2	**Pathology**	**52-53**
1.19.3	**Pathophysiology**	**53**
1.19.4	**Complications**	**53-54**
1.19.5	**Treatments & drugs**	**54**
1.19.6	**Toxicities**	**54**
1.20	**What Is Glioblastoma?**	**54**
1.20.1	**Causes**	**54**
1.20.2	**Pathology**	**55**
1.20.3	**Pathophysiology**	**55-56**
1.20.4	**Complications**	**56-57**
1.20.5	**Treatments & drugs**	**57**
1.20.6	**Toxicities**	**57**
1.21	**What Is Craniopharyngioma?**	**57-58**
1.21.1	**Causes**	**58**
1.21.2	**Pathology**	**58-59**
1.21.3	**Pathophysiology**	**59-60**
1.21.4	**Complications**	**60**

1.21.5	**Treatments & drugs**	**60**
1.21.6	**Toxicities**	**61**
1.22	**What Is Astrocytoma?**	**61**
1.22.1	**Causes**	**61**
1.22.2	**Pathology**	**61-62**
1.22.3	**Pathophysiology**	**62**
1.22.4	**Complications**	**63**
1.22.5	**Treatments & drugs**	**63**
1.22.6	**Toxicities**	**63**
1.23	**Literature review on Neurosurgery**	**63**
1.23.1	**Complication in spinal surgery: comparative survey of spine surgeons and patients who underwent spinal surgery**	**63-65**
1.23.2	**Leg weakness in a patient with lumbar stenosis & adrenal insufficiency**	**65-66**
1.23.3	**Non-traumatic spinal cord lesions: epidemiology & complications.**	**67-69**
1.23.4	**The influence of pituitary adenoma size on vision & visual outcomes after trans-sphenoidal adenectomy**	**69-71**
1.23.5	**Prevalence of diabetes mellitus in patients with acromegaly**	**71-72**
1.23.6	**Epidemiology & etiology of meningioma**	**72-75**
1.23.7	**Intracranial hemorrhage in setting of globlastoma with venous thromboembolism**	**75-76**
Chapter two	**Materials and methods**	**77-78**
Chapter three	**Results**	**79-107**
Chapter four	**Discussion**	**108-111**
	Conclusion	**112**
Chapter five	**References**	**113-125**
	Questionnaire	**126-127**

LIST OF TABLES

Table 1.1	**The etiology of non traumatic spinal cord lesion**	**68**
Table 1.2	**The frequency of common medical complications**	**68**
Table 3.1	**Prevalence of type of patients**	**81-82**
Table 3.2	**Medicine used in neurosurgery patients**	**86-100**
Table 3.3	**Associated disease in concomitant with major disease**	**102-106**

LIST OF FIGURES

Figure 1.1	**MRI of the neck showing multilevel compression of the spinal cord**	**12**
Figure 1.2	**The normal & abnormal blood vessels**	**15**
Figure 1.3	**X-ray showing a small bone spur is marked by the arrow**	**20**
Figure 1.4	**MRI images showing typical meningioma tumor**	**43**
Figure 1.5:	**The cross section of the ear**	**46**
Figure 1.6	**The pituitary gland**	**58**
Figure 3.1	**Prevalence of type of patients**	**83**

List of Abbreviations

NSAID	Nonsteroidal Anti-inflammatory drug
HGH	Human Growth Hormone
GH	Growth Hormone
IGF-1	Insulin-like Growth Factor-1
AVM	Arteriovenous Malformation
SDH	Subdural Haematoma
ICH	Intracerebral Hemorrhage
ICP	Intracerebral Pressure
CNS	Central Nervous System
MS	Multiple Sclerosis
WHO	World Health Organization
GBM	Glioblastoma
EGFR	Epidermal Growth Factor Receptor
CSF	Cerebrospinal Fluid
LSS	Lumbar Spinal Stenosis
AI	Adrenal Insufficiency
NTSCL	Non-traumatic Spinal Cord Lesion
ASIA	American Spinal Injury Association
VIS	Visual Impairment Score
ECMD	Early Carbohydrate Metabolism Disorder

Abstract

Background: Neurosurgery or neurological surgery is the medical specialty concerned with the diagnosis and treatment of conditions, illnesses and injuries involving the nervous system and its support structures. This includes various conditions involving the brain, the spinal cord, the actual nerves, the skull, the bones of the spine, spinal disks, as well as the blood vessels, ligaments and the protective coverings that offer support to the nervous tissues. Intervention by a neurosurgeon can be surgical but is most often non-surgical and is determined by the condition or injury as well as the general health of the person.

Objectives: The objectives of this study were to find out the type of patients and treatment intervention of the neurosurgery patients at neurosurgery department in a tertiary level hospital in Bangladesh (during 1st April, 2015 to 28th October, 2015).

Methodology: This cross sectional study was carried out among 190 patients at neurosurgery department in *Bangabandhu Sheikh Mujib Medical University* **(***BSMMU***).** Sample was selected by simple random sampling technique.

Results: Among the 190 patients, 22 types of diseases were found in neurosurgery department. The study shows that the diseases such as meningioma (16.85%), spinal stenosis (15.79%), acromegaly (11.06%), hydrocephalus (10%), cervical myelopathy (8.95%), and glioma (8.43%) were occurred in most of the patients. The most commonly used drugs were clonazepam, phenytoin, diazepam, amitriptyline, dexamethasone, naproxen, phenobarbital, flucloxacillin, paracetamol, pregabalin, and vitB1+vitB6+vitB12. Some diseases such as diabetes, hypertension, low blood pressure, hypercholesterolaemia, allergy, and fever were also associated with major diseases in patients.

Chapter One

Introduction

1.1 What Is Hydrocephalus?

Hydrocephalus is the buildup of fluid in the cavities deep within the brain. The excess fluid increases the size of the ventricles and puts pressure on the brain. Cerebrospinal fluid normally flows through the ventricles and bathes the brain and spinal column. But the pressure of too much cerebrospinal fluid associated with hydrocephalus can damage brain tissues and cause a large spectrum of impairments in brain function. Although hydrocephalus can occur at any age, it's more common among infants and older adults.[1]

1.1.1 Causes

Hydrocephalus is caused by an imbalance between how much cerebrospinal fluid is produced and how much is absorbed into the bloodstream. Cerebrospinal fluid is produced by tissues lining the ventricles of the brain. It flows through the ventricles by way of interconnecting channels and eventually flows into spaces around the brain and spinal column. It's absorbed primarily by blood vessels in tissues near the base of the brain.[2]

1.1.2 Pathology

- **Atrophy of white matter:** A primary destruction of axons, a secondary loss of myelin, and chronic astrogliosis are found. Neurons are selectively spared because of a more luxurious blood supply to the gray matter.

- **Multiple anomalies within ventricles:** Fibrosis of the choroid plexuses and stretching and denuding of the ependymal epithelium can be seen along with diverticula within the ventricles. The septum pellucidum can become fenestrated.

- **Anomalies in surrounding brain:** Spongy edema can develop in the surrounding brain, and there can be thinning and elongation of the interhemispheric commissures.[3]

1.1.3 Pathophysiology

Hydrocephalus most commonly results from impaired CSF absorption. Excessive functioning choroid plexus tissue in choroid plexus "hypertrophy" or papilloma remains the sole exception of CSF overproduction as the cause for hydrocephalus. Impairment of CSF absorption in communicating hydrocephalus may occur at the arachnoid villus, the lymphatic channels associated with cranial and spinal nerves, or the arachnoid membrane. The result is enlargement of all of the interior CSF spaces of the brain. In hydrocephalus, the following events are occur:

- **Obstruction of the basal cisterns:** There may exist an inability of CSF to reach the arachnoid villi with a delay in emptying from the ventricles.
- **Occlusion or atresia of the arachnoid villi:** Obstruction of the terminal CSF pathways results in a failure of the absorption of CSF into the venous sinuses. It is postulated that hydrocephalus may result from a congenital absence of these structures.
- **Increased sagittal sinus pressure:** Increased pressure in the venous sinuses, particularly the superior sagittal sinus, has significant effects on ICP and CSF absorption. The pressure in the intracranial compartment must rise to at least 5 mm Hg above the pressure within the sinuses, or no absorption of CSF will occur. This may be the mechanism of hydrocephalus in vein of Galen malformations, severe congenital heart disease with very high right atrial pressures, achondroplasia, and complex syndromic and nonsyndromic craniosynostosis.[4]

1.1.4 Complications

Long-term complications of hydrocephalus can vary widely and are often difficult to predict. The complications of hydrocephalus may include:

- Significant intellectual, developmental and physical disabilities
- Significant decline in memory or other thinking skills and
- Persistent symptoms after treatment of hydrocephalus.[5]

1.1.5 Treatments and drugs

One of two surgical treatments may be used to treat hydrocephalus.

Shunt is the most common treatment for hydrocephalus. It is the surgical insertion of a drainage system, called a shunt. It consists of a long, flexible tube with a valve that keeps fluid from the brain flowing in the right direction and at the proper rate.

Endoscopic third ventriculostomy is also a surgical procedure that can be used for some people.

Drug such as Acetazolamide (ACZ) and furosemide (FUR) are used to treat hydrocephalus.[6]

1.1.6 Toxicities

Toxicity of drugs can causes Liver impairment, increases the risk of kidney stones, loss of appetite, dry mouth, headache, nausea, vomiting, stomach pain and anemia.[6]

1.2 What Is Spinal Stenosis?

Spinal stenosis is the narrowing of spaces in the spine (backbone) which causes pressure on the spinal cord and nerves. About 75% of cases of spinal stenosis occur in the low back. In most cases, the narrowing of the spine associated with stenosis compresses the nerve root, which can cause pain along the back of the leg.[7]

1.2.1 Causes

There are many potential causes for spinal stenosis, including:

- **Aging:** With age, the body's ligaments (tough connective tissues between the bones in the spine) can thicken. Spurs (small growths) may develop on the bones and into the spinal canal. The cushioning disks between the vertebrae may begin to deteriorate. The facet joints (flat surfaces on each vertebra that form the spinal column) also may begin to break down. All of these factors can cause the spaces in the spine to narrow.
- **Arthritis:** Two forms of arthritis that may affect the spine are osteoarthritis and rheumatoid arthritis.
- **Heredity:** If the spinal canal is too small at birth, symptoms of spinal stenosis may show up in a relatively young person. Structural deformities of the involved vertebrae can cause narrowing of the spinal canal.
- **Instability of the spine, or spondylolisthesis:** When one vertebra slips forward on another that can narrow the spinal canal.
- **Trauma:** Accidents and injuries may either dislocate the spine and the spinal canal or cause burst fractures that produce fragments of bone that penetrate the canal.[7]

1.2.2 Pathology

The pathology of spinal stenosis may include:

- Hypertropy of the ligamentum flavum
- Hypertropy of the facet joints
- Osteophytes formations
- Disc herniations
- Synovial facet joint cysts and
- Vertebral displacements.[8]

1.2.3 Pathophysiology

Spinal stenosis pathophysiology refers to the origin and nature of narrowing within the spinal canal. There are two basic ways for spinal stenosis to develop. The most common is for age-related deterioration to give rise to anatomical abnormalities that restrict the space available in the spinal canal. The other way spinal stenosis occurs is through traumatic injury, such as a jarring hit in a car crash. Occasionally, an individual might actually have an asymptomatic case of spinal stenosis, which is then revealed when symptoms arise in the aftermath of a traumatic injury. The areas of the spine that are most susceptible to age-related spinal stenosis are the lumbar region and the cervical region. These two areas support the weight of the upper body and the head, respectively, and facilitate a wide range of stress-inducing motion. The pathophysiology of spinal stenosis often takes in to account factors such as:

- Body weight
- Age
- Family medical history
- Past injury history
- Current occupation and pastimes.[8]

1.2.4 Complications

Rarely, untreated cases of severe spinal stenosis may progress and cause permanent:

- Numbness
- Weakness
- Balance problems
- Incontinence and
- Paralysis[9]

1.2.5 Treatments and drugs

Spinal stenosis can be treated several ways. Treatment options include:

Changes in posture: People with spinal stenosis may find that flexing the spine by leaning forward while walking relieves their symptoms. Lying with the knees drawn up to the chest also can offer some relief. These positions enlarge the space available to the nerves and may make it easier for people with stenosis to walk longer distances.

Surgery: If other treatments do not ease the pain, surgery may be recommended to relieve the pressure on affected nerves.

Drugs: In some cases, the pressure on the nerves is caused by inflammatory swelling. Nonsteroidal anti-inflammatory medications (NSAIDS) such as aspirin or ibuprofen may help relieve symptoms.In some cases carbamazepine is also used.[9]

1.2.6 Toxicities

Toxicity of drugs can causes renal insufficiency, peptic ulcer disease, hepatic dysfunction, dry mouth, dry eyes, and constipation.[10]

1.3 What Is Acromegaly?

Acromegaly is a rare condition. It causes excess growth in the bones and soft tissues of the body. Children with the condition can grow to abnormal heights. They may also have an exaggerated bone structure that gives them the appearance of being a giant. Acromegaly mostly affects the arms, legs, and face.[11]

1.3.1 Causes

Human Growth Hormone (HGH) regulates growth and development. People with acromegaly have too much HGH. It makes their bones grow too long and get too thick. Because of this growth stimulation, their bones are much larger than other people's. HGH is made in the brain's pituitary gland. In people with acromegaly, the pituitary may be affected by a tumor that causes it to make too much HGH.[11]

1.3.2 Pathology

The pathology of acromegaly includes:

A marked diversity exists between the tumors which secrete growth hormone (GH) in excess, such as densely and sparsely granulated GH cell adenoma, the mixed GH prolactin cell adenoma and the mammosomatotrope adenoma. The latter two tumors produce GH and prolactin simultaneously. Densely granulated GH cell tumors may produce thyrotropin and alpha subunit as well. Somatotrope carcinomas are extremely rare. GH cell hyperplasia can also be associated with acromegaly in patients with extrapituitary GH-releasing hormone secreting tumors.[11]

1.3.3 Pathophysiology

Acromegaly is characterized by hypersecretion of growth hormone (GH), which is caused by the existence of a secreting pituitary tumor in more than 95% of acromegaly cases. Pituitary tumors are benign adenomas and can be classified according to size (microadenomas being less than 10 mm in diameter and macroadenomas being greater than 10 mm in diameter). [12][13] In rare instances, elevated GH levels are caused by extra pituitary disorders. In either situation, hypersecretion of GH in turn causes subsequent hepatic stimulation of insulin-like growth factor-1 (IGF-1).

The clinical features of acromegaly result from either:

- Pressure from the pituitary adenoma
- Elevated levels of GH and IGF-1, which work independently and in tandem to produce various signs and symptoms associated with acromegaly[12]

1.3.4 Complications

Complications may include:

- High blood pressure
- Cardiovascular disease, particularly enlargement of the heart (cardiomyopathy)
- Osteoarthritis
- Diabetes mellitus
- Precancerous growths (polyps) on the lining of colon
- Spinal cord compression
- Vision loss

Early treatment of acromegaly can prevent these complications from developing or becoming worse. If untreated, acromegaly and its complications can lead to premature death.[13]

1.3.5 Treatment and drugs

Treatment focuses on lowering production of GH, as well as reducing the negative effects of the tumor on the pituitary and surrounding tissues. Patients may need more than one type of treatment.

Surgery. Doctors can remove most pituitary tumors using a method called transsphenoidal surgery. In this procedure, surgeon works through the nose to extract the pituitary tumor. Removing the tumor can normalize GH production.

Drugs. Such as octreotide, pegvisomant, cabergoline are used.[14]

1.3.6 Toxicities

Toxicity of drugs can causes diarrhea, gas, nausea, headache, fatigue and liver function problems.[15]

1.4 What Is Cervical Myelopathy?

Cervical myelopathy refers to a constellation of symptoms and signs due to spinal cord compression in the neck. Because symptoms and signs in a patient with myelopathy can be subtle in early manifestations, the diagnosis may easily be missed or incorrectly attributed to "normal" aging process. However, because the natural history of cervical myelopathy is one of stepwise progression, early recognition and treatment is essential for optimal patient outcome before the onset of irreversible spinal cord damage.[16]

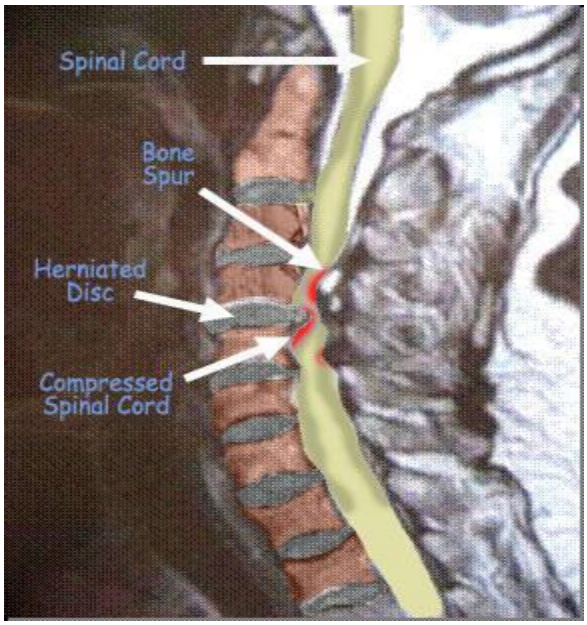

Figure 1.1: MRI of the neck showing multilevel compression of the spinal cord.[15]

1.4.1Causes

There are many causes of cervical myelopathy; anything that interrupts the normal flow of neural impulses through the spinal cord may cause a clinical myelopathy. Some of the causes are trauma, viral processes, inflammatory or autoimmune disorders, tumor, or degenerative processes including spondylosis and intervertebral disc herniation. The cervical spinal canal may become narrowed over time due to degenerative changes within the spine. Bone spurs, disc bulges, and thickened ligaments may develop and encroach on the spinal canal. In some cases, the space normally surrounding the spinal cord may be severely compromised and pressure on the spinal cord occurs.[16]

1.4.2 Pathology

The process of cervical myelopathy is due to compression of the spinal cord in the spinal canal. The physical act of compression may be a result of different conditions. In cervical myelopathy, degenerative changes of the cervical spine, known as cervical spondylotic myelopathy is occur. As the cervical spine degenerates, disc herniations or bulges along with arthritic bone spurs can all cause narrowing of the spinal canal, which in turn results in the compression of the spinal cord.[17]

1.4.3 Pathophysiology

The pathophysiology of cervical myelopathy involves static factors, which result in acquired or developmental stenosis of the cervical canal and dynamic factors, which involve repetitive injury to the cervical cord. These mechanical factors in turn result in direct injury to neurons and glia as well as a secondary cascade of events including ischemia, excitotoxicity, and apoptosis; a pathobiology similar to that occurring in traumatic spinal cord injury.[17]

1.4.4 Complications

Complications may include:

- Pseudoarthrosis
- Recurrent laryngeal nerve injury
- Hardware failure & migration
- Vertebral artery injury
- Esophageal injury
- Disphagia & alteration in speech[18]

1.4.5 Treatment and drugs

Surgery is the treatment of choice unless the patient is unwilling or unable to undergo surgery. The goal of surgical treatment for cervical myelopathy revolves around relieving the pressure and compression off of the spinal cord.[18]

Drugs such as ibuprofen, Corticosteroids, and Rituximab are used[18]

1.4.6 Toxicities

Toxicity of drugs can cause peptic ulcer disease, renal insufficiency, hepatic dysfunction, nausea and vomiting.[18]

1.5 What Is Brain Arteriovenous Malformaion?

A brain arteriovenous malformation (AVM) is a tangle of abnormal blood vessels connecting arteries and veins in the brain.The arteries are responsible for taking oxygen-rich blood from the heart to the brain. Veins carry the oxygen-depleted blood back to the lungs and heart. A brain AVM disrupts this vital process.An arteriovenous malformation can develop anywhere in the body but occurs most often in the brain or spine.[19]

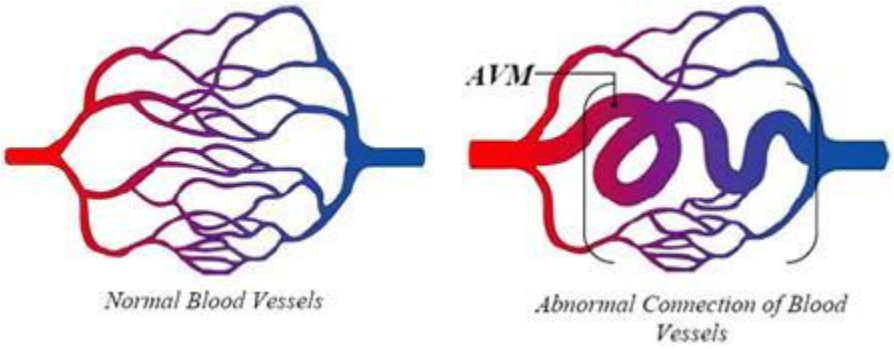

Figure 1.2: The normal and abnormal blood vessels.[19]

1.5.1 Causes

The cause of brain AVM is unknown, but researchers believe most brain AVMs emerge during fetal development. Normally, heart sends oxygen-rich blood to the brain through arteries. The arteries slow blood flow by passing it through a series of progressively smaller networks of blood vessels, ending with the smallest blood vessels (capillaries). The capillaries slowly deliver oxygen through their thin, porous walls to the surrounding brain tissue. The oxygen-depleted blood then passes into small blood vessels and then into larger veins that drain the blood from the brain, returning it to the heart and lungs to get more oxygen.[19]

1.5.2 Pathology

Arteriovenous malformations are divided into types according to the depth of involvement. One type is Deep form. It May be associated with arteriovenous shunting and soft tissue hypertrophy. Other type is superficial form. In both forms there are thick and thin-walled sized arteries and veins in close association with one another. Focally, some tumours resemble capillary and cavernous hemangioma. There may be focal thrombosis, secondary dystrophic calcification and mild inflammation. Serial sections are helpful in demonstrating continuities or shunts between arteries and veins. These lesions grow slowly and achieve only a small size.[20]

1.5.3 Pathophysiology

In a normal functioning human body, arteries carry blood away from the heart to the lungs or the rest of the body, where the blood passes through capillaries, and veins return the blood to heart. An AVM interferes with this process by forming a direct connection of the arteries and veins. AVMs can cause intense pain and lead to serious medical problems. Although AVMs are often associated with the brain and spinal cord, they can develop in any part of the body. AVMs are congenital lesions composed of a complex tangle of arteries and veins connected by one or more fistulae. The vascular conglomerate is called the nidus. The nidus has no capillary bed, and the feeding arteries drain directly to the draining veins. The arteries have a deficient muscularis layer. The draining veins often are dilated owing to the high velocity of blood flow through the fistulae. AVM is maily caused by forming an abnormal blood vessel that connects arteries and veins in the brain. How the abnormal vessels appear or exactly when the process begins is unknown.[21]

1.5.4 Complications

Complications of a brain AVM include:

- Bleeding in the brain
- Reduced oxygen to brain tissue
- Thin or weak blood vessels
- Brain damage[22]

1.5.5 Treatments and drugs

Surgery. If an AVM has bled and or is in an area that can be easily operated upon, then surgical removal may be recommended.

Stereotactic radiosurgery. An AVM that's not too large, but is in an area that's difficult to reach by regular surgery, may be treated with stereotactic radiosurgery.

Drugs such as phenytoin, carbamazepine, and valproic acid may be used.[22]

1.5.6 Toxicities

Toxicity of drugs can causes hypotension, cardiac arrhythmias, bradycardia, heart block, Hepatic injury and serious dermatological reaction.[22]

1.6 What Is Subdural Haematoma?

A subdural haematoma (SDH) is a collection of clotting blood that forms in the subdural space. A simple SDH is when there is no associated parenchymal injury.A complicated SDH is when there is associated underlying parenchymal injury, such as contusion.[23]

1.6.1 Causes

Subdural hematoma is usually caused by a head injury, such as from a fall, motor vehicle collision, or an assault. The sudden blow to the head tears blood vessels that run along the surface of the brain. This is referred to as an acute subdural hematoma. People with a bleeding disorder and people who take blood thinners are more likely to develop a subdural hematoma. A relatively minor head injury can cause subdural hematoma in people with a bleeding tendency. In a chronic subdural hematoma, small veins on the outer surface of the brain may tear, causing bleeding in the subdural space.[23]

1.6.2 Pathology

CSH is a collection of encapsulated blood, situated below the dura, mainly characterized by the presence of a membrane composed of two layers, an outer and an inner layer. The outer layer undergoes a process which leads to a meningeal reaction due to hemorrhage, which involved blood vessels, smooth muscle cells, eosinophils, erythrocytes, and the collagen fibers subsequently it continuous with neovascularization in large sinusoids with fragile thin walls, with spaces between which passage of erythrocytes and blood plasma is allowed. So it behaves as a semipermeable structure that allows the entry of liquid, favoring the increase of the volume of hematoma. It has been determined that the two membranes surrounding the hematoma are originated between the first and the fourth week of the first collection of blood. This is followed by neocapilar growth and liquefaction of the hematoma associated with enzymatic fibrinolysis. Furthermore it has been established that in the hematoma outer membrane occur local fibrinolysis, with low levels of fibrinogen and plasminogen. Degradation products of fibrin which ultimately inhibit the hemostatic cascade are also increased.[24]

1.6.3 Pathophysiology

An acute SDH is usually caused by either:

- Tearing of bridging veins from the cortex to one of the draining venous sinuses, typically occurring when bridging veins are sheared during rapid acceleration-deceleration of the head.
- Bleeding from a damaged cortical artery.

Blunt head trauma is the usual mechanism of injury but spontaneous SDH can arise as a consequence of clotting disorder, arteriovenous malformations or aneurysms or other conditions. In the subacute phase the collection of clotted blood liquifies. In the chronic phase it becomes a collection of serous fluid in the subdural space.[24]

1.6.4 Complications

Complications may include:

- Death due to cerebellar herniation.
- Raised intracranial pressure.
- Cerebral oedema.
- Recurrent haematoma formation during recovery.
- Seizures.
- Wound infection, subdural empyema, meningitis.
- Permanent neurological or cognitive deficit due to pressure effects on the brain.
- Coma or persistent vegetative state.[25]

1.6.5 Treatment and drugs

The goal of treatment is to control symptoms and reduce or prevent permanent damage to the brain.

Surgery may be needed. This may include drilling small holes in the skull to relieve pressure and allow blood and fluids to be drained.

Drugs such as carbamazepine, lamotrigine or levetiracetam may be used to control or prevent seizures.[26]

1.6.6 Toxicities

Toxicity of drugs can causes blurred vision, nausea, vomiting, constipation, dizziness, and ataxia.[27]

1.7 What Is Osteophytes?

Osteophytes or bone spurs are bony projections that develop along the edges of bones. Bone spurs often form where bones meet each otherin the joints. They can also form on the bones of the spine. Most bone spurs cause no symptoms and may go undetected for years.[28]

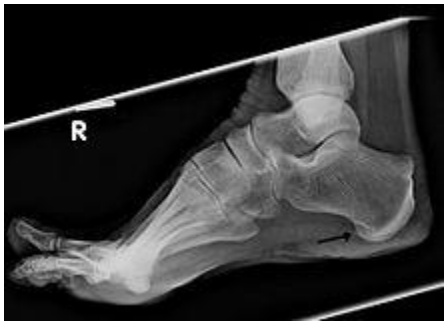

Figure 1.3: X-ray showing a small bone spur as marked by the arrow[27]

1.7.1 Causes

Joint damage from osteoarthritis is the most common cause of bone spurs. As osteoarthritis breaks down the cartilage cushioning the ends of the bones, body attempts to repair the loss by creating bone spurs near the damaged area.[28]

1.7.2 Pathology

The pathology may include:

- Cartilage has irregular surface with pitting and loss of cartilage
- Subchondral bone shows eburnation (polishing due to friction of bone against bone in joint), fractures
- Bony spurs (osteophytes), loose bodies (detached cartilage or cartilage/bone within joint space with necrotic calcified centers, may become attached to synovial membrane, revascularize and convert to viable bone)[29]

1.7.3 Pathophysiology

Osteophytes form because of the increase in a damaged joint's surface area. This is most common from the onset of arthritis. Osteophytes usually limit joint movement and typically cause pain. Osteophytes form naturally on the back of the spine as a person ages and are a sign of degeneration in the spine. In this case, the spurs are not the source of back pains, but instead are the common symptom of a deeper problem. However, bone spurs on the spine can impinge on nerves that leave the spine for other parts of the body. This impingement can cause pain in both upper and lower limbs and a numbness or tingling sensations in the hands and feet because the nerves are supplying sensation to their dermatomes. Osteophytes may also be the end result of

certain disease processes. Osteomyelitis, a bone infection, may leave the adjacent bone with a spur formation.[29]

1.7.4 Complications

Bone spurs along the spinal column can cause some serious complications. Complications may include:

- Severe neck or back pain
- Restricted movement
- Radiating arm and leg pain
- Weakness in the extremities
- Numbness and
- In some cases, disability.[30]

1.7.5 Treatment and drugs

Surgery. Bone spurs that limit the range of motion or press on nerves may require surgical removal.

Drugs such as ibuprofen, naproxen, COX-2 inhibitors and diazepam are used.[30]

1.7.6 Toxicities

Toxicity of drugs can causes ulceration, bleeding, perforation, hypertension and edema.[30]

1.8 What Is Spondylolisthesis?

Spondylolisthesis is a spinal condition that affects the lower vertebrae (spinal bones). This disease causes one of the lower vertebras to slip forward onto the bone directly beneath it. This is a painful condition, but it is treatable in most cases. Proper exercise techniques can help keep this condition from developing.[31]

1.8.1 Causes

Causes of spondylolisthesis vary based on age, heredity, and lifestyle. Children may suffer from this condition as the result of a birth defect or injury. However, patients of all ages are susceptible if the condition runs in their families. Rapid growth during adolescence may also be a contributing factor. Playing sports may also cause overstretching of the spine and stress on the lower back.[31]

1.8.2 Pathology

Spondylolisthesis is officially categorized into five different types by the Wiltse classification system: Dysplastic, Isthmic, Degenerative, Traumatic, and Pathologic. Dysplastic spondylolisthesis is a true congenital spondylolisthesis that occurs because of malformation of the lumbosacral junction with small, incompetent facet joints. Dysplastic spondylolisthesis is very rare, but tends to progress rapidly, and is often associated with more severe neurological deficits. Isthmic spondylolisthesis is the most common form of spondylolisthesis. It is thought that the vast majority of isthmic slips do not become symptomatic, but the incidence of symptoms is unknown. Degenerative spondylolisthesis is a disease of the older adult that develops as a result of facet arthritis and facet remodeling. As the facets remodel, they take on a

more sagittal orientation, allowing a mild slip to occur. Most slips are asymptomatic but can worsen the symptoms of neurogenic claudication when associated with lumbar spinal stenosis. Traumatic spondylolisthesis is very rare and may be associated with acute fracture of the inferior facets or pars interarticularis. Pathologic spondylolisthesis is the last type and is also very rare. This type can occur following damage to the posterior elements from metastases or metabolic bone disease. These slips have been reported in cases of Paget's disease of bone, tuberculosis, giant-cell tumors, and tumor metastases.[32]

1.8.3 Pathophysiology

During extension of the lumbar spine, the inferior articular process of the cranial vertebra impact on the pars inter-articularis of the caudal vertebra. When this impact is repeated, a stress fracture of the pars may develop. In other cases, direct fracture can occur as a result of acute trauma. It has also been established that spondylolisthesis is associated with defects of the intervertebral disc. Grade one spondylolisthesis accounts for 80% of all cases. Patients subject to grade one or grade two spondylolisthesis will rarely progress to higher grades once they reach skeletal maturity.[32]

1.8.4 Complications

Complications of spondylolisthesis include:

- Chronic pain in the lower back or legs
- Numbness, tingling or weakness in the legs
- Severe compression of the nerve can cause problems with bowel or bladder control.[33]

1.8.5 Treatment & Drugs

Both therapeutic and surgical methods may be used.

Drugs such as acetaminophen, ibuprofen, Neurontin, and morphine are used.[33]

1.8.6 Toxicities

Toxicity of drugs can causes stomach upset, bleeding, nausea, constipation, dizziness, drowsiness, and can also causes dependency.[33]

1.9 What Is Cervical Spondylosis?

Cervical spondylosis, also known as cervical osteoarthritis or neck arthritis, is a common, age related condition that affects the joints and discs in the neck. It develops from wear and tear of the cartilage and bones found in the cervical spine, which is in the neck. While it's largely due to age, it can be caused by other factors as well.[34]

1.9.1 Causes

Unfortunately, the bones and protective cartilage in the neck are prone to wear and tear that can lead to cervical spondylosis. Possible causes of the condition include:

- **Bone Spurs** These overgrowths of bone are the result of the body trying to grow extra bone to make the spine stronger. However, the extra bone can press on delicate areas of the spine, such as the spinal cord and nerves, resulting in pain.

- **Herniated Disks** The spinal disks can develop cracks, which allow leakage of the internal cushioning material. This material can press on the spinal cord and nerves, resulting in symptoms such as arm numbness and sciatica.

- **Injury** to the neck, such as during a fall or car accident, this can accelerate the aging process.[34]

1.9.2 Pathology

The pathology of cervical spondylosis includes:

- Age related degeneration and dehydration of interverbal disks

- Decreased cartilage between adjacent vertebral bodies

- Developmental laxity in the spinal supportive ligaments

- Hyper-mobility of spinal segment

- Bone-on bone apposition propagates bone spur formation which narrows the cervical spinal canal and may compress the cervical nerve roots and spinal cord.[35]

1.9.3 Pathophysiology

Intervertebral disks lose hydration and elasticity with age, and these losses lead to cracks and fissures. The surrounding ligaments also lose their elastic properties and develop traction spurs. The disk subsequently collapses as a result of biomechanical incompetence, causing the annulus to bulge outward. As the disk space narrows, the annulus bulges, and the facets override. This change, in turn, increases motion at that spinal segment and further hastens the damage to the

disk. Annulus fissures and herniation may occur. Acute disk herniation may complicate chronic spondylotic changes. As the annulus bulges, the cross-sectional area of the canal is narrowed. This effect may be accentuated by hypertrophy of the facet joints and of the ligamentum flavum, which becomes thick with age. Neck extension causes the ligaments to fold inward, reducing the anteroposterior diameter of the spinal canal. As disk degeneration occurs, the uncinate process overrides and hypertrophies, compromising the ventrolateral portion of the foramen. Likewise, facet hypertrophy decreases the dorsolateral aspect of the foramen. This change contributes to the radiculopathy that is associated with cervical spondylosis.[35]

1.9.4 Complications

Complications may include:

- Nerve Compression
- Spinal Stenosis
- Permanent Disability[36]

1.9.5 Treatment and drugs

Treatment for cervical spondylosis depends on the severity of signs and symptoms.

Surgery. If conservative treatment fails or if the neurological signs and symptoms such as weakness in the arms or legs worsen, patient might need surgery to create more room for the spinal cord and nerve roots.

Drugs such as ibuprofen, naproxen, prednisone, and pregabalin are used.[36]

1.9.6 Toxicities

Toxicity of drugs can causes gastropathy, renal toxicity, hypertension, liver abnormalities, bleeding, nausea and vomiting.[37]

1.10 What Is Lumbar Spondylosis?

Lumbar spondylosis is principally a disease of mid and later life. As the lumbar discs and associated ligaments undergo aging, the disc spaces frequently narrow. Thickening of the ligaments that surround the disc and those that surround the facet joints develops. This ligamentous thickening may eventually become calcified.[38]

1.10.1 Causes

Spondylosis is caused by degenerative changes within the intervertebral discs. The soft, elastic material dries out and loses height. Thickening of the ligaments that surround the disc occurs. Alterations of the alignment of the joints that connect the back of the spine also occur. These other ligaments undergo further degenerative changes, thickening and potential calcification.[38]

1.10.2 Pathology

Progressive degenerative changes in the posterior joints lead to marked destruction and instability. Similar changes in the disc result in herniation, internal disruption, and resorption. Combined changes in posterior joint and disc sometimes produce entrapment of a spinal nerve in the lateral recess, central stenosis at one level, or both of these conditions. Changes at one level often lead, over a period of years, to multilevel spondylosis.[39]

1.10.3 Pathophysiology

Lumbar spondylosis occurs as a result of new bone formation in areas where the anular ligament is stressed. When annular ligament is put to stress body responds by forming new bone i.e. osteophytes which results in lumbar spondylosis.[39]

1.10.4 Complications

Complications may include:

- Nerve compression
- Spinal stenosis.[40]

1.10.5 Treatment and drugs

Surgery is used if needed.

Drugs such as tramadol , naproxen, amitriptyline, clonazepum are used.[40]

1.10.6 Toxicities

Toxicity of drugs can causes constipation, difficulty thinking, dizziness, burning sensation, kidney disease, morning sleepiness, and drowsiness.[40]

1.11 What Is Tuberculous Spondylitis?

Tuberculous spondylitis also known as pott disease refers to vertebral body and intervertebral disc involvement with tuberculosis. The spine is the most frequent location of musculoskeletal TB and common related symptoms are back pain and lower limb weakness or paraplegia.[41]

1.11.1 Causes

Tuberculous spondylitis or tuberculosis of the spine caused by infection of the spinal column, or vertebral column, by the tuberculosis bacillus, mycobacterium tuberculosis. Pott disease is characterized by softening and collapse of the vertebrae, often resulting in a hunchback curvature of the spine. The infection begins in the body of the vertebra and spreads slowly to contiguous structures.[41]

1.11.2 Pathology

There is usually a slow collapse of one or usually more vertebral bodies, which spreads underneath the longitudinal ligaments. This results in an acute kyphotic or "gibbus" deformity. This angulation, coupled with epidural granulation tissue and bony fragments, can lead to cord compression. Unlike pyogenic infections, the discs can be preserved. In late-stage spinal TB, large paraspinal abscesses without severe pain or frank pus are common, leading to the expression "cold abscess".[42]

1.11.3 Pathophysiology

Pott disease is usually secondary to an extraspinal source of infection. Pott disease manifests as a combination of osteomyelitis and arthritis that usually involves more than 1 vertebra. The anterior aspect of the vertebral body adjacent to the subchondral plate is usually affected. Tuberculosis may spread from that area to adjacent intervertebral disks. In adults, disk disease is secondary to the spread of infection from the vertebral body. Progressive bone destruction leads to vertebral collapse and kyphosis. The spinal canal can be narrowed by abscesses, granulation tissue, or direct dural invasion, leading to spinal cord compression and neurologic deficits.[42]

1.11.4 Complications

A common complication of tuberculous spondylitis includes:

- Paravertebral, extradural or other soft tissue cold abscess.
- Pott's paraplaegia.[43]

1.11.5 Treatment and Drugs

Surgery is indicated especially in the paraplegia. It consists of excision of the disease focus and strut grafting.

Drugs: Triple therapy regimen (Streptomycin, Isoniazid, and Rifampicin) is used.[43]

1.11.6 Toxicities

Toxicity of drugs can cause renal failure, liver disease, and ototoxicity to the fetus.[43]

1.12 What Is Intracerebral Hemorrhage (ICH)?

Intracerebral hemorrhage (ICH) is a type of stroke caused by bleeding within the brain tissue itself, a very life-threatening situation. A stroke occurs when the brain is deprived of oxygen due to an interruption of its blood supply. ICH is most commonly caused by hypertension, arteriovenous malformations, or head trauma. Treatment focuses on stopping the bleeding, removing the blood clot, and relieving the pressure on the brain.[44]

1.12.1 Causes

High blood pressure is the most common cause of intracerebral hemorrhage. In younger people, another common cause is abnormally formed blood vessels in the brain. Other causes include:

- head injury or trauma
- ruptured cerebral aneurysm (weak spot in a blood vessel that bursts)
- arteriovenous malformation (a grouping of malformed blood vessels in the brain that disrupts normal blood flow)
- use of blood thinners
- bleeding tumors
- cocaine use (can cause severe hypertension and lead to hemorrhage)
- bleeding disorders (e.g., hemophilia, sickle cell anemia)[45]

1.12.2 Pathology

The most common sites of ICH are cerebral hemispheres, basal ganglia, thalamus, brainstem, and cerebellum. The gross and microscopic changes in the brain depend on the location of ICH, but the general appearance is similar. In the acute stages, the ICH consists of a liquid or semiliquid mass of blood with surrounding oedema. After a few days, the ICH changes its consistency and adopts a brown colour, while oedema begins to recede. After several months or years, depending on its size, the ICH becomes a cavity. Small ICH can be reabsorbed almost completely, leavingbehind a small linear scar. Microscopically, the ICH in its acute stages consists of extravasated well-preserved red blood cells without any inflammation. Subsequently, the RBC begins to lyse and neutrophils appear. This is followed by infiltration of macrophages whose main role is to phagocytose blood products and necrotic tissue. The brown discolouration

of the slightly older haematomas noted macroscopically is due to the presence of two major haemoglobin derived pigments, haemosiderin and haematoidin.[46]

1.12.3 Pathophysiology

Intracerebral hemorrhage can results from the following effects.

- Primary immediate effect
    - Hemorrhage growth
    - Increase ICP
- Secondary effect
    - Downstream effect
    - Edema
    - Ischemia[46]

1.12.4 Complications

Complications may include:

- impaired language skills
- fatigue
- problems with swallowing
- vision loss
- difficulty with sensations or movements on one side of the body

- pneumonia
- cognitive dysfunction (memory loss, difficulty reasoning), confusion
- swelling on the brain
- seizures
- depression, emotional problems[47]

1.12.5 Treatment and Drugs

Treatment within the first three hours of the onset of symptoms generally results in a better outcome.

Surgery can relieve pressure on the brain and repair torn arteries.

Drugs such as labetalol, nicardipine, mannitol, and phenytoin are used.[48]

1.12.6 Toxicities

Toxicity of drugs can causes nausea, vomiting, allergic reaction, hypotension and dizziness.[48]

1.13 What Is Cerebral Aneurysm?

A cerebral aneurysm (also known as an intracranial or intracerebral aneurysm) is a weak or thin spot on a blood vessel in the brain that balloons out and fills with blood. The bulging aneurysm can put pressure on a nerve or surrounding brain tissue. It may also leak or rupture, spilling blood into the surrounding tissue. Some cerebral aneurysms, particularly those that are very small, do not bleed or cause other problems. Cerebral aneurysms can occur anywhere in the brain, but most are located along a loop of arteries that run between the underside of the brain and the base of the skull.[49]

1.13.1 Causes

Cerebral aneurysms can be congenital, resulting from an inborn abnormality in an artery wall. Cerebral aneurysms are also more common in people with certain genetic diseases, such as connective tissue disorders and polycystic kidney disease, and certain circulatory disorders, such as arteriovenous malformations. Other causes include trauma or injury to the head, high blood pressure, infection, tumors, atherosclerosis, cigarette smoking, and drug abuse.[49]

1.13.2 Pathology

Grossly aneurysms are rounded lobulated focal outpouchings which usually arise at the arterial bifurcations; it may arise from the lateral wall. Most intracranial aneurysms are true aneurysms. The aneurysmal pouch is composed of thickened hyalinised intima with the muscular wall and internal elastic lamina being absent as the normal muscularis and elastic lamina terminate at the neck of the aneurysm. As the aneurysm grows it may become irregular in outline, and may have mural thrombus. Typically rupture occurs from the dome.[50]

1.13.3 Pathophysiology

The pathogenesis of cerebral aneurysms is related inherently to structural aberrations of the cerebrovasculature, although the etiology of these abnormalities may be diverse. The integrity of the internal elastic lamina is compromised, with associated elastic defects in the adjacent layers of the tunica media and adventitia. Muscular defects of the tunica media and minimal support of adjacent brain parenchyma augment the pathologic potential of chronic hemodynamic stress on the arterial wall. Focal turbulence and discontinuity of the normal architecture at vessel bifurcations may account for the propensity of saccular aneurysm formation at these locations. A

multifactorial etiology is most likely, reflecting the interaction of environmental factors, such as atherosclerosis or hypertension, and a congenital predisposition associated with various vascular abnormalities. Abnormalities of the internal elastic lamina may be congenital or degenerative. Multiple conditions have been associated with cerebral aneurysms; they include the following:

- Autosomal dominant inherited polycystic kidney disease
- Arteriovenous malformations
- Other vascular anomalies
- Other collagen type III disorders
- Systemic lupus erythematosus
- Sickle cell anemia
- Bacterial endocarditis
- Fungal infections
- Neurofibromatosis type 1
- Tuberous sclerosis[50]

1.13.4 Complications

Complications that can develop after the rupture of an aneurysm include:

- **Re-bleeding.** An aneurysm that has ruptured or leaked is at risk of bleeding again. Rebleeding can cause further damage to brain cells.
- **Vasospasm.** After a brain aneurysm ruptures, blood vessels in the brain may narrow erratically (vasospasm).

- **Hydrocephalus.** When an aneurysm rupture results in bleeding in the space between the brain and surrounding tissue. Blood can block circulation of the fluid surrounding the brain and spinal cord. This condition can result in hydrocephalus.[51]

1.13.5 Treatments and drugs

There are two common treatment options for a brain aneurysm.

Surgical clipping is a procedure to close off an aneurysm. The neurosurgeon removes a section of them skull to access the aneurysm and locates the blood vessel that feeds the aneurysm.

Endovascular coiling is a less invasive procedure than surgical clipping. The surgeon inserts a hollow plastic tube (catheter) into an artery, usually in the groin, and threads it through the body to the aneurysm.

Drugs such as acetaminophen, nimodipine, and phenytoin are used.[52]

1.13.6 Toxicities

Toxicity of drugs can causes nausea, vomiting, allergy, hypotension, insomnia and mental confusion.[52]

1.14 What Is Chiari Malformation?

Chiari malformation is a condition in which brain tissue extends into the spinal canal. It occurs when part of the skull is abnormally small or misshapen, pressing on the brain and forcing it downward. Chiari malformation is uncommon, but improved imaging tests have led to more frequent diagnoses. Chiari malformation type I develop as the skull and brain are growing. As a

result, signs and symptoms may not occur until late childhood or adulthood. The most common pediatric form, called Chiari malformation type II, is present at birth (congenital).[53]

1.14.1 Causes

Chiari malformation type I occur when the section of the skull containing a part of the brain (cerebellum) is too small or is deformed, thus putting pressure on and crowding the brain. The lower part, or tonsils, of the cerebellum are displaced into the upper spinal canal. Chiari malformation type II is nearly always associated with a form of spina bifida called myelomeningocele. When the cerebellum is pushed into the upper spinal canal, it can interfere with the normal flow of cerebrospinal fluid that protects the brain and spinal cord. This impaired circulation of cerebrospinal fluid can lead to the blockage of signals transmitted from brain to the body, or to a buildup of spinal fluid in the brain or spinal cord. Alternatively, the pressure from the cerebellum upon the spinal cord or lower brainstem can cause neurological signs or symptoms.[53]

1.14.2 Pathology

Chiari I malformation is characterised by inferior herniation of the cerebellar tonsils through the foramen magnum, due essentially to a mismatch between size and content of the posterior fossa. Chiari I need to be distinguished from tonsillar ectopia, which is an asymptomatic and incidental finding in normal individuals, whereby the tonsils protrude through the foramen magnum by no more than 3-5 mm.[54]

1.14.3 Pathophysiology

The most widely accepted pathophysiological mechanism by which Chiari type I malformations occur is by a reduction or lack of development of the posterior fossa as a result of congenital or acquired disorders. Congenital causes include hydrocephalus, craniosynostosis (especially of the lambdoid suture), hyperostosis (such as craniometaphyseal dysplasia, osteopetrosis, erythroid hyperplasia), X-linked vitamin D-resistant rickets, and neurofibromatosis type I. Acquired disorders include space occupying lesions due to one of several potential causes ranging from brain tumors to hematomas. Head trauma may cause cerebellar tonsillar ectopia, possibly because of dural strain. Additionally, ectopia may be present but asymptomatic until whiplash causes it to become symptomatic. Posterior fossa hypoplasia causes reduced cerebral and spinal compliance.[55]

1.14.4 Complications

In some people, Chiari malformation can become a progressive disorder and lead to serious complications. The complications associated with this condition include:

- **Hydrocephalus.** (An accumulation of excess fluid)
- **Spina bifida.** (A condition in which spinal cord or its covering isn't fully developed. Part of the spinal cord is exposed, which can cause serious conditions such as paralysis)
- **Syringomyelia.** (Some people with Chiari malformation also develop a condition called syringomyelia, in which a cavity or cyst forms within the spinal column)[56]

1.14.5 Treatment and Drugs

Treatment for Chiari malformation depends on the severity and the characteristics of condition. If the symptoms aren't severe, doctors may recommend just monitoring the situation with regular MRI's and treating the symptoms individually. However, if symptoms are interfering with quality of life, are getting worse, or if the nervous system is being impaired, doctors may recommend surgery.

Surgery. The most common surgical treatment, performed by a neurosurgeon, is known as decompression surgery.

Drugs. When headaches or other types of pain are the primary symptom, doctors may recommend pain medication. Drugs such as pregabalin, paracetamol may be used.[56]

1.14.6 Toxicities

Toxicity of drugs can causes nausea, vomiting, thrombocytopenia, leucopenia, and skin rashes.[56]

1.15 What Is Multiple Sclerosis?

Multiple sclerosis is a disease which causes demyelination of the brain and spinal cord nerve cells. When this occurs, axons (the parts of the nerve cells which conduct impulses to other cells), don't work as well. Myelin acts like insulation on electrical wires. As more areas or nerves are affected by this loss of myelin, patients develop symptoms because the ability of axons to conduct impulses is diminished or lost.[57]

1.15.1 Causes

While multiple sclerosis is considered an autoimmune disorder, the exact cause hasn't yet been found. There are many theories regarding the reason that people develop multiple sclerosis; these theories range from vitamin D deficiency to a viral infection. Even consuming too much salt is being looked at as possible cause of multiple sclerosis. However, none of these theories have been proven, and the cause of multiple sclerosis remains unknown.[57]

1.15.2 Pathology

Multiple sclerosis can be pathologically defined as the presence of distributed scars in the central nervous system. These sclerosis are the remainings of previous demyelinating lesions in the CNS white matter of a patient showing special characteristics, like for example confluent instead of perivenous demyelination.[58] At least five characteristics are present in CNS tissues of MS patients: Inflammation beyond classical white matter lesions, intrathecal Ig production with oligoclonal bands, an environment fostering immune cell persistence, Follicle-like aggregates in the meninges and a disruption of the blood–brain barrier also outside of active lesions. The scars that give the name to the condition are produced by the astrocyte cells healing old lesions.[59]

1.15.3 Pathophysiology

Multiple sclerosis (MS) is a condition where the CNS of a person presents a special kind of distributed glial scars (sclerosis) which are a remaining of a previous inflammatory demyelination. MS pathophysiology is complex and still under investigation and there is no agreement about its scope. There are two phases for how an unknown underlying condition may cause damage in MS: First some MRI-abnormal areas with hidden damage appear in the brain

and spine (NAWM, NAGM, DAWM). Second, there are leaks in the blood–brain barrier where immune cells infiltrate causing the known demyelination and axon destruction. Some clusters of activated microglia, transection of axons and myelin degeneration is present before the BBB breaks down and the immune attack begins.[58]

1.15.4 Complications

Complications may include:

- Muscle stiffness or spasms
- Paralysis, typically in the legs
- Problems with bladder, bowel or sexual function
- Mental changes, such as forgetfulness or mood swings
- Depression
- Epilepsy[60]

1.15.5 Drugs

Drugs such as Interferon, teriflunomide, fingolimod, and natalizumab are used[60]

1.15.6 Toxicities

Toxicity of drugs can causes depression, heart disease , severe liver injury, and brain infection.[60]

1.16 What Is Meningioma?

Meningioma is the most common type of primary brain tumor, accounting for approximately 30% of all brain tumors. Meningiomas originate in the meninges**,** which are the outer three layers

of tissue between the skull and the brain that cover and protect the brain just under the skull. Meningiomas grow out of the middle layer of the meninges, called the arachnoid. When they grow, they press against the brain or spinal cord. About 85% of meningiomas are benign (non-cancerous, slow growing) tumors. Some meningiomas may not need immediate treatment and can often remain undetected for many years.[61]

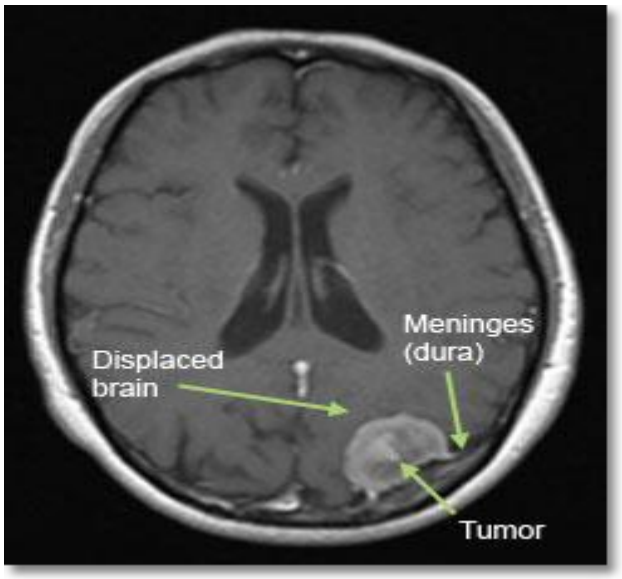

Figure 1.4: MRI image showing a typical meningioma tumor.[61]

1.16.1 Causes

It isn't clear what causes a meningioma. Doctors know that something alters some cells in meninges to make them multiply out of control, leading to a meningioma tumor. But whether this occurs because of genes, hormones or a combination of these factors remains unknown.[61]

1.16.2 Pathology

Meningiomas are classified according to the World Health Organization (WHO). The pathology of meningioma may include:

- WHO grade I meningiomas do not meet any of the criteria for a higher grade lesion based upon morphologic criteria.

- WHO grade II meningiomas have increased mitotic activity and three or more of the following features: increased cellularity, small cells with a high nuclear: cytoplasmic ratio, prominent nucleoli, uninterrupted patternless or sheet-like growth, or foci of spontaneous or geographic necrosis.

- WHO grade III meningiomas have ≥20 mitoses per ten high powered fields and or malignant characteristics resembling carcinoma, sarcoma, or melanoma. Features that support the diagnosis of malignant meningioma include the loss of usual meningioma growth patterns, infiltration of underlying brain, abundant mitoses with atypical forms, and multifocal microscopic foci of necrosis.[62]

1.16.3 Pathophysiology

Meningiomas may occur intracranially or within the spinal canal. They are thought to arise from arachnoidal cap cells, which reside in the arachnoid layer covering the surface of the brain. Meningiomas commonly are found at the surface of the brain, either over the convexity or at the skull base. In rare cases, meningiomas occur in an intraventricular or intraosseous location.[62]

1.16.4 Complications

A meningioma and its treatment, typically surgery and radiation therapy, can cause long-term complications, including:

- Difficulty concentrating
- Memory loss
- Personality changes
- Seizures[63]

1.16.5 Treatment and drugs

The treatment for a meningioma depends on many factors, including the size of meningioma, where it's situated and how aggressive it's believed to be.

Surgery. If meningioma causes signs and symptoms or shows signs that it's growing, doctor may recommend surgery. Surgeons work to remove the meningioma completely. But because a meningioma may occur near many delicate structures in the brain or spinal cord, it isn't always possible to remove the entire tumor. In those cases, surgeons remove as much of the meningioma as possible.

Drugs such as hydroxyurea, octreotide, lanreotide are sometimes used.[64]

1.16.6 Toxicities

Toxicity of drugs can causes nausea, vomiting, headache, diarrhea, abdominal pain, bradycardia and allergy.[64]

1.17 What Is Schwannoma?

Schwannoma is a benign tumor of the nerve of hearing (the 8th cranial nerve). It is an uncommon, noncancerous and usually slow-growing tumor that develops on the main nerve leading from inner ear to brain. Schwannoma also known as acoustic neuroma.[65]

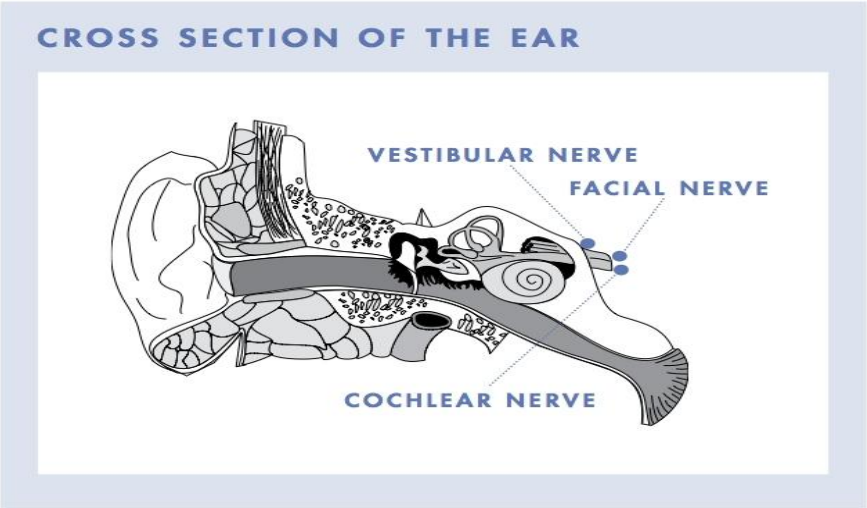

Figure1.5: The cross section of the ear.[65]

1.17.1 Cause

Like many tumor types, the exact cause of schwannoma is not known. However, it is believed to occur when there is a defect in a gene that normally prevents tumors from forming.[65]

1.17.2 Pathology

Acoustic schwannomas are benign tumours, which usually arise from the intracanalicular segment of the vestibular portion of the vestibulocochlear nerve near the transition point between glial and Schwann cells. In over 90% of cases these tumours arise from the inferior division of the vestibular nerve. They are well circumscribed encapsulated masses, which unlike neuromas,

arise from but are separate from nerve fibers, which they usually spray and displace rather than incorporated. They can display two types of growth pattern:

Antoni A

- elongated cells with cytoplasmic processes arranged in fascicles
- little stromal matrix

Antoni B

- loose meshwork of cells
- less densely cellular
- microcysts change[66]

1.17.3 Pathophysiology

The vast majority of acoustic neuromas develop from the Schwann cell investment of the vestibular portion of the vestibulocochlear nerve. Less than 5% arise from the cochlear nerve. The superior and inferior vestibular nerves appear to be the nerves of origin with about equal frequency. Although some tumors adhere to one or another of these growth patterns, others appear to alternate between periods of no or slow growth and rapid growth. Tumors that have undergone cystic degeneration (presumably because they have outgrown their blood supply) are sometimes capable of relatively rapid expansion because of enlargement of their cystic component. Because acoustic tumors arise from the investing Schwann cell, tumor growth generally compresses vestibular fibers on the surface. Destruction of vestibular fibers is slow; consequently, many patients experience little or no disequilibrium or vertigo. Once the tumor has grown sufficiently large to fill the internal auditory canal, it may continue growth either by

expanding bone or by extending into the cerebellopontine angle. Growth within the cerebellopontine angle is generally spherical. Acoustic tumors, like other space-occupying lesions, produce symptoms by any of 4 recognizable mechanisms: (1) compression or distortion of the spinal fluid spaces, (2) displacement of the brain stem, (3) compression of vessels producing ischemia or infarction, or (4) compression and or attenuation of nerves.[67]

1.14.4 Complications

Schwannoma may cause a variety of permanent complications, including:

- Hearing loss
- Facial numbness and weakness
- Difficulties with balance
- Ringing in the ear[68]

1.17.5 Treatments and Drugs

Treatment may vary, depending on the size and growth of the acoustic neuroma.

Stereotactic radiosurgery. Doctor may recommend stereotactic radiosurgery, if patient has an acoustic neuroma. Some very large tumors can't be treated with stereotactic radiosurgery.

Sugery. Patient may need surgery to remove an acoustic neuroma. Surgeon may use one of several techniques for removing an acoustic neuroma, depending on the size of tumor, preoperative hearing status and other factors. The goal of surgery is to remove the tumor, preserve the facial nerve to prevent facial paralysis and preserve hearing when possible.

Drugs such as naproxen, clonazepum may be used.[69]

1.17.6 Toxicities

Toxicity of drugs can causes gastrointestinal discomport, bleeding, nausea, and headache.[69]

1.18 What Is Glioma?

Glioma is a type of tumor that occurs in the brain and spinal cord. Gliomas begin in the gluey supportive cells (glial cells) that surround nerve cells and help them function.Three types of glial cells can produce tumors. Gliomas are classified according to the type of glial cell involved in the tumor.Types of glioma include:

- **Astrocytomas,** including astrocytoma, anaplastic astrocytoma and glioblastoma
- **Ependymomas,** including anaplastic ependymoma, myxopapillary ependymoma and subependymoma
- **Oligodendrogliomas,** including oligodendroglioma, anaplastic oligodendroglioma and anaplastic oligoastrocytoma[70]

1.18.1 Causes

Although no specific cause of glioma has been identified, research suggests that the following factors play a role:

- Several acquired gene mutations are associated with the condition. For example, the tumor suppressor p53 has been found to be mutated early on in the course of glioma.
- Increasing age is another risk factor associated with glioma.
- Males are at a slightly greater risk of this condition than females.[70]

1.18.2 PATHOLOGY

Pathology reports typically include a section dealing with microscopic descriptions of hematoxylin and eosin stained tumour tissue viewed on a glass slide. Pathology report will also include genetic analysis indicating genomic mutations, copy number alterations (gains or losses of genes or multi-gene regions of DNA) and polysomy or monosomy (gain or loss of an entire chromosome).[71]

1.18.3 Pathophysiology

High-grade gliomas are highly vascular tumors and have a tendency to infiltrate. They have extensive areas of necrosis and hypoxia. Often, tumor growth causes a breakdown of the blood brain barrier in the vicinity of the tumor. As a rule, high-grade gliomas almost always grow back even after complete surgical excision, so are commonly called recurrent cancer of the brain. Several acquired genetic mutations have been found in gliomas. Tumor suppressor protein 53 (p53) is mutated early in the disease. P^{53} is the "guardian of the genome," which, during DNA and cell duplication, makes sure the DNA is copied correctly. When p53 itself is mutated, other mutations can survive. Phosphatase and tensin homolog (PTEN), another protein that also helps destroy cells with dangerous mutations, is itself lost or mutated. Epidermal growth factor receptor, a growth factor that normally stimulates cells to divide, is amplified and stimulates cells to divide too much. Together, these mutations lead to cells dividing uncontrollably, a hallmark of cancer. Recently, mutations in *IDH1* and *IDH2* were found to be part of the mechanism and associated with a more favorable prognosis. The *IDH1* and *IDH2* genes are significant because they are involved in the citric acid cycle in mitochondria. Mitochondria are involved in apoptosis. Furthermore, the altered glycolysis metabolism in some cancer cells leads to low

oxygen (hypoxia). The normal response to hypoxia is to stimulate the growth of new blood vessels (angiogenesis). So, these two genes may contribute to both the lack of apoptosis and vascularization of gliomas.[72]

1.18.4 Complications

Complications of glioma include:

- Brain herniation
- Coma
- Inability to speak
- Inability to swallow
- Weakness or fatigue
- Numbness
- Disability[73]

1.18.5 Treatments and Drugs

Treatment for glioma depends on the type, size, grade and location of the tumor, as well as age, overall health and preferences.

Surgery to remove as much of the tumor as possible is usually the first step in treating most types of gliomas. Surgery to remove a glioma carries risks, such as infection and bleeding.

Radiation therapy usually follows surgery in treatment of glioma, especially high-grade gliomas. Radiation uses high-energy beams, such as X-rays or protons, to kill tumor cells.

Drugs such as bevacizumab, dexamethasone, and phenytoin are used.[74]

1.18.6 Toxicities

Toxicity of drugs can causes nausea, vomiting, headache, diarrhoea, hypertension, diabetes, and allergy.[74]

1.19 What Is Medulloblastoma?

Medulloblastoma is a malignant (cancerous) brain tumor that arises from the cerebellum, the part of the brain that controls balance, coordination and other complex functions. Most patients have symptoms of headache and vomiting, and can also have problems with balance and vision when they are diagnosed.[65]

1.19.1 Cause

Like many tumor types, the exact cause of medulloblastoma is not known. However, scientists are making significant strides in understanding its biology. Changes have been identified in genes and chromosomes that may play a role in the development of this tumor. There are also a few rare, genetic health syndromes that are associated with increased risk for developing this tumor.[65]

1.19.2 Pathology

The tumour is pink or grey, and invades surrounding brain tissue without a capsule. Microscopically, the tumour cells are small round blue cells that may arrange into Homer-Wright rosettes. The cells are usually arranged in sheets. Other patterns may be apparent:

- Desmoplastic or nodular medulloblastoma contains nodules of larger, differentiated cells surrounded by a 'stroma' of small round blue cells.

- Medulloblastoma with extensive nodularity occurs when there are large nodules of differentiated cells.
- Anaplastic change is sometimes seen in medulloblastoma; cells may be variable sizes, have high mitotic counts.
- Large cell medulloblastoma is formed exclusively by large, malignant cells and overlaps with anaplastic changes; the nuclei are often vesicular and there is often eosinophilic cytoplasm.
- Minor variants with melanocytic or rhabdoid differentiation may be seen.[75]

1.19.3 Pathophysiology

Medulloblastoma arises from the cerebellar stem cells, which are normally involved in the anatomical development of the cerebellum and posterior cranial fossa structures. Medulloblastoma is an invasive and rapidly growing brain tumor which may metastasize to different organs of the body. Genes involved in the pathogenesis of medulloblastoma include CTNNB1 gene, PTCH1 gene, MLL2 gene, SMARCA4 gene, DDX3X gene, CTDNEP1 gene, KDM6A gene, and TBR1 gene. Medullobastomas are associated with a number of syndromes that include Gorlin syndrome and Turcot syndrome.[75]

1.19.4 Complications

Complications of medulloblastoma include:

- Brain herniation: Damage to the lower portion of the brain due to increased pressure within the brain
- Loss of communication skills: Aphasia

- Permanent neurologic impairment
- The loss of the ability to perform basic self-care
- Coma[73]

1.19.5 Treatment and Durgs

Treatment consists of surgical removal of as much tumor as possible, radiation, and then chemotherapy.

Surgery is always the first treatment because it decreases the disease "burden" so that radiation and chemotherapy can effectively treat any remaining tumor cells.

Drugs such as dexamethasone, hydrocortisone, prednisolone and, mannitol are used.[65]

1.19.6 Toxicities

Toxicity of drugs can causes nausea, vomiting, headache, diarrhea, muscle weakness, osteoporosis and allergy.[65]

1.20 What Is Glioblastoma (GBM)?

Glioblastomas (GBM) are tumors that arise from astrocytes, the star-shaped cells that make up the glue-like or supportive tissue of the brain. These tumors are usually highly malignant because the cells reproduce quickly and they are supported by a large network of blood vessels.[65]

1.20.1 Cause

Like many tumor types, the exact cause of glioblastoma is not known.[65]

1.20.2 Pathology

The World Health Organization (WHO) characterizes GBM as a grade IV tumor. GBM can present as either a primary or secondary tumor, in which the primary GBM has spread to another part of the brain. Primary tumors are more aggressive and have lower survival rates, while secondary tumors are usually the opposite. Common pathologic characteristics of GBM include hyperchromatic nuclei and the presence of necrotic tissue. Diffuse margins and microvascular proliferation allow GBMs to easily grow and metastasize. Tumors with diffuse margins more readily invade surrounding cerebral tissue which makes complete surgical resection difficult. Microvascular proliferation allows for excessive tumor growth. The pathologic characteristics of these malignant tumors provide insight into the causes of the poor prognosis of GBM patients.[76]

1.20.3 Pathophysiology

Glioblastomas can be classified as primary or secondary. Evidence indicates that primary and secondary glioblastomas constitute distinct disease entities that evolve through different genetic pathways, affect patients at different ages, and differ in response to some of the present therapies. Of all the astrocytic neoplasms, glioblastomas contain the greatest number of genetic changes, which, in most cases, result from the accumulation of multiple mutations.Over the past decade, the concept of different genetic pathways leading to the common phenotypic endpoint (ie, glioblastoma multiforme) has gained general acceptance. Genetically, primary and secondary glioblastomas show little overlap and constitute different disease entities. Studies are beginning to assess the prognoses associated with different mutations.

Some of the more common genetic abnormalities are described as follows:

- Loss of heterozygosity (LOH): LOH on chromosome arm 10q is the most frequent gene alteration for both primary and secondary glioblastomas.
- P^{53}: Mutations in p^{53}, a tumor suppressor gene, were among the first genetic alterations identified in astrocytic brain tumors.
- Epidermal growth factor receptor (EGFR) gene: The EGFR gene is involved in the control of cell proliferation. Multiple genetic mutations are apparent, including both overexpressions of the receptor as well as rearrangements that result in truncated isoforms.
- MDM2: Amplification or overexpression of MDM2 constitutes an alternative mechanism to escape from p53-regulated control of cell growth by binding to p53 and blunting its activity. Overexpression of MDM2 is the second most common gene mutation in glioblastoma.

Glioblastoma multiformes occur most often in the subcortical white matter of the cerebral hemispheres. When a tumor in the frontal cortex spreads across the corpus callosum into the contralateral hemisphere, it creates the appearance of a bilateral symmetric lesion.[77]

1.20.4 Complications

Complications of glioblastoma include:

- Brain neoplasm
- Coma
- Inability to speak

- Inability to swallow
- Weakness or fatigue
- Numbness
- Disability[73]

1.20.5 Treatments and Drugs

Glioblastoma can be difficult to treat because the tumors contain so many different types of cells.

Surgery: The first step in treating glioblastoma is a procedure to make a diagnosis, relieve pressure on the brain, and safely remove as much tumor as possible through surgery. Because gliblastomas have finger-like tentacles, they are very difficult to completely remove.

Radiation and chemotherapy may be used to slow the growth of tumors that cannot be removed with surgery. Chemotherapy may also be used to delay the need for radiation in young children.

Drugs such as vincrsistine, temozolomide, and bevacizumab may be used.[65]

1.20.6 Toxicities

Toxicity of drugs can causes nausea, vomiting, constipation, hypertension, and constipation.[65]

1.21 What Is Craniopharyngioma?

A craniopharyngioma is a noncancerous tumor that develops at the base of the brain near the pituitary gland. Pressure on the pituitary gland by the tumor reduces the availability of the hormone vasopressin, raising the pressure within the cranium. A craniopharyngioma usually

includes hard, calcified components within the tumor itself and affects the development of the adjacent skull.[65]

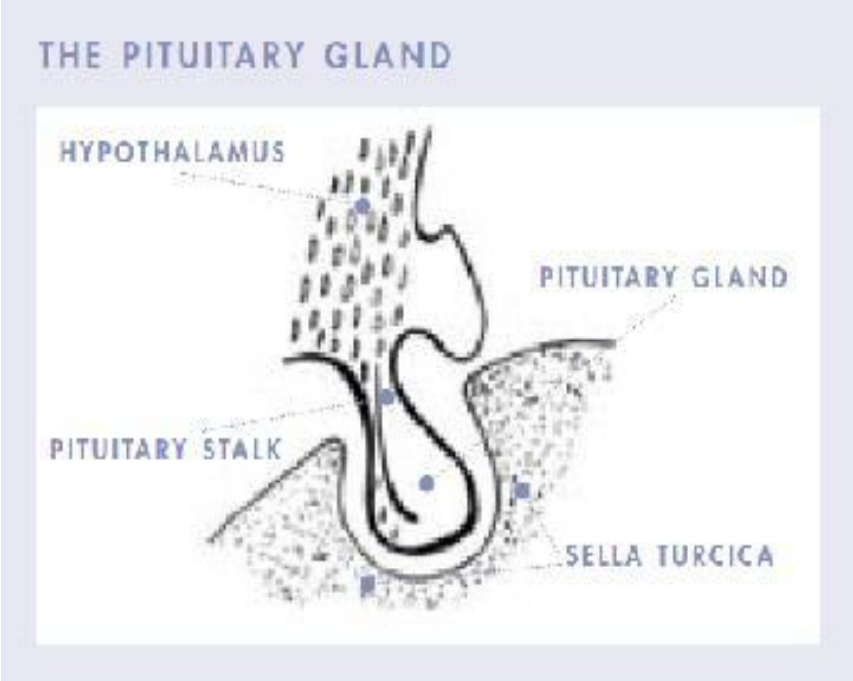

Figure 1.6: The pituitary gland[65]

1.21.1 Cause

Like many tumor types, the exact cause of craniopharyngioma is not known.This tumor most commonly affects children 5 to 10 years of age. Adults can sometimes be affected. Boys and girls are equally likely to develop this condition.[78]

1.21.2 Pathology

Craniopharyngiomas are epithelial tumors that usually arise in the pituitary stalk in the suprasellar region, adjacent to the optic chiasm. A small percentage arises within the sella, and a few tumors have been described within the optic system or the third ventricle.

- Embryonic cell rests of enamel organs located adjacent to the tuber cinereum along the pituitary stalk
- Cellular remnants of Rathke's cleft – papillary variant
- Metaplasia in cells of the adenohypophysis
- Predominantly appears as calcified cystic suprasellar mass which produces visual and pituitary defects
- May also appear in other locations such as nasal, 3rd ventricular, pineal, and infratentorial
- Rupture of cyst and leakage of content can cause recurrent asceptic meningitis or Mollaret's meningitis
- Often associated with tenacious adherence to adjacent normal vascular and neural structures which makes complete surgical resection challenging.[79]

1.21.3 Pathophysiology

Craniopharyngiomas are believed to arise from cellular remnants of the Rathke pouch, which is an embryologic structure that forms both the infundibulum and anterior lobe of the pituitary gland. These tumors have been identified extensively in suprasellar, parasellar, and ectopic locations. Typically, the tumors arise within the sella or adjacent suprasellar space. Transformation of normal cells into neoplastic ones likely involves multiple genomic changes, including loss of tumor-suppressor genes, activation of oncogenes, and alterations in DNA repair and methylation mechanisms. Although these events have started to be elucidated for neuroepithelial neoplasms, little progress has been made in understanding these events in craniopharyngiomas. Some chromosomal abnormalities, including deletions, translocations, and

increased copy numbers, have been recognized but are largely nonspecific. However, studies have identified the beta-catenin pathway as playing a potential role in the pathogenesis of these tumors. Beta-catenin is a downstream component of the Wnt signal transduction pathway that plays critical roles in the regulation of cellular proliferation, morphology, and development. One study showed that the accumulation of nuclear beta-catenin as measured immunohistochemically was able to help differentiate craniopharyngiomas from Rathke cleft cysts.[80]

1.21.4 Complications

Complications include:

- Seizures.
- Visual disturbances.
- CSF leakage.
- Herpes simplex encephalitis.[81]

1.21.5 Treatment and Drugs

Surgery. Surgery is the main treatment for craniopharyngioma.

Radiation therapy. In tumors that cannot be removed completely with surgery alone, radiation therapy is usually necessary.

Drug therapy currently is not usually a component of standard care for craniopharyngioma. Interferon and intracystic injection of chemotherapeutic agents (bleomycin) are occasionally used in cases of recurrent disease.[78]

1.21.6 Toxicities

Toxicity of drugs can cause nausea, vomiting, headache, diarrhea and weight loss.[78]

1.22 What Is Astrocytoma?

Astrocytomas are tumors that arise from astrocytes, star-shaped cells that make up the "glue like" or supportive tissue of the brain. These tumors are "graded" on a scale from I to IV based on how normal or abnormal the cells look. There are low-grade astrocytomas and high-grade astrocytomas. Low-grade astrocytomas are usually localized and grow slowly. High-grade astrocytomas grow at a rapid pace and require a different course of treatment. Most astrocytoma tumors in children are low grade. In adults, the majority are high grade.[65]

1.22.1 Cause

Like many tumor types, the exact cause of astrocytoma is not known. Some studies suggest that brain tumors may occur more frequently in people who have occupational exposure to certain chemicals, including some pesticides, formaldehyde, vinyl chloride, phenols, acrylonitrile, N-nitroso compounds, polycyclic aromatic hydrocarbons, lubricating oils, and organic solvents.[65]

1.22.2 Pathology

The Kernohan grading system is a 4-point scale of progressive malignancy that is structured as follows:

- Benign Astrocytoma (Grade I)
- Low-Grade Astrocytoma (Grade II)

- Anaplastic Astrocytoma (Grade III)
- Glioblastoma Multiforme (Grade IV)

Grades I and II

- Well-differentiated or low-grade tumors
- Low to moderate cellularity without mitoses
- Generally follow an indolent course

Grades III and IV

- Poorly differentiated or high-grade tumors
- Highly cellular neoplasms that are mitotically active and have potential for metastases
- Clinically aggressive[82]

1.22.3 Pathophysiology

Astrocytoma causes regional effects by compression, invasion, and destruction of brain parenchyma, arterial and venous hypoxia, competition for nutrients, release of metabolic end products (e.g., free radicals, altered electrolytes, neurotransmitters), and release and recruitment of cellular mediators (e.g., cytokines) that disrupt normal parenchymal function. Secondary clinical sequelae may be caused by elevated intracranial pressure (ICP) attributable to direct mass effect, increased blood volume, or increased cerebrospinal fluid (CSF) volume.[82]

1.22.4 Complications

Complications of astrocytoma include:

- Coma
- Loss of communication skills
- Permanent neurologic impairment
- The loss of the ability to perform basic self care.[73]

1.22.5 Treatment and Drugs

Treatment options depend on the type, size, and location of the tumor.

Surgery. Astrocytomas are often removed by surgery.

Drugs such as phenytoin, dexamethasone, and temozolomide may be used[65]

1.22.6 Toxicities

Toxicity of drugs can causes hypotension, nausea, vomiting, headache, mental disturbance and euphoria.[65]

1.23 Literature review on neurosurgery: Some literature on neurosurgery are given below-

1.23.1 Complications in spinal surgery: comparative survey of spine surgeons and patients who underwent spinal surgery.

Ratliff Jk et al. performed a study to investigate the complications in spinal surgery. Definitions of complications in spinal surgery are not clear. Therefore, the authors assessed a group of practicing spine surgeons and, through the surgeons' responses to an online and emailed survey,

developed a simple definition of operative complications due to spinal surgery. The authors surveyed a cohort of practicing spine surgeons via email and a web-based survey. Surgeons were presented with various complication scenarios and were asked to grade the presence or absence of a complication as well as complication severity, with responses limited to "major complication" and "minor complication or adverse event." The authors administered a similar assessment, modified for lay persons, to patients in a spinal surgery clinic.

Complete responses were obtained from 229 surgeons; orthopedic surgeons comprised the majority of respondents (73%). The authors obtained completed surveys from 197 patients. Overall, there was consistent agreement between physicians and patients regarding the presence or absence of a complication in the majority of scenarios (8 [73%] of 11 scenarios with agreement that a complication was present). The overall kappa value, evaluating major versus minor complication, and presence or absence of a complication over the entire cohort, was fair (kappa = 0.21). The authors found greater variation between the cohorts when evaluating complication severity. Patients were consistently more critical than physicians in the majority of scenarios in which a difference was evident. In 4 scenarios, patients were more likely than surgeons to deem the scenario a complication and to grade the complication as major versus minor ($p < 0.01$). In 3 additional scenarios, patients were more likely than physicians to grade a major complication as opposed to minor complication ($p < 0.01$). In only 1 scenario were patients less likely than physicians to report a complication ($p < 0.001$).

Comparing responses of spine surgeons and patients who underwent spinal surgery in assessing a group of common postoperative events, the authors found significant agreement on perception of presence of a complication in the majority of scenarios reviewed. However, patients were consistently more critical than surgeons when differences in reporting were found. The authors'

data underscore the importance of reconciling differing opinions regarding complications through open discussions between physicians and patients to ensure accurate patient expectations of planned medical or surgical interventions.[83]

1.23.2 Leg Weakness in a Patient with Lumbar Stenosis and Adrenal Insufficiency

Kyoung-Tae Kim et al. performed a study to find out leg weakness in a patient with lumbar stenosis. Lumbar spinal stenosis (LSS) is a common spinal disease in the elderly. Katz et al. reported that the more than 30,000 surgical procedures were performed in United States in 1994 to treat LSS. The rate of diagnosis is growing rapidly because of improvements in diagnostic imaging tools and surgical techniques, and the aging of the population. The cardinal symptom of LSS is neurogenic claudication, defined as pain in the buttocks and legs, and numbness or cramping of one or both legs induced by walking, which is relieved when sitting and bending forward. However, not all patients present with typical symptoms and clinical symptoms in elderly patients are often confused with symptoms of peripheral neuropathy, musculo-skeletal disease and other medical conditions. The decision to employ surgery must be made with caution in elderly patients with rapidly progressing leg weakness, because this condition rarely occurs in the absence of other combined conditions, and the incidence of combined disease in the elderly patients is high.

Neurological deficits, such as leg motor weakness and cauda equina syndrome are not common in LSS patients, because neural compression is relieved by positional change. In particular, the LSS symptom of rapidly progressing leg weakness must be distinguished from other causes, such as combined diseases or trauma. In the case presented here, whole spine MRI showed LSS at L4-5 and radiculopathy in L4, L5, and S1 with mild diabetic neuropathy, respectively. LSS was the

main cause of leg weakness and radiating pain. Many people do not consider supplements to be medications and often take them without medical advice. This type of behavior has recently been worsened by the increased use of the internet market, which allows easy access to materials, foods and even drugs without detailed confirmation. This can be dangerous, because the components of some supplements are often not clear.

Some drugs, such as anticoagulants, fungal agents, phenobarbital, phenytoin, rifampin, imipramine, chloropromazine, opiate drugs and glucocorticoid (systemic or topical), can induce primary or secondary AI. In particular, excessive glucocorticoids have long been associated with risk of osteoporosis, or iatrogenic AI. AI symptoms include general weakness, fatigue, nausea and vomiting, fever, dehydration, hypotension, hypoglycemia, hyponatremia, confusion, muscle weakness, shock and even death, induced by hypocortisolism. Cortisol has many important metabolic and endocrine functions that are essential for human survival, particularly during stress. Surgery, anesthesia, trauma, and severe illness, including infection, demand high cortisol level. Surgery is a potent activator of the hypothalamus-pituitary-adrenal axis, and AI patients need adequate perioperative glucocorticoid coverage. In case presented here, the patient was probably in a stage of hypocortisolism before surgery, which aggravated the hypocortisolism. Hypocortisolism also decreases the activation of the immune system, which might have triggered pseudomembranous colitis and cystitis. These infections, in turn, would have re-aggravated the hypocortisolism. This vicious cycle corresponded to the clinical course, but leg weakness and radiating pain, and voiding difficulty showed somewhat different patterns. AI can present as muscle weakness, but in this case, the patient complained of definite leg motor weakness.[84]

1.23.3 Non-traumatic spinal cord lesions: epidemiology& complications.

Gupta at el. Performed a prospective study consisted of 64 patients with NTSCL (Non-traumatic spinal cord lesions) admitted in neurological rehabilitation unit over a period of 32 months (June 2005–January 2008). During this period, overall 106 patients with spinal cord lesions (both traumatic and non-traumatic) were admitted for in-patient rehabilitation. Patients who were medically stable and able to participate actively for at least 2 hrs per day in the rehabilitation were included in the study. Patients with cardio respiratory co-morbidities, requiring ventilator for assisted respiration and with unstable vertebral injuries were excluded. They found that NTSCL constituted 60% (64 of 106) of the total SCL patients admitted for rehabilitation during the same period. Female patients outnumbered males (56.25%) in the study. Mean age, duration of illness and duration of stay in rehabilitation were 30.64±13.67 years (6–57), 7.09±9.15 months (1–48) and 55.75±40.91 days (14–193), respectively. The ratio of paraplegia and quadriplegia was 2:1. Forty-four patients (68.75%) had incomplete cord lesion according to the ASIA impairment scale. Spinal tumors (26.6%) were found to be the most common etiology, followed by Pott's spine (25%) and transverse myelitis (22%). Urinary tract infection was found to be the most common complication (50%), followed by spasticity (35.93%) and urinary incontinence (31.25%). The mean BI scores showed significant (P=0.000) functional recovery during rehabilitation using paired Student's *t*-test. The ASIA (American Spinal Injury Association) impairment scale showed significant neurological recovery (P=0.001) using the Wilcoxon non-parametric test. The etiology of NTSCL is given in table 1.1

Table 1.1: The etiology of NTSCL

Diagnosis	No. of patients	%	Level of lesion
Spinal tumors	17	26.6	C-7, HD-1, LD-8, L-1
Pott's spine	16	25	C-5, HD-2, LD-9
Transverse myelitis	14	22	C-3, HD-2, LD-9
OPLL	7	10.9	C-3, LD-4
Demyelination	4	6.3	C-1, HD-1, LD-2
Spinal arachnoiditis	2	3.1	LD-2
PIVD	2	3.1	C-1, CE-1
Ischemic myelopathy	1	1.6	C-1
Organo-phosphorus poisoning induced myelopathy	1	1.6	LD-1
Total	64	100	

Abbreviations: C, cervical lesion; CE, cauda equina syndrome; HD, high dorsal lesion (D1–D6); L, lumbar lesion; LD, low dorsal Lesion (D7–D12); OPLL, ossified posterior longitudinal ligament; PIVD, prolapsed inter-vertebral disc.

The frequency of common medical complications is shown in table 1.2

Table 1.2: The Frequency of Common Medical complications

Complications	No. of patients	%
Urinary tract infection	32	50
Spasticity	23	35.93
Urinary incontinence	20	31.25
Pressure ulcers	16	25
Constipation	15	23.5
Pain	14	21.87
Depression	14	21.87
Deep vein thrombosis	4	6.3
Flexor spasms	4	6.3
Genital ulcer/trauma	2	3.1
No complications	6	9.4

The most common etiology in this study was spinal tumors. Some earlier studies have also reported tumors as the most common etiology in their series. But as compared to these studies, the present study has all primary tumors. Spasticity was the second most common complication with 35.93% (23 of 64), urinary incontinence in 31.25% (20 of 64) and pressure ulcers in 25% of patients (16/64). Nair reported genitor-urinary complications in 70% of the participants in their series, with 60% of patients having at least one episode of UTI.[85]

1.23.4 The Influence of Pituitary Adenoma Size on Vision and Visual Outcomes after Trans-Sphenoidal Adenectomy.

Ren-wen Ho M.D. et al performed a study to investigate the quantitative relationship between pituitary macroadenoma size and degree of visual impairment, and assess visual improvement after surgical resection of the tumor. The medical records of patients with pituitary adenoma, who had undergone trans-sphenoidal adenectomy between January 2009 and January 2011, were reviewed. Patients underwent an ocular examination and brain MRI before and after surgery. The visual impairment score (VIS) was derived by combining the scores of best-corrected visual acuity and visual field. The relationship between VIS and tumor size/tumor type/position of the optic chiasm was assessed.

Pituitary adenomas reported an overall prevalence of 16.7% comprise a diverse group of tumors. Histologically, pituitary adenomas are considered to be benign. However, they may grow large and extend into surrounding structures resulting in neurological complications including visual impairment. If pituitary adenomas are not treated, vision will continue to deteriorate and blindness might result.

Typically, nonfunctioning adenomas present as macroadenomas that cause neurological symptoms due to intracranial mass effects since hormonal inactivity leads to a delay in diagnosis compared with functioning pituitary adenomas. It has been reported that 96.5% of nonfunctioning adenomas present as macroadenomas and that 67.8% of patients with these tumors experience visual defects. For nonfunctioning pituitary adenomas, neurosurgery is the treatment of choice.

Functioning pituitary adenomas that secrete prolactin account for 40 to 60% of pituitary adenomas. Treatment of these tumors can be begun with a dopamine-agonist such as bromocriptine. Surgical resection, usually with the trans-sphenoidal approach, should be considered for pituitary adenomasthat secrete prolactin and show rapid deterioration in visual function as well as adenomas that secrete adrenocorticotropic hormone, growth hormone, or thyroid-stimulating hormone. When medical and surgical treatments are unsuccessful, radiotherapy may be used post-operatively.

The study shows that seventy-eight patients were included (41 male, 37 female). Thirty-two (41%) patients experienced blurred vision or visual field defect as an initial symptom. Receiver operating characteristic curve analysis showed that tumors <2.2 cm tended to cause minimal or no visual impairment. Statistical analysis showed that 1) poor preoperative vision is related to tumor size, displacement of the optic chiasm in the sagittal view on MRI and optic atrophy, and 2) poorer visual prognosis is associated with greater preoperative VIS. In multivariate analysis the only factor significantly related to VIS improvement was increasing pituitary adenoma size, which predicted decreased improvement.

Results from this study show that pituitary adenomas larger than 2 cm cause defects in vision while adenomas 2 cm or smaller do not cause significant visual impairment. Patients with a large macroadenoma or giant adenoma should undergo surgical resection as soon as possible to prevent permanent visual loss.[86]

1.23.5 Prevalence of diabetes mellitus in patients with acromegaly.

Clemmons DR et al. performed a study to assess the prevalence of ECMDs (early carbohydrate metabolism disorder) and DM in patients with acromegaly undergoing treatment at a large tertiary referral centre in Moscow and to compare the results with the prevalence of such glucose disturbances in adults without acromegaly using two population-based surveys. A total of 97 patients with acromegaly undergoing treatment or long-term follow-up at the outpatient clinic of the Moscow Regional Clinical Research State Institute underwent an extensive evaluation, which included measurement of height, weight and blood pressure

Disturbances of glucose metabolism are frequently observed in patients with acromegaly. In one of the first papers to be published on this topic, abnormal glucose tolerance was found in over 60% of patients with acromegaly. The glucose anomalies in these patients are now known to include diabetes mellitus (DM), impaired glucose tolerance (IGT) and impaired fasting glucose (IFG) and have been discussed extensively in a review by Colao *et al*. Looking more specifically at early carbohydrate metabolism disorders (ECMDs) – defined as IFG, IGT or their combination- its prevalence in patients with acromegaly has been shown to vary between 16 and 46%. While most epidemiological studies have shown the prevalence of ECMDs to be higher than that of overt diabetes, not all studies report the same prevalence. The development of ECMDs and/or progression to diabetes in patients with acromegaly may depend on several

factors, such as age and gender, the levels of growth hormone (GH), as well as the duration of acromegaly and duration of exposure to elevated GH levels. However, other authors have found no differences in GH levels and insulin-like growth factor 1 (IGF1) levels or disease duration between those with glucose disturbances and those who were normoglycaemic. A further possible factor involved in the early development of diabetes is a positive family history of DM. A final factor that may also influence the development of glucose disturbances is the specific treatment for acromegaly. Somatostatin analogues may influence glucose metabolism both by lowering insulin secretion and by lowering GH and IGF1 levels.

In 24 patients with acromegaly, DM had previously been diagnosed during the course of their disease. Out of 24, six were being treated with a diet only, six with oral blood glucose lowering agents (either metformin or sulphonylurea), two with insulin alone and ten with insulin plus an oral agent (metformin or sulphonylurea). In 27 of the 73 patients, diabetes was diagnosed, resulting in a total number of 51 (52.5%) patients with DM. This prevalence was 3.5 times higher than the prevalence of T2DM in the general population (14.3%). In patients with acromegaly, DM as well as all disturbances in glucose metabolism is more prevalent when compared with the general population in all age groups. Only 28.8% of patients with acromegaly were proved to have normal glucose tolerance (NGT).[87]

1.23.6 Epidemiology and etiology of meningioma

Joseph wiemels et al. performed this study. In this study they were described about the epidemiology and etiology of meningioma. Although most meningiomas are encapsulated and benign tumors with limited numbers of genetic aberrations, their intracranial location often leads to serious and potentially lethal consequences. They are the most frequently diagnosed primary

brain tumor accounting for 33.8% of all primary brain and central nervous system tumors reported in the United States between 2002 and 2006. Inherited susceptibility to meningioma is suggested both by family history and candidate gene studies in DNA repair genes. People with certain mutations in the neurofibromatosis gene (*NF2*) have a very substantial increased risk for meningioma. Growing emphasis on brain tumor research coupled with the advent of new genetic and molecular epidemiologic tools in genetic and molecular epidemiology promise hope for advancing knowledge about the causes of intra-cranial meningioma.

Only a few epidemiologic studies of intracranial tumors to date have been adequately powered to study separately risk factors for meningioma. These include the large European cohorts such as the Interphone, and the Million Women Study in the United Kingdom. Several large European country- or region-specific case–control studies were spawned from the Interphone study. In 2002, The Benign Brain Tumor Cancer Registries Amendment Act (H.R. 5204) was passed, mandating registration of benign brain tumors such as meningioma in the United States. This legislation has and will continue to enhance reporting of both incidence rates and survival times for patients with meningioma. Before this act, meningioma mortality rate estimates were hampered by incomplete reporting and potential selection biases with respect to the individuals who were included in the databases, as well as limited follow-up information.

The prevalence of pathologically-confirmed meningioma is estimated to be approximately 97.5/100,000 in the United States with over 170,000 individuals currently diagnosed with this tumor. Since a proportion of meningiomas are not surgically managed, these estimates are low. In addition, autopsy and imaging studies have estimated subclinical meningioma rates of up to 2.8% in women. Data from the Central Brain Tumor Registry of the United States (CBTRUS) demonstrates a more than twofold higher incidence among females [age-adjusted incidence rate

(per 100,000 person years) of 8.36 and 3.61 for females and males, respectively]. The female: male ratio of approximately 2:1 may be inverted for rare pre-pubertal meningiomas. Atypical and malignant meningiomas comprise a small fraction of the total (~5%) and have a slight male predominance. Reported rates for Black Non-Hispanics are slightly higher (6.67) than for White Non-Hispanic and Hispanics (5.90 and 5.94, respectively).

Meningioma cells exhibit a striking similarity to arachnoid cap cells, which are the likely tumor cell of origin. Despite the fact that meningioma has a benign pathophysiology in 95% of cases, like carcinoma it always results from a clonal outgrowth derived from a single cell as exemplified by cytogenetic and array-comparative genomic hybridization (array-CGH) studies. Sporadic meningiomas are typically associated with one or more focal chromosomal deletion(s), and atypical and malignant grades tend to have multiple chromosomal copy number alterations consistent with the acquisition of "mutator" mutations which foster genomic instability. Deletion and inactivation of *NF2* on chromosome 22 is a predominant feature in sporadic meningiomas, and biallelic deletions are common. Additional genes are likely involved as well, since loss of *NF2* occurs in only 1/3 of patients who exhibit loss of heterozygosity of chromosome 22. Additional genomic regions which are recurrently lost in meningiomas include 14q, 1p, 6q, and 18q. Although in one study, familial meningiomas did not demonstrate inherited copy number alterations, such families typically have a germline defect in *NF2* or other predispositing mutations. Indeed, meningiomas are reported in families of several cancer predisposition syndromes including those involving the genes *NF1*, *PTCH*, *CREBBP*, *VHL*, *PTEN*, and *CDKN2A* (reviewed in. Epigenetic aberrations in meningioma have not been thoroughly assessed, but one study suggests that DNA methylation events may impact meningioma biology more significantly than DNA copy number mutations. Clearly, complexity of genetic aberrations

in meningioma increases with tumor grade. A relatively small number of mutations may be necessary for most meningiomas; however their slow growth makes long latency an issue, lending difficulty in identifying the source and timing of the initiating mutations, presenting a further complication for epidemiology studies.[88]

1.23.7 Intracranial hemorrhage in setting of glioblastoma with venous thromboembolism

Michael Nabil Khoury Performed a study to investigate relationship between intracranial hemorrhages in setting of glioblastoma with venous thromboembolisom. Venous thromboembolism (VTE) is a complication of glioblastoma. An Anticoagulating patient with glioblastoma carries a theoretical risk of intracranial hemorrhage (ICH). They performed a retrospective cohort study of consecutive glioblastoma patients (2007–2013) diagnosed with VTE.

The study population comprised of 523 glioblastoma patients of whom 173 (33%) had VTE events. Seventeen (10%) had ICH: 6 (35%) subdural hematomas and 11 (65%) intratumoral hemorrhages. In total, 4 patients with ICH required neurosurgical intervention. Enhancement in the area of subsequent intratumoral hemorrhage was noted in 9 of 10 with available pre-ICH scans. Multivariable regression did not show associations between ICH and tumor enhancement diameter or use of vascular-endothelial-growth-factor inhibitor. Fifteen (16%) patients receiving anticoagulation had ICH compared with 2 (2.6%) not receiving anticoagulation (P = .005). The method of anticoagulation was not associated with development of ICH. Median survival times from nondistal VTE diagnosis to death were 8.0 and 3.5 months (P = .05) in patients receiving anticoagulation and those not on anticoagulation, respectively.

Patients with glioblastoma and VTE on anticoagulation have increased incidence of ICH. However, development of ICH was not associated with lower median survival from time of VTE. Intratumoral hemorrhage occurred within the enhancing portion of tumor; however, no relationship was identified between the developments of ICH.[89]

Chapter Two

Materials and Methods

2. Methods:

To investigate the type of patients and treatment intervention in neurosurgery patients, the personal and clinical data was retrieved from the patient's profile at neurosurgery department under ***Bangabandhu Sheikh Mujib Medical University* (*BSMMU*)** during 1st April, 2015 to 28th October, 2015. Firstly researcher collected information from patient's profile which was kept in nurse room. For, further information, after explaining the purpose of the study to the respondents and obtaining their verbal consent, the researcher interviewed all the respondents by asking questions in Bengali and using a thoroughly pre-tested questionnaire.

The questionnaire contains patient's age, sex, diagnosis period of disease, hair color, height, weight, blood group, blood pressure, blood glucose level, smoking habit, surgery history, target organ of injury, diagnosis of patients, diagnosis impression, disease state, complications/associated disease, and medications.

The sample size was 190.

Data was entered into a computer database and doubled checked before analysis. Means and proportions for the patient's socio-demographic characteristics and treatment intervention were compared between the groups of the study (cases and controls).

Chapter Three

Results

Different types of diseases were found in patients of neurosurgery department. 22 types of diseases were found in patients. Among the 190 patients, meningioma were found in 32 (16.85%) patients, spinal stenosis were found in 30 (15.79%) patients, acromegaly were found in 21 (11.06%) patients, hydrocephalus were found in 19 (10%) patients, glioma were found in 16 (8.43%) patients, cervical myelopathy were found in 17 (8.95%) patients, arteriovenous malformation were found in 5 (2.64%) patients, haematoma were found in 3 (1.58%) patients, spondylolisthesis were found in 3 (1.58%) patients, osteophytes were found in 1 (0.53%) patient, cervical spondylosis were found in 6 (3.16%) patients, tuberculous spondylitis were found in 4 (2.11%) patients, lumbar spondylosis were found in 6 (3.16%) patients, Intracerebral haemorrhage were found in 3 (1.58%) patients, Aneurysm were found in 2 (1.06%) patients, chiari malformation were found in 2 (1.06%) patients, Sclerosis were found in 2 (1.06%) patients, schwannoma were found in 9 (4.74%) patients, medulloblastoma were found in 2 (1.06%) patients, glioblsatoma were found in 1 (0.53%) patient, craniopharyngioma were found in 2 (1.06%) patients, and astrocytoma were found in 4 (2.11%) patients. The list of different type of diseases and their prevalence are given in table 3.1.

Table 3.1: Prevalence of type of patients

Type of Diseases	Number Of Patients	Prevalence (%)
1. Cervical Myelopathy	**17**	**8.95%**
2. Spinal Stenosis	**30**	**15.79%**
3. Acromegaly	**21**	**11.06%**
4. Arteriovenous Malformation	**5**	**2.64%**
5. Haematoma	**3**	**1.58%**
6. Hydrocephalus	**19**	**10%**
7. Spondylolisthesis	**3**	**1.58%**
8. Osteophyte	**1**	**0.53%**
9. Cervical Spondylosis	**6**	**3.16%**
10. Tuberculous Spondylitis	**4**	**2.11%**
11. Lumbar Spondylosis	**6**	**3.16%**
12. Intracerebral Haemorrhage	**3**	**1.58%**
13. Aneurysm	**2**	**1.06%**

14. Chiari Malformation	**2**	**1.06%**
15. Multiple Sclerosis	**2**	**1.06%**
16. Meningioma	**32**	**16.85%**
17. Schwannoma	**9**	**4.74%**
18. Glioma	**16**	**8.43%**
19. Medulloblastoma	**2**	**1.06%**
20. Glioblastoma	**1**	**0.53%**
21. Craniopharyngioma	**2**	**1.06%**
22. Astrocytoma	**4**	**2.11%**

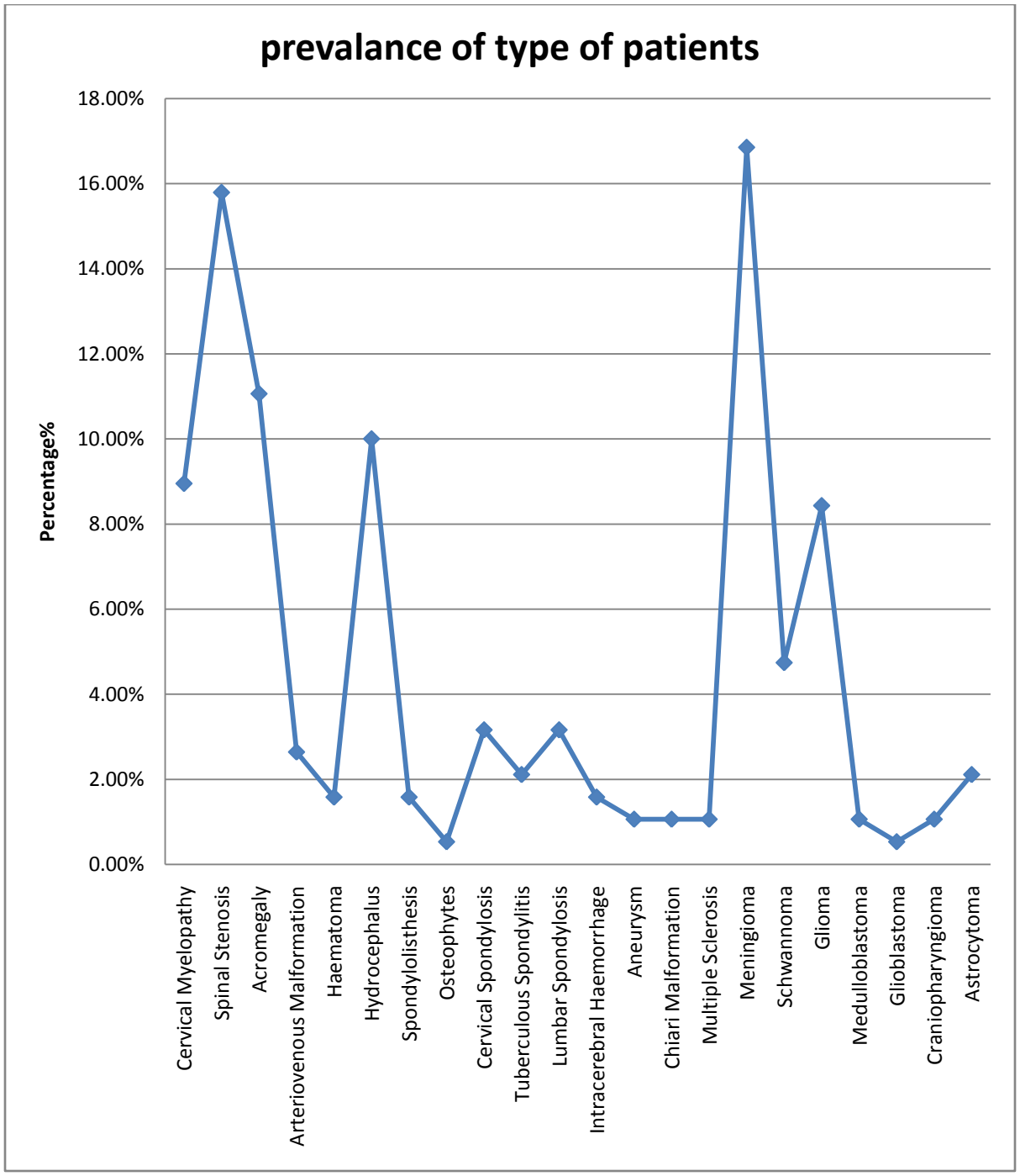

Figure 3.1: Prevalence of type of patients

The diseases were treated with different classes of medicine in neurosurgery department. Cervical myelopathy was treated with baclofen (10.42%), tolperisone hydrochloride (4.17%), clonazepam (6.25%), bromazepam (4.17%), pregabalin (2.09%), naproxen (2.09%), vitB1+vitB6+vitB12 (16.67%), and amitriptyline (2.09%). Spinal stenosis was treated with clonazepam (4%), Phenobarbital (1%), dexamethasone (1%), naproxen (4%), paracetamol (9%), vitB1+vitB6+vitB12 (20%), baclofen (1%), tolperisone hydrochloride (6%), amitryptylline (5%), diazepam (1%), diclofenac sodium (1%), pregabalin (5%), indomethacine (3%), tramadol (1%), fluphenazine hydrochloride+nortriptylene hydrochloride (2%), amikacin (1%), and flucloxacillin (4%). Acromegaly was treated with flucloxacillin (3.45%), ceftazimide pentahydrate (1.73%), prednisolone (1.73%), dexamethasone (6.90%), bromazepum (3.45%), hydrocortisone (8.63%), paracetamol (10.35%), clonazepam (13.80%), thyroxine Sodium (5.18%), vitB1+vitB6+vitB12 (5.18%), naproxen (1.73%), Phenobarbital (1.73%), pseudoephedrine+guniphensin+triprolidine (1.73%), and calcitriol (1.73%). Arteriovenous malformations were treated with phenytoin (6.25%), paracetamol (12.5%), flucloxacillin (6.25%), naproxen (6.25%), Phenobarbital (6.25%), clonazepam (12.5%), diclofenac (6.25%), dexamethasone (6.25%), and vitB1+vitB6+vitB12 (12.5%). Haematoma was treated with dexamethasone (25%), riboflavin (6.25%), phenobarbital (18.75%), diazepam (12.5%), flucloxacillin (12.5%), and naproxen (6.25%). Hydrocephalus was treated with dexamethasone (12.5%), phenytoin (2.09%), paracetamol (8.34%), flucloxacillin (8.34%), Phenobarbital (6.25%), clonazepam (6.25%), pregabalin (2.09%), amitriptyline (2.09%), prednisolone (2.09%), diclofenac (2.09%), and diazepam (2.09%). Spondylolisthesis was treated with naproxen (18.19%), diazepam (9.10%), vitB1+vitB6+vitB12 (18.19%), beclofen (9.10%), and diclofenac (9.10%). Osteophyte was treated with naproxen (50%), and omeprazole (50%). Cervical spondylosis was treated with paracetamol (5%), diazepam (5%),

vitB1+vitB6+vitB12 (25%), naproxen (10%), pregabalin (5%), aceclofenac (10%), and amoxicillin (5%). Tuberculous spondylitis was treated with rifampicin+isoniazid+pyrazinamide+ethambutol (20%), vitB1+vitB6+vitB12 (20%), naproxen (10%), tolperisone (10%), and paracetamol (10%). Lumbar spondylosis was treated with aceclofenac (5.89%), etoricoxib (5.89%), vitB1+vitB6+vitB12 (11.77%), pregabalin (5.89%), baclofen (11.77%), mecobalamin (5.89%), bromazepum (5.89%), and dexamethasone (5.89%). Intracerebral haemorrhage was treated with paracetamol (7.15%), bromazepum (14.29%), phenytoin (7.15%), Phenobarbital (21.43%), diclofenac (7.15%), dexamethasone (7.15%), and tramadol (7.15%). Aneurysm was treated with paracetamol (11.12%), naproxen (22.23%), dexomethasone (11.12%), Phenobarbital (22.23%), nimodipine (11.12%). Chiari malformations were treated with pregabalin (14.29%), (flupenthixol dihydrchloride & melitracen hydrochloride) (14.29%), paracetamol (14.29%), vitB1+vitB6+vitB12 (28.58%), and mecobalamin (14.29%). Multiple sclerosis was treated with paracetamol (16.67%), naproxen (16.67%), prednisolone (16.67%), and vitB1+vitB6+vitB12 (16.67%). Meningioma was treated with carbamazepine (2.60%), flucloxacillin (6.50%), dexamethasone (14.29%), Phenobarbital (7.80%), paracetamol (2.60%), diclofenac (2.60%), vitB1+vitB6+vitB12 (10.39%), baclofen (2.60%), clonazepam (5.20%), sodium valporate (2.60%), trifluoperazine (1.30%), sertraline (1.30%), ceftazimide pentahydrate (2.60%), amikacin (1.30%), naproxen (2.60%), pregabalin (1.30%), bromazepam (1.30%), prednisolone (1.30%), and amitriptylline (1.30%). Schwannoma was treated with naproxen (6.25%), cefixime trihydrate (6.25%), flucloxacillin (12.5%), vitB1+vitB6+vitB12 (25%), clonazepum (6.25%), and paracetamol (6.25%). Glioma was treated with dexamethasone (16.28%), paracetamol (13.96%), naproxen (4.66%), cefixime Trihydrate (2.33%), phenobarbital, diazepam (4.66%), diclofenac (2.33%), mannitol (2.33%), phenytoin (6.98%), and

vitB1+vitB6+vitB12 (4.66%). Medulloblatoma was treated with flucloxacillin (25%), Phenobarbital (25%), paracetamol (25%) and cefixime (25%). Glioblastoma was treated with dexamethasone (33.34%) and amitryptylline (33.34%). Craniopharyngioma was treated with dexamethasone (25%), hydrocortisone (12.5%), clonazepam (12.5%), and paracetamol (25%). Asrtocytoma was treated with vitB1+vitB6+vitB12 (18.19%), baclofen (9.10%), paracetamol (13.64%), dexamethasone (13.64%), Phenobarbital (4.55%), flucloxacillin (4.55%), piracetam (4.55%), and prednisolone (4.55%). The list of medicines that were prescribed by doctors in neurosurgery department is given in table 3.2.

Table 3.2: Medicine used in neurosurgery patients

Type of Diseases	**Medicine given**
	Brand name (Generic name) (No. of patients) (Percentage %)
1. Cervical Myelopathy	**Ciprocin (Ciprofloxacin) (1) (2.09%)** **Fluclox (Flucloxacillin) (2) (4.17%)** **Cotson (Hydrocortisone) (1) (2.09%)** **Torax (Ketorolac Tromethamine) (4) (8.34%)** **Rejubion (Vitamin B1+VitaminB6+Vitamin B12) (8) (16.67%)** **Beclo (Baclofen) (5) (10.42%)**

	Myolax (Tolperisone hydrochloride) (2) (4.17%) **Sandocal (Calcium Carbonate) (2) (4.17%)** **Epitra (Clonazepam) (3) (6.25%)** **Lexotanil (Bromazepam) (2) (4.17%)** **Pregaba (Pregabalin) (1) (2.09%)** **Pantonix (Pantoprazole) (9) (18.75%)** **Cefotil (Cefuroxime) (1) (2.09%)** **Sedil (diazepam) (1) (2.09%)** **Diproxen (Naproxen Sodium) (1) (2.09%)** **Cefazid (Ceftazimide Pentahydrate) (1) (2.09%)** **Amilin (Amitriptyline hydrochloride) (1) (2.09%)** **Napa (paracetamol) (2) (4.17%)** **Zithrox (Azithromycin) (1) (2.09%)**
2. Spinal Stenosis	**Barbit (Phenobarbital) (1) (1%)** **Oradexon (Dexamethasone) (1) (1%)** **Clonapin (Clonazepam) (4) (4%)**

	Rolac (Ketorolac tromethamine) (8) (8%) **Ometid (Omeprazole) (18) (18%)** **Naprosyn (Naproxen Sodium) (4) (4%)** **Napa (Paracetamol) (9) (9%)** **Neurobest (Vit B1+Vit B6+Vit B12) (20) (20%)** **Beclo (Baclofen) (1) (1%)** **Acefenac (Aceclofenac) (2) (2%)** **Myolax (Tolperisone hydrochloride) (6) (6%)** **Amilin (Amitryptyline) (5) (5%)** **Sedil (Diazepam) (1) (1%)** **Moxacil (Amoxycillin) (1) (1%)** **Voltalin (Diclofenac Sodium) (1) (1%)** **Pregaba (pregabalin) (5) (5%)** **Reumacap (Indomethacine) (3) (3%)** **Methicol (Mecobalamin) (1) (1%)** **Doloran (Tramadol) (1) (1%)**

	Bopam (Bromazepam) (1) (1%) **Norflu (Fluphenazine hydrochloride +Nortriptylene hydrochloride) (2) (2%)** **Fluclox (Flucloxacillin) (4) (4%)** **Kacin (Amikacin) (1) (1%)**
3. Acromegaly	**Fluclox (Flucloxacillin) (2) (3.45%)** **Cefazid (Ceftazimide Pentahydrate) (1) (1.73%)** **Cortan (Prednisolone) (1) (1.73%)** **Oradexon (Dexamethasone) (4) (6.90%)** **Rolac (Ketorolac Tromethamine) (2) (3.45%)** **Lexotanil (Bromazepam) (2) (3.45%)** **Cotson (Hydrocortisone Sodium) (5) (8.63%)** **Napa (Paracetamol) (6) (10.35%)** **Pantonix (Pantoprazole) (11) (18.97%)** **Disopan (Clonazepam) (8) (13.80%)** **Thyrox (Thyroxine Sodium) (3) (5.18%)**

	Orcef (Cefixime trihydrate) (4) (6.90%) **Neurobest (VitB1+VitB6+VitB12) (3) (5.18%)** **Naprosyn (Naproxen Sodium) (1) (1.73%)** **Barbit (Phenobarbital Sodium) (1) (1.73%)** **Sedil (Diazepam) (1) (1.73%)** **Tyrex (Pseudoephedrine+Guniphensin+Triprolidine) (1) (1.73%)** **Calcin (Calcium carbonate) (1) (1.73%)** **Rocaltrol (Calcitriol) (1) (1.73%)**
4.Arteriovenous Malformation	**Ometid (Omeprazole) (3) (18.75)** **Diphedan (Phenytoin) (1) (6.25%)** **Napa (Paracetamol) (2) (12.5%)** **Fluclox (Flucloxacillin) (1) (6.25%)** **Naprosyn (Naproxen Sodium) (1) (6.25%)** **Barbit (Phenobarbital Sodium) (1) (6.25%)** **Epitra (Clonazepam) (2) (12.5%)**

	Voltalin (Diclofenac Sodium) (1) (6.25%) **Cef 3 (Cefixime Trihydrate) (1) (6.25%)** **Oradexon (Dexamethasone) (1) (6.25%)** **Neurobest (VitB1+VitB6+VitB12) (2) (12.5%)**
5. Haematoma	**Ometid (Omeprazole) (2) (12.5%)** **Oradexon (Dexamethasone) (4) (25%)** **Riboflavin (Riboflavine) (1) (6.25%)** **Barbit (Phenobarbital Sodium) (3) (18.75%)** **Sedil (Diazepam) (2) (12.5%)** **Fluclox (Flucloxacillin) (2) (12.5%)** **Rolac (Ketorolac tromethamine) (1) (6.25%)** **Naprosyn (Naproxen Sodium) (1) (6.25%)**
6. Hydrocephalus	**Oradexon (Dexamethasone) (6) (12.5%)** **Diphedan (Phenytoin) (1) (2.09%)** **Napa (Paracetamol) (4) (8.34%)** **Fluclox (Flucloxacillin) (4) (8.34%)**

	Bextram (Multivitamin+Cod liver oil preparation) (2) (4.17%) **Cefazid (Ceftazimide Pentahydrate) (6) (12.5%)** **Barbit (Phenobarbital Sodium) (3) (6.25%)** **Kacin (Amikacin Sulphate) (1) (2.09%)** **Epitra (Clonazepam) (3) (6.25%)** **Fimoxyclav (Co-Amoxyclav) (1) (2.09%)** **Pregaba (Pregabalin) (1) (2.09%)** **Rolac (Ketorolac tromethamine) (2) (2.09%)** **Maxpro (Esomeprazole) (9) (18.75%)** **Amilyn (Amitriptyline) (1) (2.09%)** **Cortan (Prednisolone) (1) (2.09%)** **Difen (Diclofenac) (1) (2.09%)** **Ronem (Meropenem Trihydrate) (1) (2.09%)** **Sedil (Diazepam) (1) (2.09%)**
7. Spondylolisthesis	**Naprosyn (Naproxen Sodium) (2) (18.19%)** **Sedil (Diazepam) (1) (9.10%)**

	Maxpro (Esomeprazole) (3) (27.28%) **Neurobest (VitB1+VitB6+VitB12) (2) (18.19%)** **Beklo (Beclofen) (1) (9.10%)** **Voltalin (Diclofenac Sodium) (1) (9.10%)** **Rolac (Ketorolac tromethamine) (1) (9.10%)**
8. Osteophyte	**Naprosyn (Naproxen Sodium) (1) (50%)** **Ometid (Omeprazole) (1) (50%)**
9. Cervical Spondylosis	**Napa (Paracetamol) (1) (5%)** **Sedil (Diazepam) (1) (5%)** **Maxpro (Esomeprazole) (5) (25%)** **Neurobest (VitB1+VitB6+VitB12) (5) (25%)** **Naprosyn (Naproxen Sodium) (2) (10%)** **Rolac (Ketorolac tromethamine) (1) (5%)** **Pregaba (Pregabalin) (1) (5%)** **Reservix (Aceclofenac) (2) (10%)** **Clamox (Amoxycillin) (1) (5%)**

	Bopam (Bromazepam) (1) (5%)
10. Tubercular **Spondylitis**	**Rimstar4-FDC** **(Rifampicin+Isoniazid+Pyrazinamide+Ethambutol) (2) (20%)** **Pyrovit (Pyridoxine hydrochloride) (1) (10%)** **Neurobest (VitB1+VitB6+VitB12) (2) (20%)** **Rolac (Ketorolac tromethamine) (1) (10%)** **Naprosyn (Naproxen Sodium) (1) (10%)** **Maxpro (Esomeprazole) (1) (10%)** **Myolax (Tolperisone) (1) (10%)** **Napa (Paracetamol) (1) (10%)**
11.Lumbar Spondylosis	**Rolac (Ketorolac tromethamine) (3) (17.65%)** **Ometid (Omeprazole) (3) (17.65%)** **Reservix (Aceclofenac) (1) (5.89%)** **Etorex (Etoricoxib) (1) (5.89%)** **Neurobest (VitB1+VitB6+VitB12) (2) (11.77%)** **Pregaba (Pregabalin) (1) (5.89%)**

	Beclo (Baclofen) (2) (11.77%) **Nervex (mecobalamin) (1) (5.89%)** **Laxotanil (Bromazepam) (1) (5.89%)** **Oradexon (Dexamethasone) (1) (5.89%)** **Ciprocin (Ciprofloxacin) (1) (5.89%)**
12.Intracerebral Haemorrhage	**Ometid (Omeprazole) (3) (21.43%)** **Napa (Paracetamol) (1) (7.15%)** **Laxotanil (Bromazepam) (2) (14.29%)** **Diphedin (Phenytoin) (1) (7.15%)** **Barbit (Phenobarbital Sodium) (3) (21.43%)** **Voltalin (Diclofenac) (1) (7.15%)** **Oradexon (Dexamethasone) (1) (7.15%)** **Rolac (Ketorolac tromethamine) (1) (7.15%)** **Anadol (Tramadol hydrochloride) (1) (7.15%)**
13. Aneurysm	**Napa (Paracetamol) (1) (11.12%)** **Naprosyn (Naproxen Sodium) (2) (22.23%)**

	Pantid (Pantoprazole) (2) (22.23%) **Decason (Dexomethasone Sodium) (1) (11.12%)** **Barbit (Phenobarbital Sodium) (2) (22.23%)** **Nimocal (Nimodipine) (1) (11.12%)**
14.Chiari Malformation	**Pregaba (Pregabalin) (1) (14.29%)** **Deleta (Flupenthixol dihydrchloride & Melitracen hydrochloride) (1) (14.29%)** **Napa (Paracetamol) (1) (14.29%)** **Maxpro (Esomeprazole) (1) (14.29%)** **Solbion (VitB1+VitB6+VitB12) (2) (28.58%)** **Mecol (Mecobalamin) (1) (14.29%)**
15. Multiple Sclerosis	**Napa (Paracetamol) (1) (16.67%)** **Maxpro (Esomeprazole) (2) (33.34%)** **Naprosyn (Naproxen Sodium) (1) (16.67%)** **Cortan (Prednisolone) (1) (16.67%)** **Neurocare (VitB1+VitB6+VitB12) (1) (16.67%)**

16. Meningioma	**Zeptol (Carbamazepine) (2) (2.60%)** **Ometid (Omeprazole) (16) (20.78%)** **Fluclox (Flucloxacillin) (5) (6.50%)** **Rolac (Ketorolac tromethamine) (3) (3.90%)** **Oradexon (Dexamethasone) (11) (14.29%)** **Barbit (Phenobarbital Sodium) (6) (7.80%)** **Napa (Paracetamol) (2) (2.60%)** **Voltalin (Diclofenac sodium) (2) (2.60%)** **Solbion (VitB1+VitB6+VitB12) (8) (10.39%)** **Beclo (Baclofen) (2) (2.60%)** **Calbo (Calcium carbonate) (1) (1.30%)** **Pase (Clonazepum) (4) (5.20%)** **Epilim (Sodium valporate) (2) (2.60%)** **Telazine (Trifluoperazine) (1) (1.30%)** **Sertal (Sertraline) (1) (1.30%)** **Cefazid (Ceftazimide Pentahydrate) (2) (2.60%)**

	Kacin (Amikacin) (1) (1.30%) **Naprosyn (Naproxen Sodium) (2) (2.60%)** **Aristovit-B (VitB Complex) (3) (3.90%)** **Atropine (Atropine Sulphate) (1) (1.30%)** **Pregaba (Pregabalin) (1) (1.30%)** **Lexotanil (Bromazepam) (1) (1.30%)** **Cortan (prednisolone) (1) (1.30%)** **Denvar (Cefixime Trihydrate) (3) (3.90%)** **Amilyn (Amitriptyline hydrochloride) (1) (1.30%)**
17. Schwannoma	**Naprosyn (Naproxen Sodium) (1) (6.25%)** **Orcef (Cefixime trihydrate) (1) (6.25%)** **Esotid (Esomeprazole) (3) (18.75%)** **Cefazid (Ceftazimide pentahydrate) (2) (12.5%)** **Fluclox (Flucloxacillin) (2) (12.5%)** **Neurobest (VitB1+VitB6+VitB12) (4) (25%)** **Rivotril (Clonazepam) (1) (6.25%)**

	Becosules (VitB Complex) (1) (6.25%) **Renova (Paracetamol) (1) (6.25%)**
18. Glioma	**Ometid (Omeprazole) (8) (18.61%)** **Oradexon (Dexamethasone) (7) (16.28%)** **Renova (Paracetamol) (6) (13.96%)** **Ciprozid (Ciprofloxacin) (1) (2.33%)** **Naprosyn (Naproxen Sodium) (2) (4.66%)** **Denvar (Cefixime Trihydrate) (1) (2.33%)** **Barbit (Phenobarbital Sodium) (5) (11.63%)** **Sedil (Diazepam) (2) (4.66%)** **Voltalin (Diclofenac Sodium) (1) (2.33%)** **Fluclox (Flucloxacillin) (2) (4.66%)** **Rolac (Ketorolac tromethamine) (2) (4.66%)** **Manisol (Mannitol) (1) (2.33%)** **Diphedin (Phenytoin) (3) (6.98%)** **Neurobest (VitB1+VitB6+VitB12) (2) (4.66%)**

19. Medulloblastoma	**Fluclox (Flucloxacillin) (1) (25%)** **Barbit (Phenobarbital Sodium) (1) (25%)** **Napa (Paracetamol) (1) (25%)** **T-Cef (Cefixime) (1) (25%)**
20. Glioblastoma	**Oradexon (Dexamethasone) (1) (33.34%)** **Amilyn (Amitryptyline hydrochloride) (1) (33.34%)** **Pantonix (Pantoprazole) (1) (33.34%)**
21. Craniopharyngioma	**Oradexon (Dexamethasone) (2) (25%)** **Pantonix (Pantoprazole) (2) (25%)** **Cotson (Hydrocortisone sodium succinate) (1) (12.5%)** **Pase (Clonazepam) (1) (12.5%)** **Napa (Paracetamol) (2) (25%)**
22. Astrocytoma	**Pantonix (Pantoprazole) (4) (18.19%)** **Neurobest (VitB1+VitB6+VitB12) (4) (18.19%)** **Flexibac (Baclofen) (2) (9.10%)** **Napa (Paracetamol) (3) (13.64%)**

	Oradexon (Dexamethasone) (3) (13.64%) **Barbit (Phenobarbital Sodium) (1) (4.55%)** **Fluclox (Flucloxacillin) (1) (4.55%)** **Neurolep (Piracetam) (1) (4.55%)** **Methicol (mecobalamine) (1) (4.55%)** **Cortan (Prednisolone) (1) (4.55%)** **Cef-3 (Cefixime) (1) (4.55%)**

Other diseases were associated with major disesase in patients of neurosurgery department. Diabetes (25%) and LBP (75%) were present in 4 patients of cervical myelopathy. Hypercholesterolaemia (6.67%), diabetes (33.34%), HBP (13.4%) and LBP (46.67%) were present in 15 patients of spinal stenosis. Asthma (7.15%), diabetes (64.29%), HBP (7.15%), and diarrhoea (7.15%) were present in 12 patients of acromegaly. Diabetes (33.34%) and LBP (66.67%) were present in 3 patients of arteriovenous malformations. LBP (33.34%), Constipation (33.34%) and diabetes (33.34%) were present in 3 patients of haematoma. Diarrhoea (40%), HBP (40%) and fever (20%), were present 5 in patients of hydrocepcephalus. Diabetes (50%) and LBP (50%) were present in 2 patients of spondylolisthesis. LBP was present in 1 patient of osteophytes. Diabetes (50%) and HBP (50%) were present in 2 patients of cervical spondylosis. Diabetes (50%) and LBP (50%) were present in 2 patients of lumbar spondylosis. Fever (33.34%) and LBP (66.67%) were present in 3 patients of tuberculous spondylitis. Fever

(33.34%) and LBP (66.67%) were present in 3 patients of intracerebral haemorrhage. LBP (50%) and fever (50%) were present in 2 patients of aneurysm. LBP (50%) and HBP (50%) were present in 2 patients of sclerosis. Epilepsy (33.34%), HBP (33.34%) and schizophrenia (33.34%) were present in 3 patients of meningioma. Diabetes (50%) and LBP (50%) were present in 2 patients of schwannoma. Diabetes (28.58%), HBP (28.58%), LBP (28.58%), and allergy (14.29%) were present in 7 patients of glioma. LBP (50%) and diabetes (50%) were present in 2 patients of medulloblastoma. LBP was present in 1 patient of glioblastoma. Anxiety (50%) and LBP (50%) were present in 2 patients of craniopharyngioma. LBP (66.67%) and constipation (33.34%) were present in 3 patients of astrocytoma. Few symptoms such as headache, vomiting, nausea, fatigue and weakness were also associated with patients. The medicines such as paracetamol, metformin, lovastatin, ramipril, domperidone, metronidazole, glimepiride, sertraline, and fexofenadine were used to treat associated diseases and symptoms. The list of associated diseases, symptoms and their medicines are given in table 3.3.

Table 3.3: Associated diseases in concomitant with major disease

Major Diseases	Associated diseases (No. of patients) (Percentage %)	Associated symptoms (No. of patients) (Percentage %)	Medicines for associated diseases and symptoms (No. of patients) (Percentage %)
Cervical Myelopathy	Diabetes (1) (25%) LBP (3) (75%)	Headache (1) (33.34%) Weakness (2) (66.67%)	1. Paracetamol (1) (50%) 2. Metformin (1) (50%)
Spinal Stenosis	Diabetes (5) (33.34%) LBP (7) (46.67%) Hypercholesterolaemia (1) (6.67%) HBP (2) (13.4%)	Headache (3) (75%) Weakness (1) (25%)	1. Metformin (4) (36.37%) 2. Indapamide (1) (9.10%) 3. Lovastatin (1) (9.10%) 4. Paracetamol (3) (27.28%) 5. Ramipril (2) (18.19%)
Acromegaly	Diabetes (9) (64.29%) Asthma (1) (7.15%) HBP (1) (7.15%) Diarrhoea (1) (7.15%) LBP (2) (14.29%)	Vomiting (3) (42.86%) Headache (4) (57.15%)	1. Ramipril (2) (11.77%) 2. Dexamethasone (1) (5.89%) 3. Paracetamol (3) (17.65%) 4. Domperidone (3) (17.65%) 5. Metformin (7) (41.18%)

			6. Metronidazole (1) (5.89%)
Arteriovenous Malformation	**Diabetes (1) (33.34%)** **LBP (2) (66.67%)**	**Headache (1) (33.34%)** **Weakness (2) (66.67%)**	**1. Paracetamol (1) (50%)** **2. Insulin (Actrapid) (1) (50%)**
Haematoma	**LBP (1) (33.34%)** **Diabetes (1) (33.34%)** **Constipation (1) (33.34%)**	**Headache (1) (50%)** **Tiredness (1) (50%)**	**1. Lactulose (1) (33.34%)** **2. Paracetamol (1) (33.34%)** **3. Glimepiride (1) (33.34%)**
Hydrocephalus	**HBP (2) (40%)** **Diarrhoea (2) (40%)** **Fever (1) (20%)**	**Headache (4) (50%)** **Vomting (4) (50%)**	**1. Paracetamol (5) (38.47%)** **2. Domperidone (4) (30.77%)** **3. Ramipril (2) (15.39%)** **4. Metronidazole (2) (15.39%)**
Spondylolisthesis	**Diabetes (1) (50%)** **LBP (1) (50%)**	**Weakness (1) (50%)** **Vomiting (1) (50%)**	**1. Metformin (1) (50%)** **2. Domperidone (1) (50%)**
Osteophyte	**LBP (1) (100%)**	**Vomting (1) (100%)**	**1. Domperidone (1) (100%)**

Cervical Spondylosis	**Diabetes (1) (50%)** **HBP (1) (50%)**	**Weakness (1) (50%)** **Fatigue (1) (50%)**	**1. Glimepiride (1) (50%)** **2. Ramipril (1) (50%)**
Tubercular Spondylitis	**Fever (1) (33.34%)** **LBP (2) (66.67%)**		**1. Paracetamol (1) (100%)**
Lumbar Spondylosis	**Diabetes (1) (50%)** **LBP (1) (50%)**	**Vomiting (1) (50%)** **Weakness (1) (50%)**	**1. Metformin (1) (50%)** **2. Domperidone (1) (50%)**
Intracerebral Haemorrhage	**Fever (1) (33.34%)** **LBP (2) (66.67%)**	**Vomiting (1) (33.34%)** **Weakness (2) (66.67%)**	**1. Ondansteron (1) (50%)** **2. Paracetamol (1) (50%)**
Aneurysm	**LBP (1) (50%)** **Fever (1) (50%)**	**Headache (1) (50%)** **Vomiting (1) (50%)**	**1. Paracetamol (1) (50%)** **2. Domperidone (1) (50%)**
Multiple Sclerosis	**LBP (1) (50%)** **HBP (1) (50%)**	**Vomiting (1) (50%)** **Tiredness (1) (50%)**	**1. Domperidone (1) (50%)** **2. Losartan (1) (50%)**
Meningioma	**HBP (1) (33.34)** **Epilepsy (1) (33.34)** **Schizophrenia (1) (33.34)**	**Headache (8) (72.73%)** **Vomiting (3) (27.28%)**	**1. Paracetamol (8) (57.15%)** **2. Sodium valporate (1) (7.15%)** **3. Domperidone (3) (21.43%)**

			4. Losartan (1) (7.15%) **5. Sertraline (1) (7.15%)**
Schwannoma	**Diabetes (1) (50%)** **LBP (1) (50%)**	**Headache (3) (75%)** **Vomiting (1) (25%)**	**1. Paracetamol (3) (60%)** **2. Domperidone (1) (20%)** **3. Metformin (1) (20%)**
Glioma	**Diabetes (2) (28.58%)** **HBP (2) (28.58%)** **LBP (2) (28.58%)** **Allergy (1) (14.29%)**	**Headache (3) (60%)** **Vomiting (2) (40%)**	**1. Paracetamol (3) (30%)** **2. Metformin (2) (20%)** **3. Domperidone (2) (20%)** **4. Losartan potassium (2) (20%)** **5. Fexofenadine (1) (100%)**
Medulloblastoma	**LBP (1) (50%)** **Diabetes (1) (50%)**	**Headache (1) (50%)** **Weakness (1) (50%)**	**1. Paracetamol (1) (50%)** **2. Metformin (1) (50%)**
Glioblastoma	**LBP (1) (100%)**	**Headache (1) (100%)**	**1. Paracetamol (1) (50%)**
Craniopharyngioma	**Anxiety (1) (50%)** **LBP (1) (50%)**	**Headache (1) (50%)** **Nausea (1) (50%)**	**1. Paracetamol (1) (50%)** **2. Amitryptyline (1) (50%)**

Astrocytoma	**Constipation (1) (33.34%)** **LBP (2) (66.67%)**	**Headache (1) (50%)** **Weakness (1) (50%)**	**1. Paracetamol (1) (50%)** **2. Lactulose (1) (50%)**

Chapter Four

Discussion

Discussion:

This prospective study was conducted with 190 patients at **Bangabandhu Sheikh Mujib Medical University (BSMMU).**

This study shows that the 22 types of diseases were found in 190 patients.

The patients were admitted in neurosurgery department for the treatment of different type of diseases e.g. cervical myelopathy, spinal stenosis, acromegaly, arteriovenous malformations, haematoma, hydrocephalus, spondylolisthesis, osteophytes, cervical spondylosis, tubercular spondylitis, lumbar spondylosis, intracerebral haemorrhage, aneurysm, chiari malformations, sclerosis, meningioma, schwannoma, glioma, medulloblastoma, glioblastoma, craniopharyngioma, and astrocytoma.

The study shows that meningioma were found in 32 (16.85%) patients, spinal stenosis were found in 30 (15.79%) patients, acromegaly were found in 21 (11.06%) patients, hydrocephalus were found in 19 (10%) patients, glioma were found in 16 (8.43%) patients, cervical myelopathy were found in 17 (8.95%) patients, arteriovenous malformation were found in 5 (2.64%) patients, haematoma were found in 3 (1.58%) patients, spondylolisthesis were found in 3 (1.58%) patients, osteophytes were found in 1 (0.53%) patients, cervical spondylosis were found in 6 (3.16%) patients, tubercular spondylitis were found in 4 (2.11%) patients, lumbar spondylosis were found in 6 (3.16%) patients, Intracerebral haemorrhage were found in 3 (1.58%) patients, Aneurysm were found in 2 (1.06%) patients, chiari malformation were found in 2 (1.06%) patients, Sclerosis were found in 2 (1.06%) patients, schwannoma were found in 9 (4.74%) patients, medulloblastoma were found in 2 (1.06%) patients, glioblsatoma were found in 1

(0.53%) patients, craniopharyngioma were found in 2 (1.06%), and astrocytoma were found in 4 (2.11%) patientss.

This study shows that patients of meningioma (16.85%), spinal stenosis (15.79%), acromegaly (11.06%), hydrocephalus (10%), cervical myelopathy (8.95%), and glioma (8.43%) were found in the most of the patients.

This study also shows that meningioma had occurred in most of the patients.

Meningioma is one type of brain tumor. The term “brain tumours” refers to a mixed group of neoplasms originating from intracranial tissues and the meninges with degrees of malignancy ranging from benign to aggressive. Approximately 4400 people are newly diagnosed with a brain tumour each year in the UK.[90]

One study shows that the overall prevalence rate of individuals with a nonmalignant brain tumor was 209.0 per 100 000 in 2004 and 221.8 per 100 000 in 2010. The averaged prevalence rate for malignant tumors was 166.5 per 100 000.[90]

Another study shows that the prevalence of pathologically-confirmed meningioma is estimated to be approximately 97.5/100,000 in the United States with over 170,000 individuals.[91]

The study shows that few drugs such as clonazepum, phenytoin, diazepam, amitriptyline, dexamethasone, naproxen, phenobarbital, pregabalin, flucloxacillin, esomeprazole, ketotorolac tromethamine, paracetamol, and vitB1+vitB6+vitB12 were common for most of the patients in neurosurgery department.

Although, Surgery was the only treatment for most of the patients, different classes of medicines were used to treat the patients. These medicines were mainly used to relieve few symptoms or to relieve surgical compications.

This study shows that the diseases such as diabetes, hypertension, low blood pressure, hypercholesterolaemia, asthma, allergy, diarrhoea, fever and schizophrania were associated with major diseases in patients. Few symptoms such as headache, vomiting, nausea, fatigue and weakness were also associated.

The study shows that diabetes, hypertension, and low blood pressure were associated with most of the patients. Diabetes was mainly associated with acromegaly. Among the 21 patients, diabetes was present in 9 patients.

One study shows that Disturbances of glucose metabolism was frequently observed in patients with acromegaly. In one of the first papers to be published on this topic, abnormal glucose tolerance was found in over 60% of patients with acromegaly. The glucose anomalies in patients are now known to include diabetes mellitus (DM), impaired glucose tolerance (IGT) and impaired fasting glucose.[92]

The study also shows that among the 30 patients, diabetes was present in 5 patients.

One study shows that diabetes mellitus was associated with spinal stenosis in patients.[93]

The survey result also shows that the drugs such as paracetamol, metformin, lovastatin, ramipril, domperidone, metronidazole, glimepiride, sertraline, and fexofenadine were used to treat associated diseases and symptoms.

Conclusion:

Neurosurgery is the specialized field of surgery that treats diseases that affect the CNS. The field of neurosurgery is one of the most sophisticated surgical specialties and encompasses advanced surgical and imaging technology and new research in molecular neurosurgery and gene therapy. The World Health Organization estimated in 2006 that neurological disorders and their sequelae affect as many as one billion people worldwide. The objectives of this study were to find out the type of patient and treatment intervention of neurosurgery patients.The study describes the different type of diseases with their causes, pathology, pathophysiology, treatment and their toxicities. The study result represents the different type of diseases and the prevalence of type of patients in neurosurgery department. The study also represents the list of medicines with their percentage that were used in neurosurgery patients. Other diseases were associated with major diseases in patients. The study also represents the associated diseases and symptoms with their treatment intervention.

Neurosurgical diseases have been ignored by governmental and private health organizations in developing countries. Neurosurgery in developing countries can be promoted if the first working neurosurgeons take up their responsibilities as pioneers. This role requires that they initiate the training of young neurosurgeons as soon as possible and that they find in the local conditions the necessary factors to promote neurosurgery and to integrate it into the health care development of their country.

Chapter Five

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Questionnaire

Name of the experiment: A Cross sectional study on the type of patients and treatment intervention in a neurosurgery department of a tertiary hospital in Bangladesh.

Patient name: Age: Sex:

Diagnosis period: Hair color: Height: Weight:

Blood group: Blood pressure: Diabetes:

Smoking habit: Alcoholic:

Previous Stroke History: Number of surgery/Types of injury/disease:

Target organ of injury: Use of tradition/folk medicines:

Cause of injury/disease:

Genetic background of injury/Family history of particular injury:

Probable reasons/diagnosis/pathology/pathophysiology:

1.

2.

3.

4.

5.

6.

Complications/associated disease:

1.

2.

3.

4.

5.

6.

7.

8.

9.

Drugs/Prescription patterns:

Brand name	Generic name