

University of Khartoum
Faculty of Medicine
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The Clinical Pattern and Etiology of Non Traumatic paraplegia

By

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Dedication

To My brother Salah

To My family

To my teachers

To my colleagues

&

To All patients with paraplegia

Acknowledgement

I would like to thank Dr Abbashar Hussein and Dr Ammar Al Tahir for their great supports and invaluable efforts and advice during all the research steps. My great thanks to Dr. Dalia, Dr. Mussab, Dr. Husham and Dr. Naglaa, and the working team in ministry of health, Khartoum state, the researches and training unit. All thanks to those patients who are willingly and generously were included in the study, hoping to enjoy happy life and full recovery. Finally my thanks to the working staff in the neurology ward in Al Shaab hospital.

Abstract

Worldwide paraplegia is a major disabling health problem . Its importance is due to the incapacitating disability that often persist despite treatment .

It encompasses a large range of disease entities ranging from demyelination, infection, nutritional, toxic, heredo-familial to degenerative conditions.

This disease has particularly invoked great interest among neurologists as it strikes apparently healthy individuals in the prime of their lives, who are left with variable degree of sequelae .

The study tried to identify the various etiologies , the ways of presentation and helpful diagnostic tools to each .

The study took place in El Shaab teaching hospital in the year 2002-2003 . Patients included in the study are those who present to the hospital or referred to the neurologist , with LL weakness.

100 patients were included in the study . 56% of whom are males and 44% are females , and originally from different states of the country and different racial groups .

Complete paralysis was detected in 56% of the study patients, 22% are in need of support and 22% do well independently .

Guillian Barre (19%) , Transverse myelitis (15%) , Potts disease of the spine (12%) , primary (3%) and secondary (13%) tumors constituted the etiological bulk of paraplegia in the study .

Imaging studies ; MRI was done to 78 patients seventy four of them were found to have abnormal images , X-ray was done to 69 patients thirty six of them were found to have abnormal image .

Laboratory tests including the full blood count, E.S.R, urea and electrolytes, blood glucose and urinalysis were done to all the patients. Liver chemistry, B12, bone marrow and other investigations were done. Cerebrospinal fluid analysis was abnormal in all patients with multiple sclerosis, 18 patients with acute inflammatory demyelinating polyneuropathy 9 patients with acute transverse myelitis and two patients with chronic inflammatory demyelinating polyneuropathy.

ملخص الأطروحة

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List of figures and tables

	Page
Table -1	52
Table -2	53
Table-3	54
Table-4	55
Table-5	56
Table-6	57
Table-7	58
Table-8	59
Figure -1	60
Figure -2	61
Figure -3	62
Figure -4	63
Figure -5	64
Figure -6	65
Figure -7	66
Figure -8	67
Figure -9	68
Figure -10	69
Figure -11	70
Figure -12	71
Figure -13	72
Figure -14	73

Abbreviations

A.D.E.M	Acute Disseminated Encephalomyelitis.
A.I.D.P	Acute Inflammatory Demyelinating. Poly neuropathy .
A.M.A.N	Acute Motor Axonal Neuropathy.
A.T.M	Acute Transverse Myelitis
C.E.S	Cauda Equina Syndrome.
C.I.D.P	Chronic Inflammatory Demyelinating. Poly neuropathy .
C.S.F	Cerebro Spinal Fluid.
H.S.P	Hereditary Spastic Paraparesis .
L.M.N.L	Lower Motor Neuron Lesion.
M.S	Multiple Sclerosis.
M.N.D	Motor Neuron Disease.
N.C.S	Nerve Conduction Study .
T.S.P	Tropical Spastic Paraparesis .

Paraplegia

Definition:-

Paraplegia is paralysis of the lower half of the body and legs , caused by an insulting injury mostly to the spinal cord , anterior horn cells , or the nerves supplying the lower limbs .

Paralysis is either flaccid , when it is due to lower motor neuron type or acute upper motor neuron type , or spastic due to chronic cord lesion

Small lesion can produce paraplegia or quadriplegia and sensory deficit below the involved segment , while with partial lesion both sensory and motor deficits are incomplete , as in Brown Sequard or hemi section of the cord .⁽¹⁾

Classification :

The onset of paralysis is either acute , subacute , or chronic .

Acute and subacute :- sudden or rapidly advancing neurological weakness of the LL presents as an urgent medical emergency .

The paralysis may be flaccid , the tendon reflex absent and the planter response is unobtainable . A sensory level on the trunk is strong evidence of spinal cord lesion , which may be ;

1- Intrinsic ; caused by viral myelitis , demyelinating disease :

M.S , A.D.E.M, Devic disease , S.L.E , sarcoidosis or cord infarction . A complete cord lesion is more likely to be due to an

isolated episode of myelitis than M.S , where the lesion is incomplete .⁽²⁾

2- Spinal cord compression ; due to the following ;- epidural abscess or hematoma , cervical spondylotic myelopathy spontaneous dislocation of the cervical vertebrae in R.A , arachnoiditis or tumors primary and metastatic .⁽³⁾

ii- Chronic and progressive paraplegia ; the lesion is also intrinsic or due to compression .

1- Intrinsic ; among which is the progressive spinal form of M.S , M.N.D , F.S.P , T.S.P and AIDS myelopathy, in addition to subacute combined degeneration of the cord , and syringomyelia .

2- Cord compression ; commonly due to benign cause like cervical spondylotic myelopathy , prolapsed dorsal disc , neurofibroma , meningioma and A-V malformation . Chronic epidural abscess is usually tuberculous . Malignant causes include myeloma , chordoma and glioma . Sensory symptoms usually precede weakness , root pain may be present . Motor involvement is usually symmetrical , and the degree of sphincters involvement is variable .⁽³⁾

Upper motor neuron lesion;

Characterized by spasticity , brisk reflexes often with ankle and / or knee clonus and up going planter response It is an evidence of cord injury

Lower motor neuron lesion ;

Characterized by flaccidity , absent or diminished reflexes and an unobtainable planter response .

Anatomy of the spinal cord :-

The spinal cord is a cylinder , some what flattened from front to back , whose lower end tapers into a cone .

The spinal cord posses two symmetrical enlargements which occupy the segments of the limbs plexus as the cervical enlargement (C5 – T1) and lumbosacral enlargement (L2- S3) .

The anterior and posterior roots unite within the intervertebral foramina . With in the subarachnoid space the nerve roots are attached to the spinal cord by rootlets. They evaginate the dura separately before uniting to form the mixed spinal nerve . ⁽⁴⁾

Below the first lumbar vertebra , the root pass almost vertically down ward through the subarachnoid space forming the cauda equina .The filum terminal (pia matter) extends down from the tip of the conus medullaris among the nerve roots of the cauda .

The spinal cord consists of a central mass of grey matter – cell bodies , enclosed in a cylindrical mass of white matter – fibers .

Afferent pathways : There are three destinations for all incoming fibers , the cortex of the opposite cerebral hemisphere via thalamic relay for consciousness sensation , the cerebellar for muscular coordination and the brain stem or spinal cord for reflex arc

Efferent pathways : For the control of the skeletal muscles , supplied by spinal nerves . Two main systems of neurons are

involved ; the direct corticospinal tract , consisting of two groups of neurons , the cell bodies of the first are in the cerebral cortex and their fibers ascend through the internal capsule , brain stem and to the anterior horn cells . The second neurons are the anterior horn cells and their axons which end as the motor end plates on skeletal muscle fibers .

The most important ascending tracts fall into two groups ; those in the lateral and anterior white columns are concerned with pain , temperature , crude touch and sensation of itch , tickle and sexual orgasm

The posterior white column is wholly occupied by ascending fibers of the gracile and cuneate tracts .They are concerned with light touch , vibration sensation and proprioception and the sensation of fullness of fullness in the bladder and rectum . the fibers from the lower parts of the body lie nearest the midline and incoming fibers added progressively laterally . ⁽⁴⁾

Blood supply :

The spinal cord is supplied by the single anterior , and (right and left) posterior arteries which ascend from the level of the foramen magnum , supplemented at variable levels by anastomosis of variable numbers of radicular arteries . ⁽⁵⁾

The anterior spinal artery is a midline vessel formed at the foramen magnum by the union of the two anterior spinal branches , each given off by the vertebral artery above .

Although it is usually larger than the posterior arteries , and runs

the whole length of the cord , it may become so small in places especially in the thoracic region , that it may be considered absent . It supplies the whole cord anterior to the posterior grey columns . ⁽⁵⁾

The posterior spinal artery on each side arises from the posterior inferior cerebellar or vertebral artery . It is usually double and there is sometimes anastomosis between the vessels of the two sides , with rather scanty connections with the anterior spinal artery . the posterior spinal artery supplies the grey and white posterior columns of its own side

The radicular arteries make highly important contribution to reinforce the longitudinal trunk . They vary in number and position , and blood from them flow up and / or down the cord freely . ⁽⁵⁾

Etiology:-

The spinal cord can be injured in many ways of pathological processes include ;

- Compression from the enclosing vertebrae and discs or a mass in between (epidural) , or within (intradural) which may be either intra – or extramedullary .
- Inflammatory process whether infectious (biological) , drugs (chemical) or radiation (physical) .
- Vascular causes , include infarction , A-V malformation and vasculitides .

- Demyelinating process , like Guillian Barre , M.S , Devic disease and acute disseminating encephalomyelopathy .

-Hereditary, include ; F.S.P , Fredrick ataxia and the leukodystrophies .

A- Cord compression

-Disc prolapse:-

Spondylotic changes incidence increases with age . These include chronic disc bulge with osteophytes formation , facet hypertrophy and minor slips of one vertebra over the other causing pressure on the cord . ⁽⁶⁾

The course is variable but commonly progression occurs over a few months . Tingling and weak hands and legs and sphincters dysfunction , often with a sensory level occur with cervical disc prolapse . ⁽⁶⁾

In case of lumbar disc prolapse the lower limbs are weak and floppy , sensory and sphincters affection may occur . ⁽⁷⁾

Cauda Equina Syndrome :-

The cauda equina (CE) is formed by nerve roots caudal to the level of spinal cord termination. Cauda equina syndrome (CES) has been defined as low back pain, unilateral or usually bilateral sciatica, saddle sensory disturbances, bladder and bowel dysfunction, and variable lower extremity motor and sensory loss. CES may result from any lesion that compresses CE nerve

roots. These nerve roots are particularly susceptible to injury, since they have a poorly developed epineurium . ⁽⁸⁾

The pt present with low back pain , acute or chronic radiating pain , unilateral or bilateral lower extremity motor and/or sensory abnormality bowel and/or bladder dysfunction usually with associated saddle (perineal) anesthesia . ⁽⁹⁾

Causes: Trauma , lumbar disc disease , abscess , spinal anesthesia , tumor, metastatic, or CNS elements , late-stage ankylosing spondylitis and idiopathic . ⁽¹⁰⁾

Imaging Studies: Plain radiographs - Unlikely to be helpful, but obtain in search of destructive changes, disc space narrowing, or spondylolysis . CT scan with and/or without contrast - Lumbar myelogram followed by CT scan . MRI - Superiority of MRI over CT scan only suggested by case reports (early consultation with appropriate subspecialty is encouraged to guide imaging studies . ⁽¹¹⁾

No proven medical treatment exists, and therapy generally is directed at the underlying cause of CES. Some may suggest methylprednisolone in a regimen similar to that for traumatic spinal cord injury or another regimen of steroid for the acute syndrome . For penetrating trauma, steroids have not shown significant benefit. Surgery is controversial. The timing of

decompression is controversial, with immediate, early, and late surgical decompression showing varying results . (12 & 13)

Spinal cord tumors :

They are less common than the intracranial tumors (about 1:10). In most series, the average age at diagnosis is 40 years, ranging between 11 days & 74 years. Both sexes are equally involved. More than 70% of the tumors were located in the thoracic part or cervical (in that order)of the spinal cord . (14)

It is convenient to classify the spinal cord tumors by their location within the spinal cord, as extramedullary intradural, intramedullary, and extradural.

a)Extramedullary intradural tumors are the commonest spinal cord tumor (84% of all intradural tumors). Neurofibromas(29%) and meningiomas(25%) are the common ones. Exophytic ependymomas and astrocytomas account for about 20%. Sarcomas, vascular tumors epidermoids, lipomas etc are occasionally encountered . (15)

b)Intramedullary tumors are the commonest spinal cord tumors in children. Gliomas make up almost 70% of all intramedullary tumors. In adults alone ependymomas may account for up to 56% of all intramedullary tumors. Vascular tumors, represented by haemaingioblastomas and cavernomas, add up to almost 15% of all intramedullary tumors. Among the other tumors are found

cases of sarcoidosis, neurofibromas, ganglioglioma, gangliocytoma, oligodendroma and astrogliosis. In the literature there have also been case reports on primary malignant lymphomas and neurocytomas in the spinal cord .⁽¹⁶⁾

c)Extradural tumors are mostly metastatic. They spread into spinal cord from contiguous structures. About 5% of all patients with cancer develop vertebral metastasis. Lately, primary non osseous lymphomas are being reported increasingly.^(17 & 19)

Clinical features:- spinal cord tumors produce symptoms due to compression of nerve root or cord, and ischemia vascular compression. Tethering of the cord by the dentate ligaments and filum terminale may result when expanding lesions oppose this resistance .⁽¹⁵⁾

Extramedullary tumors grow in relation to a nerve root. Chronic progressive radicular pain, especially at night, may precede all other symptoms. Autonomic symptoms are delayed as the center of the cord is involved late unlike the intramedullary tumors. Radicular pain may simulate an angina at times.^(14 & 15)

Intramedullary tumors infrequently progress slowly, & for a long time often with rather mild symptoms and ill-defined pain. Since these tumors often destroy structures near the centre of the spinal cord, the crossing pain and temperature fibers are frequently damaged and there is early involvement of bladder

fibers. In the classical case the tumor therefore presents with an early segmental differential sensory deficit, later followed by long tract signs, with subsequent weakness & wasting of musculature in the extremities .Different degrees of paraesthesia, sensory loss, motor deficits and atrophy of the extremity musculature atrophy are then also encountered . (16)

Imaging Studies: MRI of the affected area provides the best definition of spinal lesions and is the procedure of choice.

With MRI, the entire spine may be visualized rapidly (sagittal images) . Usually, MRI can be used to differentiate a collapsed vertebra secondary to osteoporosis or trauma from malignant disease. The intervertebral space usually is not involved in tumors of the spine. When the disc space is obliterated, infection is more likely . (14)

Surgical excision is the treatment for extramedullary tumors. Total excision along with involved dura in case of meningiomas is possible and recommended . (18)

The traditional treatment of intramedullary gliomas has been biopsy followed by radiation therapy. (16)

Radiation therapy for intramedullary tumors has been controversial during the last decade. Clearly, radiation is accompanied by a risk of spinal injury .Radiation sensitivity

increases with the length of the cord irradiated, the size of the daily dose, and the total dose given . (16 & 18)

Pott's disease – tuberculous spondylitis

Tuberculous spondylitis has been documented in ancient mummies from Egypt and Peru and one of the oldest demonstrated disease of humankind. Percival Pott presented the classic description of spinal tuberculosis in 1779 . (19)

Pathophysiology : Pott's disease is usually secondary to an extra spinal source of infection . The basic lesion is a combination of osteomyelitis and arthritis . Typically more than one vertebra is involved , the area usually affected is the anterior aspect of the vertebral body adjacent to the subchondral plate . Progressive bone destruction leads to vertebral collapse and kyphosis . the spinal canal can be narrowed by abscess , granulation tissue or direct dural invasion . (20)

Presentation depends on the stage of the disease , site and presence of complications such as neurologic deficit , abscess or sinus tracts .

The reported average duration of symptoms at time of diagnosis is 3-4 months .Back pain is the earliest and most common symptom , usually for weeks prior to presentation , and can be spinal or radicular . Fever and weight loss may occur .

Neurologic complications occur in 50% of cases and can include

spinal cord compression with paraplegia , paraparesis , impaired sensation , nerve root pain or cauda equina syndrome . (21)

Cervical spine tuberculosis is a less common presentation but is potentially more serious because severe neurologic complications is more likely . The condition is characterized by pain and stiffness . Stridor , hoarseness of voice , torticollis and dysphagia can occur . (22)

Tuberculin test is positive in 84-95% of patients who are H.I.V negative .

E.S.R may be markedly elevated $> 100\text{mm/hr}$.

Bone tissue or abscess culture and stain for A.F.B is positive in only 50% of cases . (23)

Plane radiograph demonstrates lytic destruction of the anterior portion of the vertebral body , increased anterior wedging , collapse of vertebral body , reactive sclerosis on a progressive lytic process , intervertebral discs destruction and abscess shadow . (24)

C.T scanning produce much better bony details of irregular lytic lesions , sclerosis and disc collapse . (25)

M.R.I is the criterion standard for evaluating disc space infection , osteomyelitis and the extension of the disease into the soft tissues and compression . (25)

Medical care ;- Before the advent of effective antituberculous chemotherapy, Pott disease was treated with immobilization

using prolonged bed rest or a body cast. The mortality rate was 20%, and relapse was frequent (30%) . ⁽²²⁾

Indications for surgery ; -

Neurologic deficit (acute neurologic deterioration, paraplegia)

Spinal deformity with instability

No response to medical therapy in one month . ⁽²⁶⁾

Spinal cord abscess

Intramedullary spinal cord abscesses are infrequently encountered in everyday neurosurgical practice. Fewer than 100 cases have been reported since then . ⁽²⁷⁾

Spinal cord abscesses arise in spinal cord parenchyma and can be solitary or multiple, contiguous or isolated, and chronic or acute, depending upon the organism and individual patient . As may be expected, solitary lesions are more common and most likely appear in the thoracic cord. Holocord abscesses have been reported in approximately 5 patients . ⁽²⁸⁾

Frequency: Spinal cord abscesses occur more frequently in males than females with a peak incidence in the first and third decades of life. Patients with a history of intravenous drug abuse are at particularly high risk . ⁽²⁹⁾

Etiology: The most common organisms cultured from spinal cord abscesses include Staphylococcus and Streptococcus species, followed by gram-negative organisms. Mixed flora abscesses are also encountered. Other unusual organisms have been reported, including Actinomyces, Listeria, Proteus, and parasites . (29)

Pathophysiology: Spinal cord abscesses have many of the same characteristics of abscesses in other locations. Blood vessel involvement surrounded by an area of infection characterizes hematogenous spread. Areas of softening and early abscess formation characterize subacute infections (1-2 wk duration), whereas a classic abscess wall of fibrotic gliosis surrounding necrotic purulent material characterizes chronic infections. However, spinal cord abscesses do not destroy fiber tracts. Instead, the abscess displaces fiber tracts and spreads along axonal pathways . (30)

Clinical: In an acute presentation, symptoms of infection (e.g, fever, chills, back pain, malaise) are common. Neurological symptoms and signs include weakness, paraesthesia, dysesthesia, bladder and bowel incontinence, and acute paraplegia. Clinical symptoms are similar to those of patients with epidural abscesses, but percussion tenderness is not noted . (28)

In more chronic cases, signs and symptoms mimic those of an intramedullary tumor, and neurological symptoms predominate over those of a systemic infection. The neurological progression is gradual . (28)

Lab Studies: Cerebrospinal fluid (CSF) examination may show elevated protein and leukocyte levels but can be within reference ranges . (28)

Cultures with sensitivities from abscess aspirate are needed to identify infective organisms. Cultures should include tests for aerobic and anaerobic bacteria, fungi, and tuberculosis . (28)

The quickest and most reliable method for demonstrating a spinal cord mass is gadolinium-enhanced MRI. If a high probability of spinal abnormality is present, an MRI of the area will demonstrate the mass. MRI is also valuable in demonstrating any associated disease process (e.g, epidural or subdural infection, bone involvement, dermal sinus) . (28)

MRI does not differentiate among the types of masses, i.e., between tumor and abscess . Spinal cord abscesses produce homogenous spinal cord enlargement on T1-weighted images but produce high signal intensity on T2-weighted images . The abscess margin enhances brightly with gadolinium . (28)

Medical therapy: Treatment involves a combination of 3 modalities: surgical drainage of the abscess cavity, identification

of the infecting organism, and administration of appropriate antibiotics for a proper length of time . ⁽²⁹⁾

During treatment, steroids are used to reduce spinal cord swelling and edema associated with the abscess . ⁽³¹⁾

Acute Inflammatory Demyelinating Polyradiculoneuropathy:

Background: Acute inflammatory demyelinating polyneuropathy (AIDP) is an autoimmune process that is characterized by progressive weakness. Many variants exist. This is associated with distal paresthesias and loss of deep tendon reflexes . ⁽³²⁾

Myelin breakdown and axonal degeneration were observed in nerve biopsies from patients with AIDP by Haymaker and Kernohan in 1949. An allergic etiology was suggested by Krucke in 1955 after he observed lymphocytic infiltrates within biopsy specimens . ⁽³³⁾

Pathophysiology: AIDP is believed to be caused by an immunologic attack that is directed against myelin components. This results in a demyelinating polyneuropathy. Both cellular and humoral immune mechanisms appear to play a role. Early inflammatory lesions consist of a lymphocytic infiltrate that is adjacent to segmental demyelination. Macrophages are more prominent several days later. ⁽³³⁾

The changes are observed in nerve roots, peripheral nerves, and cranial nerves. In acute motor axonal neuropathy (AMAN, an AIDP variant), deposited complement is found at the nodes of Ranvier, while myelin often is left undamaged .⁽³⁴⁾

Damage to the myelin sheath leads to segmental demyelination. This results in decreased nerve conduction velocity and, at times, conduction block. Occasionally, in more severe and rapid cases, axonal degeneration also is observed which results in wallerian degeneration .⁽³⁴⁾

Frequency: In the US: AIDP is the most common acquired demyelinating polyneuropathy. The incidence is 0.6-1.7 cases per 100,000 per year. No significant seasonal variation has been noted .⁽³⁵⁾

Internationally: Frequency is not well documented. In rural China, the AMAN variant occurs in clusters during the late summer. Similar outbreaks have been reported in Mexico, Spain, and Jordan .⁽³⁵⁾

AIDP occurs in all races and in all regions of the world. Male-to-female ratio is 1.1-1.7:1. Patients have ranged in age from 2 months to 95 years. In China (and other countries) frequent outbreaks in children aged 2-12 years have been reported .⁽³⁶⁾

Mortality/Morbidity: In 3 recent large studies, mortality rate ranged from 2-6%. In general, death is due to complications of

ventilation. Causes include cardiac arrest, pulmonary embolus, sepsis, bronchospasm, pneumothorax, adult respiratory distress syndrome (ARDS), and dysautonomia . More than 75% of patients have complete or near-complete recovery with no deficit or only mild residual fatigue and distal weakness. Other patients, almost all of whom required ventilation, report severe dysesthesias or moderately severe distal weakness as residual symptoms . (37)

Causes: AIDP is thought to be caused by a dysregulated immune response against myelin. This response may be triggered by several illnesses and conditions. Two thirds of patients with AIDP recall an antecedent upper respiratory or gastrointestinal infection or syndrome from 1-6 weeks prior to the onset of weakness . (38)

Viral infection with influenza, coxsackie, Epstein-Barr virus, or cytomegalovirus can cause upper respiratory infection.

Immunoglobulin M (IgM) antibodies to each have been identified in some individuals .Acute infection with either herpes simplex virus or human immunodeficiency virus (HIV) also has been associated with AIDP in some individuals.

Patients with HIV-associated AIDP often have a pleocytosis with up to 200 WBC/mL CSF. Rare cases also have been reported after infection with rubella, measles, varicella-zoster, hepatitis B, Q fever, and Hantavirus . (38)

Bacterial strains of *Campylobacter jejuni* that cause enteritis are associated closely with the subsequent development of AMAN .
(36)

In children, an association exists between AIDP and *Mycoplasma pneumoniae* infection .Other: Rare cases of AIDP in individuals infected with toxoplasma, malaria, or filaria have been reported . (38)

Many cases of AIDP were reported after vaccination for swine influenza (especially in 1976).Several cases have been reported after immunization against rabies, influenza, measles, mumps, or rubella . (38)

History: classically, AIDP presents as an ascending paralysis . Even in these cases, the clinical presentation and course vary. Additionally, many variants exist that differ markedly from classic AIDP in disease onset or course .

The hallmark of classic AIDP is progressive weakness that usually begins in the feet before involving all 4 limbs. At presentation, 60% of patients have weakness in all 4 limbs. Weakness plateaus at 2 weeks after onset in 50% of patients and by 4 weeks in over 90%. It is usually symmetric, although mild asymmetry is not uncommon early in the disease course. In the arms, weakness may be worse proximally than distally. At presentation, half of patients have some facial weakness,

although only 5% have varying degrees of ophthalmoplegia . (32 & 39)

Forty percent have oropharyngeal or respiratory weakness at presentation. Improvement in strength usually begins 1-4 weeks after the plateau. About one third of patients require mechanical ventilation because of respiratory failure . (32)

Sensory symptoms , mild to moderately severe paresthesias in the distal limbs are common and often precede the onset of weakness by 1 or more days. Proximal sensory changes are uncommon but may occur in more severe cases of AIDP. (40)

About two thirds of patients have one or more autonomic abnormalities . Sustained sinus tachycardia is the most common dysfunction. Postural hypotension leading to presyncope or syncope can occur. Sweating dysfunction is common but rarely noted by patients. Urinary retention and constipation are more likely to occur later in the course of AIDP. (41)

The Miller-Fisher variant, presenting with ophthalmoplegia, areflexia, and ataxia, is the most common variant and is seen in as many as 5% of patients with AIDP. Although usually seen in adults, this variant is also common in children. Most patients with the Miller-Fisher variant have antibodies against ganglioside GQ1b . (42)

Regional variants of GBS, such as pharyngeal-cervical-brachial weakness or only leg weakness, are rare and resemble AIDP in time course .⁽⁴³⁾

The AMAN variant is seen in China and in developing countries. It presents with weakness only .⁽³⁶⁾

Acute motor-sensory axonal neuropathy resembles classic GBS in presentation but is related pathologically to AMAN.⁽³⁶⁾

Physical: weakness although patients often report only weakness in the legs, careful examination usually demonstrates arm weakness (proximally and distally). Some patients with Miller-Fisher or other regional variants may have weakness of cranial muscles only .⁽³²⁾

Deep tendon reflexes , hyporeflexia or areflexia is seen in 70% of patients at presentation and eventually in all patients. A progressive decrease in reflexes is a useful finding that may precede electromyographic (EMG) changes .⁽³²⁾

Fluctuations in heart rate, specifically a sustained sinus tachycardia, are seen often. Some intubated patients also may have bradycardia, especially after vagal stimulation with Valsalva and/or tracheal suctioning maneuvers .⁽⁴¹⁾

Orthostatic hypotension can occur and is likely due to dysfunction of the baroreceptor reflex .⁽⁴¹⁾

At times, the labile blood pressure is observed with severe hypertension that may be due to dysfunction of the afferent limb of the baroreceptor reflex .⁽⁴¹⁾

Findings that are inconsistent with a diagnosis of AIDP :⁽³³⁾

1-Weakness that remains markedly asymmetric .

2-Sharp sensory level .

3-Severe bladder or bowel dysfunction at onset .

C.S.F; Increased CSF protein without an increased WBC count (albuminocytologic dissociation) is observed classically in AIDP. However, this finding is not specific to AIDP. About two thirds of patients have this CSF finding during the first week of symptoms and 82% have it by 2 weeks after symptom onset .

Although protein values can be elevated by 10-fold or more, no association exists between protein level and clinical severity.

Some patients have oligoclonal banding of the CSF. Myelin basic protein also is increased in some patients . More than 90% of patients have fewer than 10 WBC/mL, with a mean of 3 WBC/mL. If more than 50 WBC/mL are present, an alternative diagnosis should be considered, including HIV, Lyme disease, polio, or other infections. Patients with HIV-associated AIDP often have >50 WBC/mL (mean, 23 WBC/mL) .⁽³⁴⁾

If intravenous immunoglobulin (IVIg) therapy is anticipated in non critical cases, immunoglobulin A (IgA) levels should be drawn before treatment . ⁽⁴⁴⁾

Stool cultures may confirm *C jejuni* enteritis. Patients with this condition may have a more aggressive course and a slightly worse prognosis . ⁽³⁸⁾

Nerve conduction studies (NCS) can document demyelination, the hallmark of AIDP. Early on, findings of NCS studies are often normal. However, 90% are abnormal within 3 weeks of symptom onset . ⁽⁴⁵⁾

Pulmonary function tests, useful in determining the timing of intensive care unit (ICU) transfers and elective intubation, should be performed in all patients . ⁽³⁷⁾

Transfer to an ICU generally is indicated when forced vital capacity (FVC) is less than 20 mL/kg. Intubation usually is warranted when FVC drops to 15 mL/kg or negative inspiratory pressure drops to less than -25 cm H₂O . ⁽³⁷⁾

Electrocardiogram (ECG) and cardiac monitoring can be helpful when arrhythmias occur. Other possible abnormalities include atrioventricular block, QRS widening, and T-wave abnormalities . ⁽³⁷⁾

Medical Care: mechanical ventilatory assistance is required in about one third of patients with AIDP and lasts for an average of 49 days. Tracheostomy usually is recommended if mechanical ventilation will be required for more than 2-3 weeks . ⁽³⁷⁾

Cardiac monitoring is necessary. Chronic sinus tachycardia often responds to beta-blockers or calcium channel blockers. Bradycardia requires atropine treatment, if symptomatic. Heart block may require temporary pacing. Hypertension responds well to beta-blockers . ⁽³⁷⁾

Immunomodulation with IVIg and plasmapheresis has led to faster recovery, relatively mild disability, and shorter hospital stays. IV steroid therapy alone is not indicated for the treatment of AIDP. Treatment is less likely to be effective if initiated more than 2 weeks after the onset of symptoms. Some patients with mild weakness, especially those presenting during the plateau, may not require immunomodulatory therapy . ⁽⁴⁶⁾

Prognosis: About 75% of patients have an excellent recovery and regain their premorbid condition . Some of these patients experience easy fatigability for many years . Death occurs in only 2-6% of patients and is usually due to cardiac arrest, ARDS, pulmonary embolism, severe bronchospasm, pneumonia, or sepsis . About 10% of patients have a relapse 1-6 weeks after completing immunomodulatory therapy. These

patients can be treated with a second course of immunomodulation .⁽⁴⁷⁾ **Multiple sclerosis**

Background: Multiple sclerosis (MS) is an inflammatory, demyelinating disease of the central nervous system (CNS). MS lesions, characterized by perivascular infiltration of monocytes and lymphocytes, appear as indurated areas in pathologic specimens; hence, the term "sclerosis in plaques."⁽⁴⁸⁾

Despite intensive efforts at finding the source of the disease, no etiologic agent for MS has been identified. The disease presumably can be exacerbated by hormonal changes during the postpartum period. Some argue that MS could be a heterogeneous disorder triggered by several different environmental agents. In fact, only 1 of every 4 MS attacks is associated with a viral infection .⁽⁴⁹⁾

The disease can present in different forms, such as primary progressive, relapsing remitting, relapsing progressive, and secondary progressive phenotypes. Genetic susceptibility factors may play a role, as the disease is more common in Caucasian populations living in northern latitudes .⁽⁵⁰⁾

Pathophysiology: MS is characterized by perivenular infiltration of lymphocytes and macrophages in the parenchyma of the brain, brain stem, optic nerves, and spinal cord. Expression of adhesion molecules on the surface seems to

underlie the ability of these inflammatory cells to penetrate the blood-brain barrier . The elevated immunoglobulin G (IgG) level in the cerebrospinal fluid (CSF), which can be demonstrated by an oligoclonal band pattern on electrophoresis, suggests an important humoral (i.e., B cell activation) component to MS. In fact, variable degrees of antibody-producing plasma cell infiltration have been demonstrated in MS lesions . ⁽⁴⁹⁾

The favorable clinical responses to the newer disease-modifying immunomodulatory agents (i.e., interferon beta-1a and -1b,) suggest that these medications modify disease progression on the basis of their ability to counteract the pro-inflammatory phenotype of immune cells . ⁽⁵¹⁾

Frequency: MS has a prevalence of nearly 350,000 cases in the United States alone. Every year, approximately 10,000 persons are newly diagnosed with MS. Internationally: More than 1 million worldwide are affected . ⁽⁵²⁾

Mortality/Morbidity: MS causes considerable disability in the working age group. People with MS usually die of complications rather than of MS itself, including recurrent infections (especially in bedridden patients). Patients with MS have an average life expectancy 7 years shorter than that of the general population . MS affects females more than males (1.6-2:1) . This ratio is even higher (3:1) among patients in whom

onset of MS is before age 15 years or after age 50 years, suggesting a hormonal component to the disease process. Males have a greater tendency to develop primary progressive MS, while females tend to experience more relapses. MS most commonly afflicts people between the ages of 18 and 50 years, but any age group can be affected . (52)

History: Attacks or exacerbations of MS are characterized by new symptoms that reflect CNS involvement. These symptoms typically are separated in time (e.g, weeks, months or years) and in anatomical location (e.g, one or more limbs, optic nerve, sensory symptoms). Recognizing that physical and cognitive disability in MS may occur in the absence of clinical exacerbations is important . (53)

Patients who improve after acute attacks have relapsing remitting MS (RRMS). However, during the natural course of RRMS, approximately 75-85% of patients enter a stage referred to as secondary progressive MS (SPMS). (50)

Patients with primary progressive MS (PPMS) tend to accumulate disability without interruption (ie, without remissions). Some of these patients first present with weakness of only one limb, which gradually progresses to total paralysis. Patients with PPMS typically respond poorly to the current therapeutic options for MS, accumulate disability faster than other patients, and tend to have more weakness of the legs as

well as incontinence (a reflection of greater spinal cord involvement) . ⁽⁵⁰⁾

Patients with PPMS tend to have more involvement of the spinal cord by demyelinating plaques . Patients who have RRMS but accumulate disability between and during attacks can be defined as having relapsing progressive disease (RPMS) . ⁽⁵⁰⁾

Multiple sclerosis may present in an acute and clinically fulminant form (termed Marburg variant of MS) or may present with concomitant optic nerve involvement and necrotizing myelopathy (i.e., neuromyelitis optica or Devic disease, considered by some to be an MS variant) . ⁽⁵³⁾

MS may present in various forms. Some patients have a predominance of cognitive changes, while others present with prominent ataxia, hemiparesis or paraparesis, depression, or visual symptoms . ⁽⁵³⁾

Optic neuritis presents clinically as orbital pain, at rest or during eye movement, and loss of vision. Patients may complain of "patchy loss of vision," and upon examination, a cecocentral scotoma and an afferent pupillary defect may be found. Patients may experience color desaturation even with normal visual acuity, usually manifested as the perception of red color as different shades of orange or gray . ⁽⁵⁴⁾

Patients with MS may present with facial palsies or trigeminal neuralgia. In fact, the presence of bilateral facial weakness or trigeminal neuralgia strongly suggests the diagnosis of MS. Facial myokymia also may be a presenting symptom. Nystagmus (direction-changing) and internuclear ophthalmoplegia are other manifestations . (54)

Painful limb syndromes are important to recognize. Commonly, patients complain of numbness or tingling in one or more limbs, variable weakness, or sensory level-related symptoms. Some have difficulty describing weakness or numbness, as these symptoms are obscured by incapacitating fatigue . (54)

Episodes of central (as opposed to peripheral) vertigo are not uncommon. The nystagmus accompanying central vertigo has a rapid onset, does not fatigue easily, and changes with direction of gaze. CNS vertigo usually is accompanied by other complaints that can be directly attributed to cranial nerves involvement (e.g, diplopia, dysarthria) . (54)

An often overlooked manifestation of MS is the pseudobulbar affect, whereby patients have difficulty controlling their emotions (laughing, crying) and are perceived to act inappropriately by co-workers or friends. (54)

Urinary retention and incontinence are common. Bowel habit changes may occur, but bowel incontinence is less frequent . (54)

Sexual dysfunction affects the great majority of patients with MS and includes symptoms such as lack of desire, erectile dysfunction, impaired sexual responsiveness, premature ejaculation, impaired genital sensation, or inability to physically interact with the partner due to painful leg adductor muscle spasms . ⁽⁵⁴⁾

Physical: The Kurtzke Expanded Disability Status Scale (EDSS) is used as a measure of disease progression by assigning a severity score (0-10) to the patient's clinical status. Although the scale does not correspond linearly to common progression points for many patients, its widespread use and ease of implementation allow its utilization as a standardization measure for clinical trials (Kurtzke, 1983) . ⁽⁵⁰⁾

C.S.F: Oligoclonal bands are distinct electrophoretic patterns that reflect substantial elevation of IgG produced by a restricted set of plasma cells and are demonstrated in CSF samples of approximately 85% of patients with MS . ⁽⁵²⁾

Myelin basic protein (MBP) is a major component of myelin and may be elevated in the CSF of patients with MS. Its clinical utility as a marker of disease activity or progression is limited . ⁽⁵⁵⁾

MRI; of head or spine, with and without gadolinium, should be performed according to clinical suspicion for lesion localization.

Typical MS lesions appear as T2 hyperintensities in the periventricular regions; they have an ovoid appearance, they involve only the white matter, and several arise from the corpus callosum. This characteristic configuration has been demonstrated in pathologic specimens and sometimes is referred to as "Dawson fingers". The most common infratentorial locations for plaque formation are the surface of the pons, the cerebellar peduncles, and white matter regions adjacent to the fourth ventricle. Lesions that enhance with gadolinium are thought to reflect active disease, as enhancement may correspond to breakdown of the blood-brain barrier from an ongoing subacute inflammatory process (few days to a few weeks). Usually a combination of enhancing and non-enhancing lesions is seen, reflecting the chronicity of the demyelinating process. In a patient with a first clinical attack who presents with multiple lesions by MRI, the presence of gadolinium enhancement in most or all the lesions is highly suggestive of ADEM; it is less likely to represent an extremely aggressive presentation of MS. ⁽⁵⁶⁾

Hypointensity of lesions in T1 images may reflect some degree of axonal damage or more chronic tissue damage resulting in gliosis. Additionally, a new lesion may present with T1 hypointensity, reflecting marked edema. Lesions range from a few millimeters to more than a centimeter in diameter with occasional large, rounded, tumor like lesions. The latter are seen

as areas of pronounced gliosis and demyelination on pathologic inspection . ⁽⁵⁷⁾

Evoked potential testing (visual, auditory, or somatosensory) is especially helpful in 1) detecting clinically silent lesions, and 2) documenting an organic basis for vague complaints. The most sensitive are the visual evoked potentials (50-80% sensitivity), followed by the somatosensory potentials (50-70% sensitivity) . ⁽⁵⁸⁾

Medical Care: Thus, preventing disease progression by using available medications is imperative in MS treatment, especially for patients who have been diagnosed early and probably will respond to treatment.

Prevent disease progression by using the “ABC” immunomodulatory drugs (ie, interferon beta-1a [Avonex], interferon beta-1b [Betaseron]. ⁽⁵¹⁾ **Acute exacerbations** . No highly effective treatment is currently available to counteract MS attacks. The most widely used treatment is intravenous (IV) methylprednisolone, 1 g IV qd for 3-5 days. This medication may help expedite the timing of recovery but will not affect the actual degree of recovery. High-dose IV steroids may work more effectively than oral steroids for the acute attack, and home IV therapy is recommended if the patient does not require hospitalization. Alternatively, high-dose oral methylprednisolone should be used, when feasible . ⁽⁵⁹⁾

Secondary progressive forms . These patients may be treated with Betaseron, especially when the clinical course reflects an early phase of progression . ⁽⁵¹⁾

Azathioprine also may be used as immunosuppressive treatments for MS, but these drugs should not substitute for ABC drugs as first-line agents in newly diagnosed RRMS. It is considered less suppressive than cyclophosphamide, and is being considered increasingly . ⁽⁶⁰⁾

Transverse myelitis:

Transverse myelitis is a neurological disorder caused by inflammation across both sides of one level, or segment, of the spinal cord. The term ***myelitis*** refers to inflammation of the spinal cord; ***transverse*** simply describes the position of the inflammation, that is, across the width of the spinal cord .

Attacks of inflammation can damage or destroy myelin, the fatty insulating substance that covers nerve cell fibers. This damage causes nervous system scars that interrupt communications between the nerves in the spinal cord and the rest of the body.

Symptoms of transverse myelitis include a loss of spinal cord function over several hours to several weeks . What usually begins as a sudden onset of lower back pain, muscle weakness, or abnormal sensations in the toes and feet can rapidly progress to more severe symptoms, including paralysis, urinary retention,

and loss of bowel control. Although some patients recover from transverse myelitis with minor or no residual problems, others suffer permanent impairments that affect their ability to perform ordinary tasks of daily living. Most patients will have only one episode of transverse myelitis; a small percentage may have a recurrence. ⁽⁶¹⁾

The segment of the spinal cord at which the damage occurs determines which parts of the body are affected. Damage at one segment will affect function at that segment and segments below it. In patients with transverse myelitis, demyelination usually occurs at the thoracic level, causing problems with leg movement and bowel and bladder control, which require signals from the lower segments of the spinal cord. ⁽⁶¹⁾

Transverse myelitis occurs in adults and children, in both genders, and in all races. No familial predisposition is apparent. A peak in incidence rates (the number of new cases per year) appears to occur between 10 and 19 years and 30 and 39 years. Although only a few studies have examined incidence rates, it is estimated that about 1,400 new cases of transverse myelitis are diagnosed each year in the United States, and approximately 33,000 Americans have some type of disability resulting from the disorder. ⁽⁶²⁾

Researchers are uncertain of the exact causes of transverse myelitis. The inflammation that causes such extensive damage to nerve fibers of the spinal cord may result from viral

infections, abnormal immune reactions, or insufficient blood flow through the blood vessels located in the spinal cord. Transverse myelitis also may occur as a complication of syphilis, measles, Lyme disease, and some vaccinations, including those for chickenpox and rabies. Cases in which a cause cannot be identified are called idiopathic . ⁽⁶²⁾

Transverse myelitis often develops following viral infections. Infectious agents suspected of causing transverse myelitis include varicella zoster, herpes simplex, cytomegalovirus, Epstein-Barr, influenza, echovirus, human immunodeficiency virus (HIV), hepatitis A, and rubella. Bacterial skin infections, middle-ear infections (otitis media), and *Mycoplasma pneumoniae* (bacterial pneumonia) have also been associated with the condition . ⁽⁶²⁾

In post-infectious cases of transverse myelitis, immune system mechanisms, rather than active viral or bacterial infections, appear to play an important role in causing damage to spinal nerves . ⁽⁶²⁾

Because some affected individuals also have autoimmune diseases such as systemic lupus erythematosus, Sjogren's syndrome, and sarcoidosis, some scientists suggest that transverse myelitis may also be an autoimmune disorder . ⁽⁶²⁾

An acute, rapidly progressing form of transverse myelitis sometimes signals the first attack of multiple sclerosis (MS), however, studies indicate that most people who develop transverse myelitis do not go on to develop MS. Patients with transverse myelitis should nonetheless be screened for MS because patients with this diagnosis will require different treatments . ⁽⁶³⁾

Transverse myelitis may be either acute (developing over hours to several days) or subacute (developing over 1 to 2 weeks) . Initial symptoms usually include localized lower back pain, sudden paresthesias (abnormal sensations such as burning, tickling, pricking, or tingling) in the legs, sensory loss, and paraparesis (partial paralysis of the legs) . Paraparesis often progresses to paraplegia (paralysis of the legs and lower part of the trunk). Urinary bladder and bowel dysfunction is common. Many patients also report experiencing muscle spasms, a general feeling of discomfort, headache, fever, and loss of appetite. Depending on which segment of the spinal cord is involved, some patients may experience respiratory problems as well . ⁽⁶¹⁾

From this wide array of symptoms, four classic features of transverse myelitis emerge: (1) weakness of the legs and arms, (2) pain, (3) sensory alteration, and (4) bowel and bladder dysfunction. Most patients will experience weakness of varying degrees in their legs; some also experience it in their arms.

Initially, people with transverse myelitis may notice that they are stumbling or dragging one foot or that their legs seem heavier than normal. Coordination of hand and arm movements, as well as arm and hand strength may also be compromised. Progression of the disease over several weeks often leads to full paralysis of the legs, requiring the patient to use a wheelchair .
(61)

Pain is the primary presenting symptom of transverse myelitis in approximately one-third to one-half of all patients. The pain may be localized in the lower back or may consist of sharp, shooting sensations that radiate down the legs or arms or around the torso . (61)

Bladder and bowel problems may involve increased frequency of the urge to urinate or have bowel movements, incontinence, difficulty voiding, the sensation of incomplete evacuation, and constipation . (61)

Because it is often difficult to distinguish between a patient with an idiopathic form of transverse myelitis and one who has an underlying condition, physicians must first eliminate potentially treatable causes of the condition. If a spinal cord injury is suspected, physicians seek first to rule out lesions (damaged or abnormally functioning areas) that could cause spinal cord compression. Such potential lesions include tumors, herniated or slipped discs, stenosis (narrowing of the canal that holds the

spinal cord), and abscesses. To rule out such lesions and check for inflammation of the spinal cord, patients often undergo magnetic resonance imaging (MRI), a procedure that provides a picture of the brain and spinal cord . ⁽⁶¹⁾

Blood tests may be performed to rule out various disorders such as systemic lupus erythematosus, HIV infection, and vitamin B12 deficiency. In some patients with transverse myelitis, the cerebrospinal fluid that bathes the spinal cord and brain contains more protein than usual and an increased number of leukocytes, indicating possible infection . ⁽⁶²⁾

The MRI findings in ATM and myelopathic MS have been described by various workers. Normal MRI has previously been reported in 7-50% of cases. MRI is likely to be normal if done early in the course i.e. within 5 days . Tartaglino et al found that on MR imaging, the MS plaques in spinal cord are characteristically less than 2 vertebral segments in length, peripherally located and occupy less than 2/3rd of cross section Bakshi et al described ATM as a longitudinal myelitis involving multiple segments, whereas MS plaques are more focal and involve only 1-2 segments. However, both the lesions are equally likely to expand the cord. ^(63 & 64)

As with many disorders of the spinal cord, no effective cure currently exists for people with transverse myelitis . Treatments are designed to manage and alleviate symptoms and largely

depend upon the severity of neurological involvement. Therapy generally begins when the patient first experiences symptoms. Physicians often prescribe corticosteroid therapy during the first few weeks of illness to decrease inflammation. Although no clinical trials have investigated whether corticosteroids alter the course of transverse myelitis, these drugs often are prescribed to reduce immune system activity because of the suspected autoimmune mechanisms involved in the disorder. Corticosteroid medications that might be prescribed may include methylprednisone or dexamethasone. General analgesia will likely be prescribed for any pain the patient may have. And bed rest is often recommended during the initial days and weeks after onset of the disorder. ⁽⁶⁵⁾

Subacute combined degeneration of the cord

This is due to vitamin B 12 deficiency . The brain , optic nerve , spinal cord and peripheral nerves are affected . ⁽⁶⁶⁾

First the pt notice weakness and paraesthesia , the lower limbs are involved before the upper . The examination discloses a disorder of the posterior and lateral columns of the spinal cord as the disease progresses . Vibration sensory loss is by far the most constant . The features are typically symmetrical . Optic atrophy and scotoma occurs late . ⁽⁶⁶⁾

Treatment include replacement of the loss and treating the underlying cause . ⁽⁶⁶⁾

3-Vascular diseases :-

Spinal cord infarction :

Occlusive vascular lesions affecting the spinal cord (“spinal stroke”) are diagnostic challenges. The circulation to the spinal cord has unique features related to the elongated and multimeric anatomy of the cord that affect both mechanism and clinical presentation .

Frequency :- Spinal cord infarction is not common, but only fragmentary or indirect data are available on incidence or prevalence. A large study showed that only 9 of 3784 autopsies revealed spinal cord infarction, with a rate of occurrence of 0.23% at death. Conversely, if spinal stroke is approximately 1.2% of strokes, an overall annual incidence of 12 in 100,000 can be estimated . ⁽⁶⁷⁾

Clinical features :-Spinal cord infarction is marked by acute onset, often heralded by sudden and severe spinal (back) pain, which may radiate caudally. This is associated with bilateral weakness, paraesthesia , and sensory loss . ⁽⁶⁸⁾

The spinal cord stroke, either ischemic or hemorrhagic, has an acute and often apoplectic onset evolving over minutes.

Neurologic deficit may occur without pain, but most (>80%) spinal infarcts are painful. Neurologic dysfunction stems from a lesion located in the anterior two thirds (or in the central “watershed”) of the spinal cord and spares vibration and position sense perception, which are carried by the posterior columns and relatively spared. In the acute stage (usually for several days), “spinal shock” with flaccid muscle tone and areflexia, including absent Babinski reflexes, is observed commonly. Distal to the lesion, superficial pain and temperature discrimination are lost bilaterally with preservation of light touch, vibration, and position sense . ⁽⁶⁸⁾

Causes: The pathology may involve the aorta or an intervening arterial feeder (e.g, thoracic, intercostal, or cervical branch from subclavian or vertebral artery), or the radicular artery may affect the anterior spinal artery and intrinsic arterial vessels within the spinal cord. Spinal venous pathology may produce spinal infarction, although this is clinically rare. ⁽⁶⁹⁾

Involvement of intrinsic cord vessels has been reported with arteritis, both in systemic lupus erythematosus and granulomatous arteritis, and from emboli of atheroma or even from compression by intervertebral disk fragments . ⁽⁶⁹⁾

Anterior spinal artery occlusion has been reported with arteritis, including that associated with syphilis and diabetes mellitus; after trauma; spontaneously or without recognized cause; and as

a complication of spinal angiography, cervical spondylosis, spinal adhesive arachnoiditis, administration of intrathecal phenol, and spinal anesthesia . ⁽⁶⁹⁾

Aortic disease has produced spinal infarction in a variety of situations including dissecting aneurysm; aortic surgery, aortography; atherosclerotic embolization; and aortic thrombosis . ⁽⁶⁹⁾

A crucial examination is the imaging that can identify (or exclude) a mass or space-occupying lesion that is compressing or compromising the circulation of the spinal cord (extra axial) or is within the cord tissue (intra axial). The easiest and safest procedure for this is spinal MRI . ⁽⁷⁰⁾

Myelography, especially with the greater sensitivity of the enhanced CT myelography, is quite satisfactory for definition of mass lesions and can be employed if MRI is unavailable or for any reason unsatisfactory (e.g, a very obese patient). Cranial MRI is valuable in the patient with multiple sclerosis because the abnormalities found provide confirmatory evidence . ⁽⁷⁰⁾

Spinal angiography is indicated occasionally, usually for diagnosis and treatment of a spinal arteriovenous malformation. The procedure is technically difficult and somewhat risky and usually is performed only at tertiary care medical facilities . ⁽⁷¹⁾

Medical Care: The standard drug therapy is aspirin. This is based upon the consensus recommendation for acute treatment of ischemic stroke at any site. No direct studies have examined efficacy of drug therapy in spinal cord infarction. Neuroprotective strategies, including antioxidant, antiglutamatergic, and protease inhibition, improve outcome in animal experimentation with models of acute ischemia but have not yet been reported effective in human cord ischemia . ⁽⁷²⁾

4-Heredodegenerative Diseases:-

Familial spastic paraplegia :

Strumpell first described hereditary forms of spastic paraplegia in 1883. Lorrain later described them more extensively. The common feature of these syndromes is progressive, often severe, spasticity in the lower extremities . Syndromes are classified as uncomplicated or pure when only spinal involvement occurs, and they are classified as complicated when they are associated with neurologic abnormalities such as ataxia, mental retardation, dementia, extra pyramidal dysfunctions, visual or hearing dysfunctions, adrenal insufficiency, and ichthyosis. Inheritance may be X-linked, autosomal recessive, or autosomal dominant . ⁽⁷³⁾

Pathophysiology: HSP causes degeneration of the ends of the corticospinal tracts within the spinal cord. The ends of the longest fibers, which supply the lower extremities, are affected

to a much greater extent than the fibers to the upper body. In most cases of HSP, the primary problem may be disturbance of the ends of the long axons with little or no loss of myelin and not abnormal myelin. A rare type of X-linked HSP, however, has been associated with a myelin protein gene mutation. ^(74 &75)

Symptoms ; the classic symptom of HSP is progressive difficulty in walking, but the severity varies. Some patients eventually may require the use of a wheelchair, while others may never need any type of assistive device. Patients usually have difficulty lifting their toes, which results in their dragging the toes when walking and catching them on stairs or on uneven sidewalks or curbs. In later stages, patients experience difficulty flexing the thigh muscle to raise the leg when walking. Reduced sense of balance is noted . Some people also experience urinary problems (e.g., incontinence, sense of urgency even when bladder is not full). Many symptoms common in people with HSP are not directly caused by HSP but indirectly result from muscle spasticity, weakness, or hyperactive reflexes . Abnormal gait Increasing stiffness in the legs is associated with frequent tripping, particularly when the patient is walking on uneven terrain . Uncontrollable shaking of the legs may be noted when the patient ambulates. Dragging of the feet , scissoring of the legs during ambulation, weakness and giving way at the ankles, flexor spasms of the legs during the night, and a sense of unsteadiness during walking also are common. ⁽⁷⁵⁾

The age of symptom onset, rate of symptom progression, and extent of disability are variable both within and between HSP kindreds. In contrast to the variable age of the patients at symptom onset and the extent of disability, the distribution of neurologic deficits in uncomplicated HSP is consistent and consists of spastic weakness in the legs, variable impairment of vibratory sense in the feet, and variable urinary bladder disturbance . Some autosomal dominant uncomplicated HSP kindreds exhibit onset of progressive spastic paraplegia in childhood (i.e., <6 y) and relatively little progression of symptoms beyond adolescence. These patients often do not experience urinary bladder disturbances and generally remain ambulatory with assistance . (75)

Physical: Neurologic examination reveals no evidence of reduced mentation and cranial nerve dysfunction. Although the jaw jerk may be brisk in older subjects . Upper-extremity muscle tone and strength are normal . In the lower extremities, muscle tone is increased at the hamstrings, quadriceps, and ankles . (75)

Muscle wasting may occur in patients with uncomplicated HSP, but it is mild and limited to atrophy of the shins in wheelchair-dependent elderly patients. (75)

Vibratory sensation is often mildly diminished in the distal lower extremities. When present, this deficit is useful as a

diagnostic sign that helps distinguish HSP from other disorders .
(76)

Slight terminal dysmetria is occasionally observed on finger-to-nose testing in older affected individuals. (75)

Deep tendon reflexes may be brisk in the upper extremities but are pathologically increased in the lower extremities . (77)

The patient's gait demonstrates circumduction owing to a difficulty with hip flexion and ankle dorsiflexion.

Crossed adductor reflexes, ankle clonus, and extensor plantar responses are uniformly present. (75)

High arched feet (pes cavus) are generally present and usually prominent in older patients. (75)

Rehabilitation Program: Regular physical therapy (PT) is important to maintain and improve range of motion (ROM) and muscle strength. Furthermore, PT is necessary to maintain aerobic conditioning of the cardiovascular system . (73)

Syringomyelia :

Syringomyelia is the development of a fluid-filled cavity or syrinx within the spinal cord. Hydromyelia is a dilatation of the central canal by cerebrospinal fluid (CSF) and may be included

within the definition of syringomyelia. Types of syringomyelia include the following :-

1-Syringomyelia with fourth ventricle communication

About 10% of syringomyelia cases are of this type. This communication can be observed on MRI. In some cases, a blockage of CSF circulation occurs. A shunt operation may be the best therapeutic option for these patients . ⁽⁷⁸⁾

2-Syringomyelia due to blockage of CSF circulation (without fourth ventricular communication)

Representing at least 50% of all cases, this is the most common type of syringomyelia. Obstruction of CSF circulation from the basal posterior fossa to the caudal space may cause syringomyelia of this type. The most common example is Arnold-Chiari malformation, which also is associated with communicating syringomyelia . ⁽⁷⁸⁾

3-Syringomyelia due to spinal cord injury

Fewer than 10% of syringomyelia cases are of this type. Mechanisms of injury include (1) spinal trauma, (2) radiation necrosis, (3) hemorrhage from aneurysm rupture or arteriovenous malformation or in a tumor bed, (4) infection (spinal abscess, human immunodeficiency virus, transverse

myelitis), and (5) cavitation following ischemic injury or degenerative disease . ⁽⁷⁸⁾

4-Syringomyelia and spinal dysraphism .

5-Syringomyelia due to intramedullary tumors , fluid accumulation usually is caused by secretion from neoplastic cells or hemorrhage. The tumors most often associated with syringomyelia are ependymoma and hemangioblastoma . ⁽⁷⁸⁾

6-Idiopathic syringomyelia , has an unknown cause and cannot be classified under any of the previous categories . ⁽⁷⁸⁾

Pathophysiology: Although many mechanisms for syrinx formation have been postulated, the exact pathogenesis is still unknown.

Frequency: estimated prevalence of the disease is about 8.4 cases per 100,000 people . ⁽⁷⁹⁾

Mortality/Morbidity: in one study, half of all patients with syringomyelia were in clinically stable condition for several years. About 20% of all patients died at an average age of 47 years. Occurrence of syringomyelia in different races is unknown. Familial cases have been described. The disease usually appears in the third or fourth decade of life, with a mean age of onset of 30 years ⁽⁷⁹⁾

History: Syringomyelia usually progresses slowly; the course may extend over many years. The condition may have a more acute course, especially when the brain stem is affected (i.e., syringobulbia). Syringomyelia usually involves the cervical area. Symptomatic presentation depends primarily on the location of the lesion within the neuraxis . (80)

Sensory ; syrinx interrupts the decussating spinothalamic fibers that mediate pain and temperature sensibility, resulting in loss of these sensations, while light touch, vibration, and position senses are preserved When the cavity enlarges to involve the posterior columns, position and vibration senses in the feet are lost; astereognosis may be noted in the hands . Pain and temperature sensation may be impaired in either or both arms, or in a shawl like distribution across the shoulders and upper torso anteriorly and posteriorly . Motor syrinx extension into the anterior horns of the spinal cord damages motor neurons and causes diffuse muscle atrophy that begins in the hands and progresses proximally to include the forearms and shoulder girdles. Claw hand may develop . (80)

Autonomic; impaired bowel and bladder functions usually occur as a late manifestation . Sexual dysfunction may develop. Horner syndrome may appear, reflecting damage to the sympathetic neurons in the intermediolateral cell column . (80)

A syrinx may extend into the medulla, producing a syringobulbia. This syndrome is characterized by dysphagia, nystagmus, pharyngeal and palatal weakness, asymmetric weakness and atrophy of the tongue, and sensory loss involving primarily pain and temperature senses in the distribution of the trigeminal nerve . ⁽⁸⁰⁾

Rarely, the syrinx cavity can extend beyond the medulla in the brain stem into the centrum semiovale (syringocephalus) . ⁽⁸¹⁾

Lumbar syringomyelia can occur and is characterized by atrophy of the proximal and distal leg muscles with dissociated sensory loss in the lumbar and sacral dermatomes. Lower limb reflexes are reduced or absent. Impairment of sphincter function is common . ⁽⁸¹⁾

Physical: Arm reflexes are diminished early in the clinical course .Lower limb spasticity, which may be asymmetrical, appears with other long-tract signs such as paraparesis, hyperreflexia, and extensor plantar responses . Dissociated sensory impairment may be noted . The syrinx may extend into the brain stem, affecting cranial nerves or cerebellar function . Brainstem signs are common in syringomyelia associated with Chiari malformations . ⁽⁸⁰⁾

Lab Studies: CSF pressure sometimes is elevated. A complete subarachnoid block may be noted. Cell count is rarely more

than $10/\text{mm}^3$. Mild elevation of the CSF protein content occurs in half of these cases . In cases of subarachnoid block, CSF protein may exceed 100 mg/dL . ⁽⁷⁸⁾

Imaging Studies:

Plain x-ray ; plain films cannot detect the syrinx directly.

Cervical canal commonly is widened, and the pedicles may be eroded. Basilar impression or craniovertebral anomalies may be demonstrated . ⁽⁷⁹⁾

Computerized tomography scan - Assists in detailed assessment and is especially useful in evaluation of bony spinal canal components . ⁽⁷⁹⁾

Myelography ; is performed in special situations when MRI cannot be used. Widening of the cord and complete subarachnoid block may be observed . ⁽⁷⁹⁾

M.R.I of the entire rostrocaudal extension of the cyst or cysts is important Gadolinium-enhanced images are indicated if a tumor is suspected. They help in differentiating between scar or disk material associated with a syrinx, especially in postoperative or posttraumatic cases . MRI examination should include sagittal and transverse views in T1 and T2 images Proton density scans also can be helpful . ⁽⁷⁹⁾

Magnetic resonance angiography - Can be especially helpful in cases of syringomyelia associated with vascular lesions . ⁽⁷⁹⁾

Cine phase-contrast MRI - Used to analyze CSF flow dynamics near the spinal cord cyst . ⁽⁷⁹⁾

Real-time ultrasonography - Rarely utilized for imaging syringomyelia since the development of MRI . ⁽⁷⁹⁾

Medical Care: no medical treatment is known for patients with syringomyelia. However, a chronic, stable clinical course is common. Identifying the underlying cause of syrinx formation is very important. Surgical treatment most likely will be necessary.

Neurorehabilitative care facilitates preservation of remaining neurological functions and prevents complications of quadriparesis such as infection and decubitus ulcers.

Surgical Care: A variety of surgical treatments have been proposed for syringomyelia . ⁽⁸²⁾

1-Suboccipital and cervical decompression

2-Laminectomy and syringotomy)

3-Ventriculoperitoneal shunt lumboperitoneal shunt and syringo- peritoneal shunt

Motor neuron disease :-

This results from loss of function of both upper and lower motor neurons controlling the limbs and bulbar muscles , and by far one of the frequently encountered degenerative diseases . It comprises groups of diseases , amyotrophic lateral sclerosis is the commonest of them . ⁽⁸³⁾

A.L.S occurs worldwide , and the incidence increases with age , and it is unusual before the fifth decade . The cause is unknown , but familial cases were reported . Males are affected more than females . ⁽⁸⁴⁾

Weakness of the hands muscles , wasting , fasciculation and features of upper motor neuron lesion are evident . Sphincters and autonomic dysfunctions are unusual . ⁽⁸⁴⁾

The diagnosis is clinical , N.C.S to demonstrate denervation , and imaging studies to look for possible compression . ⁽⁸⁴⁾

No treatment known to cure the disease .

Leukodystrophy :-

Metachromatic leukodystrophy caused by a cerebroside sulfatase deficiency . Patient develop the inability to walk and frequently present with gait abnormality . Pts with the juvenile onset have the disease in their 4th – 20th yr . The adult form is more subtle slowly progressive , in the 2nd to the 5th decade . dementia , motor dysfunction and indistinct speech are the features . ⁽⁸⁵⁾

Diabetic amyotrophy :-

It is a form of peripheral neuropathy , though atrophy and weakness of large muscles mimic myopathy . The upper and /or the lower limbs are involved . Pain , anorexia , depression and high C.S.F proteins are other features . ⁽⁸⁶⁾

Parasagittal meningioma

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 . ⁽⁸⁷⁾

Localized or nonspecific headaches are common. Compression of the underlying brain can give rise to focal or more generalized cerebral dysfunction, as evinced by focal weakness, dysphasia, apathy, and/or somnolence. Intraventricular meningiomas may present with obstructive hydrocephalus , in the vicinity of the sella turcica may produce panhypopituitarism and, that compress the visual pathways produce various visual field defects, depending on their location . The physical findings mirror the aforementioned symptoms and include signs secondary to raised intracranial pressure, involvement of cranial nerves, compression of the underlying parenchyma, and involvement of bone and subcutaneous tissues by the

meningioma .Involvement of the cranial nerves may lead to anosmia, visual field defects, optic atrophy, diplopia, decreased facial sensation, facial paresis, decreased hearing, deviation of the uvula, and hemi atrophy of the tongue . (87&88)

Compression of the underlying parenchyma may give rise to pyramidal signs that are exemplified by pronator drift, hyperreflexia, positive Hoffman sign, and presence of the Babinski sign. A parietal lobe syndrome may occur if the parietal lobes are compressed . Compression of the dominant (usually left) parietal lobe may give rise to Gerstmann syndrome: agraphia, acalculia, right-left disorientation, and finger agnosia

Imaging studies are the mainstay of diagnosis . (87 &88)

Tropical spastic paraparesis:-

(**H.T.L.V-1**) , was the first pathogenic retrovirus to be discovered in man , it causes diseases in only 5% of infected people , it is associated with adult T- cell leukemia /lymphoma and tropical spastic paraparesis . The disease was first reported in Caribbean 1985 and then Japan 1986 and also known as H.T.L.V-1 myelopathy . (89)

Progressive spastic paraparesis of gradual onset is the major features and by 10 years many patients are confined to a wheelchair . Sensory symptoms may precede the onset of

weakness but sensory signs are few . Bladder involvement is common , producing frequency , urgency and incontinence , constipation is also a problem . ⁽⁹⁰⁾

Other rare features include arms weakness and spasticity , sensory level on the trunk , Brown Sequard syndrome , impotence sensory neural deafness and optic atrophy . ⁽⁹⁰⁾

Serology of the virus shows higher titer , but is not diagnostic , so M.R.I , B12 and other studies should be done to rule out other causes . ⁽⁹¹⁾

Objectives

* To study the clinical pattern of non traumatic paraplegia and the etiologies in Sudanese patients .

Methodology:-

Study design :- descriptive cross sectional

Study field :- El Shaab teaching hospital (The neurology center

Study population :- patients with lower limbs weakness ,
Admitted to the hospital the year 2002 – 2003 .

Inclusion criteria : -

1/ Sudanese adult patient with lower limbs weakness .

Exclusion criteria :-

1/patients below 16 years of age .

2/ patients with myopathic weakness

3/ patients with traumatic paraplegia .

Sampling ;

Sample size :- 100 patients .

Sample design :-

Systematic random sample .

Sample frame :-

List by registration number .

Tools of data collection :-

Data were collected by ;

1/ Pre-designed questionnaire ,

2/ Clinical examination .

3/ Investigations

Data entry and analysis :-

Data was introduced into the computer from a master sheet recording .
Using S.P.S.S soft wire program , data were entered and analyzed .

using the student t test .

Age was grouped into :

- 16 -25yrs , 25-35 , 35-45 , 45-55 , 55-65 and 65-75 .

Residence was classified as ;

- Khartoum state , Jazira , west , east, north and south ,

Symptoms considered as ;

1/ Antecedent events in form of ;

- Fever , wt loss , cough and diarrhea .

2/ Presenting complains

- weakness , sensory complains , sphincters dysfunction , upper limbs , cranial nerves and cortical symptoms .

3/ Physical signs were grouped into ;

- general examinations systemic and neurologic

4/ Neurologic examinations were further arranged as ;

- cortical , cerebellar , cranial nerves , upper and lower limbs .

5/ work up as ;

- general investigations and specific ones .

6/ The specific investigations were ;

- Imaging , C.S.F , Mantoux , V.D.R.L , bone marrow and sputum .

Results

* Table 1 , showed that patients of age group distribution 16-25 to be 23% . 25-35 , 23% . 35-45 , 22% . 45-55 , 8% . 55-65 , 15% . 65-75 , 7% and 75-85 2% .

* Table 2 , showed the antecedent events , fever was reported in 40% , backache in 45% , weight loss in 15% , diarrhea in 5% and cough in 3% .

* Table 3 , showed that weakness was reported by 92% , unsteadiness was reported by 41% , sensory symptoms were reported by 38% and root pain was reported by 21% .

* Table 4 , showed that 2% of patients had cortical symptoms , 10% had cranial nerves symptoms , and 9% had upper limbs symptoms .

* Table 5 , showed that Guillian Barre was the final diagnosis in 19% , followed by transverse myelitis 15% , tumors , 13% for secondary and 3% for primary , Pott's disease 11% , multiple sclerosis 7% , 4% for each of diabetic amyotrophy , H.S.P and S.C.D.C , 3% for each of disc disease , C.E.S , ischemic myelitis and C.I.D.P , arachnoiditis 2% and 1% for each of syringomyelia , abscess , Devic's and A.D.E.M .

* Table 6 , showed that 74% of patients had abnormal M.R.I , while 4% had a normal M.R.I .

* Table 7 , showed that plane x- ray was abnormal in 36% while it was normal in 33% of patients .

* Table 8 , showed that the level of the lesion in the cord to be 31 % in the lower thoracic segments , 25% in the upper thoracic , 14% in the cervical segments , 6% in the lumbar segments and diffuse in 2% .

* Figure 1 showed that 56% of patients are male and 44% are female

* Figure 2 , showed that 40% of patients are resident in Khartoum , 19% in Jazira , 14 % in the north , 13% in the east , 12% in the west , and only 2% in the south .

* Figure 3 showed that the onset was rapid or acute in 40% , subacute in 14% of patients and gradual in 46% .

* Figure 4, showed that urinary sphincter disturbance occurred in 46% of patients while the remainder had no disturbance

* Figure 5 , showed that bowel sphincter disturbance occurred in 28% of patients while the remainder had no disturbance .

* Figure 6, showed that 22% of patients had muscles wasting while no wasting was seen in 78% .

* Figure 7, showed that 54% of patients had hypotonia , 36% had hypertonia , and 10% had normal tone .

* Figure 8, showed that 56% of patients had brisk reflexes , 43% had absent reflexes and 1% had a normal reflex .

* Figure 9, showed that the power was grade 0 in 13% of patients , grade 1 in 14% , grade 2 in 25% , grade 3 in 27% , and grade 4 in 21% .

* Figure 10, showed that 64% of the patients had an up going planter response , 22% had mute response and 14% had down going response . * Figure 11, showed that the sensation was absent in 48% of the patients , normal in 37% , diminished in 12% and dissociated in 3% .

* Figure 12, showed the abdominal reflexes were absent in 65% of patients and intact in 35% .

* Figure 13 , showed that 56% of patients were wheelchair dependent , 22% could walk if supported and 22% could walk independently .

* Figure 14 , showed that the gait was spastic in 23% , stamping in 11% , ataxic in 3% and unsteady in 1% of patients .

Table No 1
Age distribution of patients

Age group	Percent
16-25	23
25-35	23
35-45	22
45-55	8
55-65	15
65-75	7
>75	2
Total	100

Table No 2
The antecedent events

Symptom	Fever	Wt loss	Diarrhea	cough	Backache
Percent	40	15	5	3	45

Table No 3
The presenting symptoms

Symptom	Weakness	Unsteadiness	Sensory	Root pain
Percent	92	41	38	21

Table No 4

Concomitant cortical , cranial and upper limbs symptoms

Symptom	Cortical	Cranial	Upper limbs
Percent	2	10	9

Table No 5
The different diagnoses

Diagnosis	Percent
A.I.D.P	19
A.T.M	15
Secondary tumors	13
Pott's disease	12
M.S	9
H.S.P	4
S.C.D.C	4
Diabetic amyotrophy	4
Disc disease	3
Cauda equina syndrome	3
C.I.D.P	3
Ischemic myelitis	3
Degenerative diseases	3
Primary tumors	3
Arachnoiditis	2
Total	100

Table No 6
The M.R.I

The result	Normal	Abnormal
No of patients	4	74

Table No 7
The plane x-ray

The result	Normal	Abnormal
No of patients	33	36

Table No 8

The level of the lesion in the cord

The level	Lower thorax	Upper thorax	Cervical	Lumbar	Diffuse
No of patients	32	19	14	7	2

Figure No 1
The gender of patients

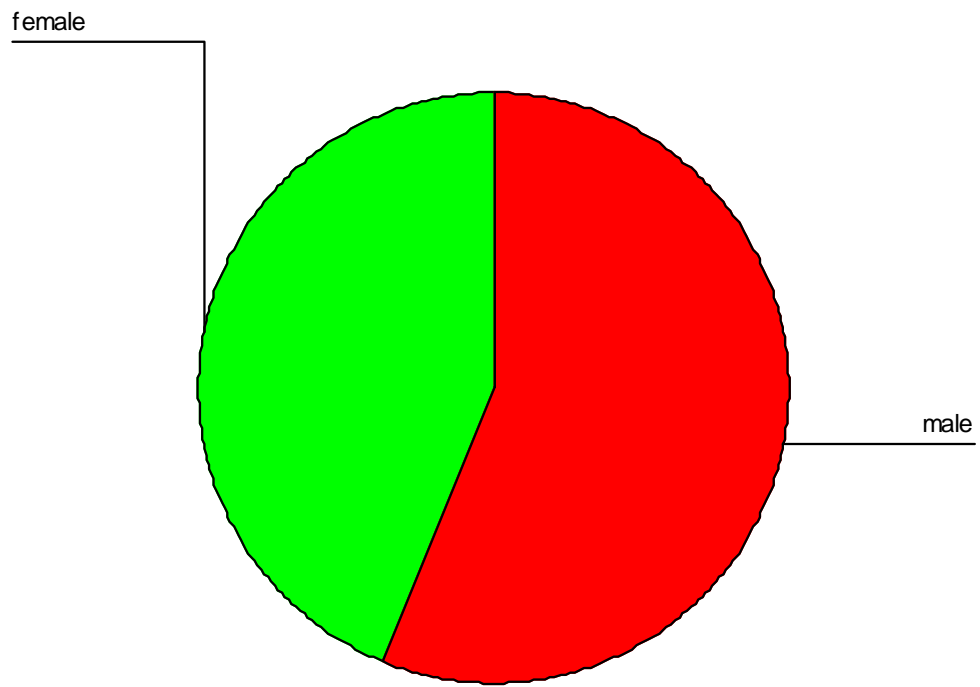


Figure No 2
The residence

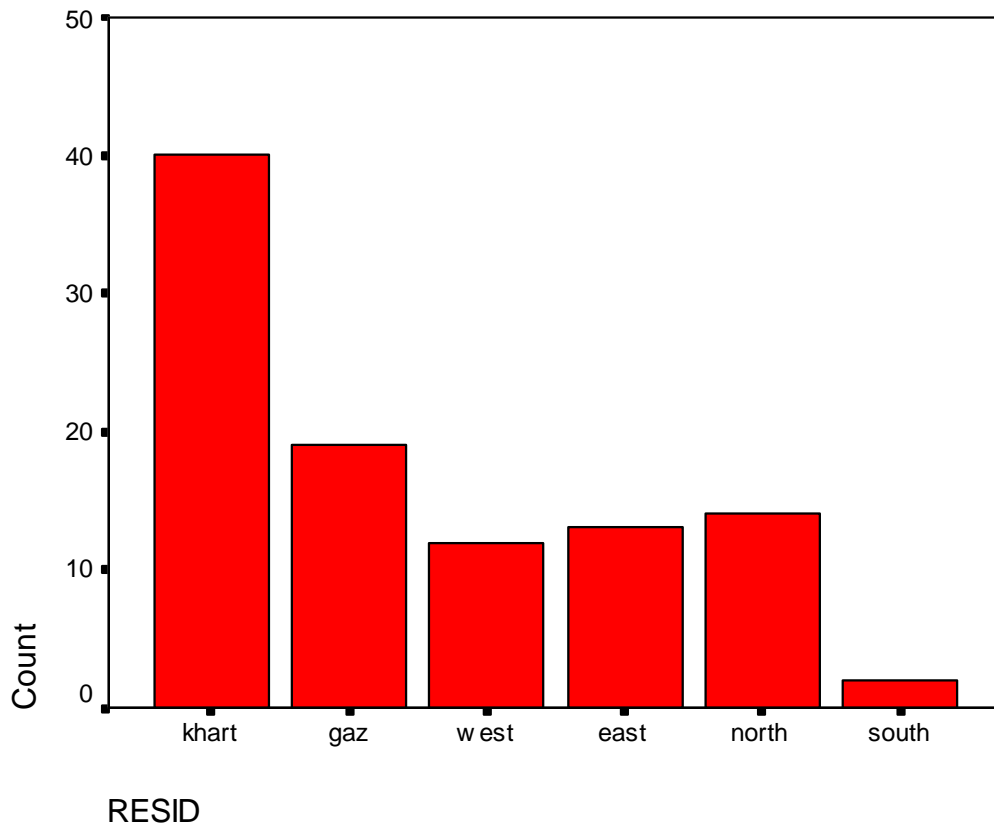


Figure No 3
The onset of weakness

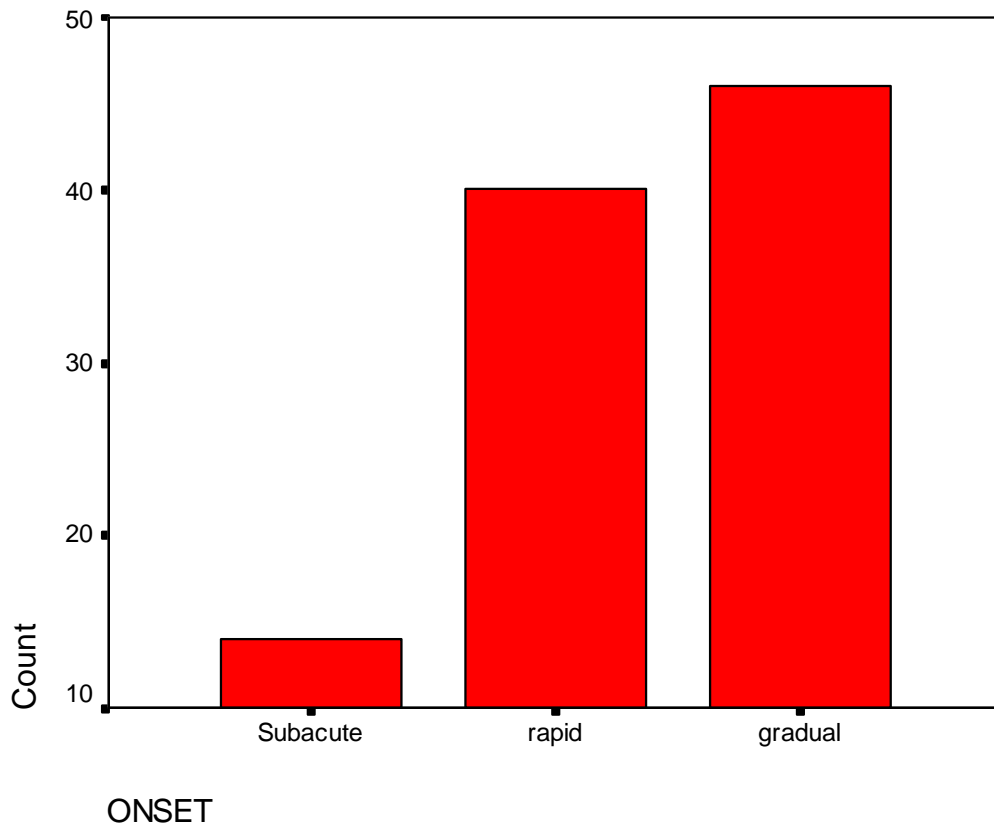


Figure No 4
The state of urine sphincter

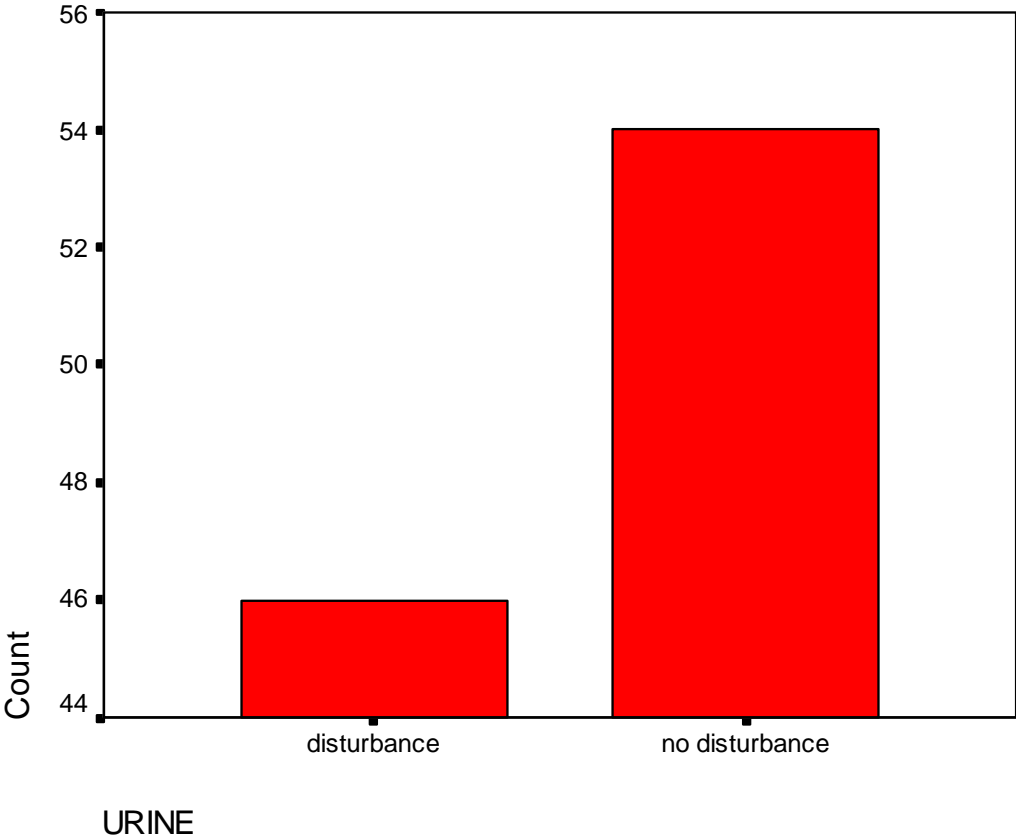


Figure No 5

The state of bowel sphincter

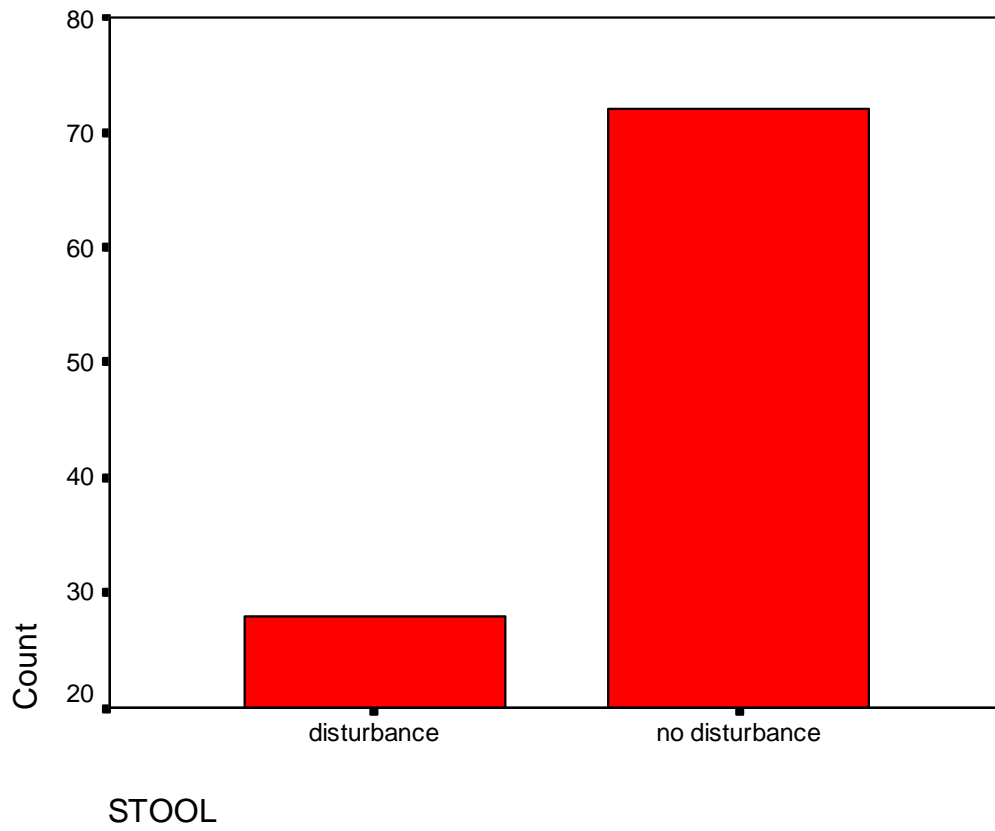


Figure No 6
The muscle waste state

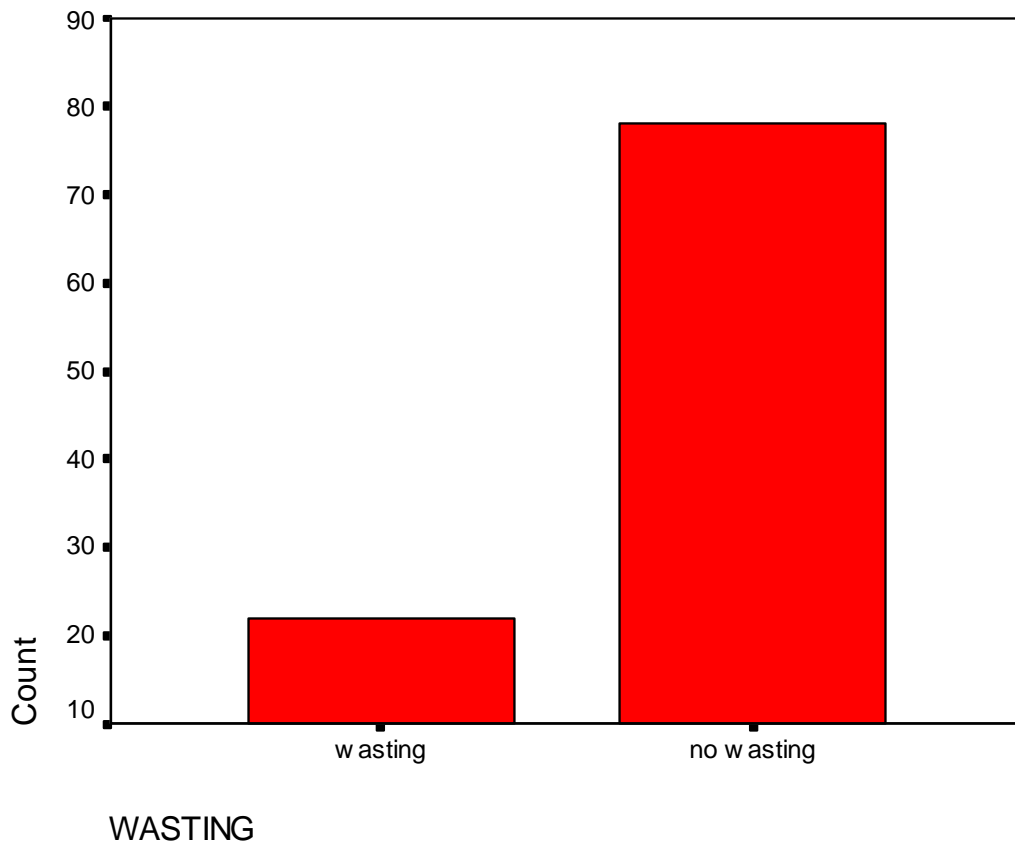


Figure No 7
The states of the tone

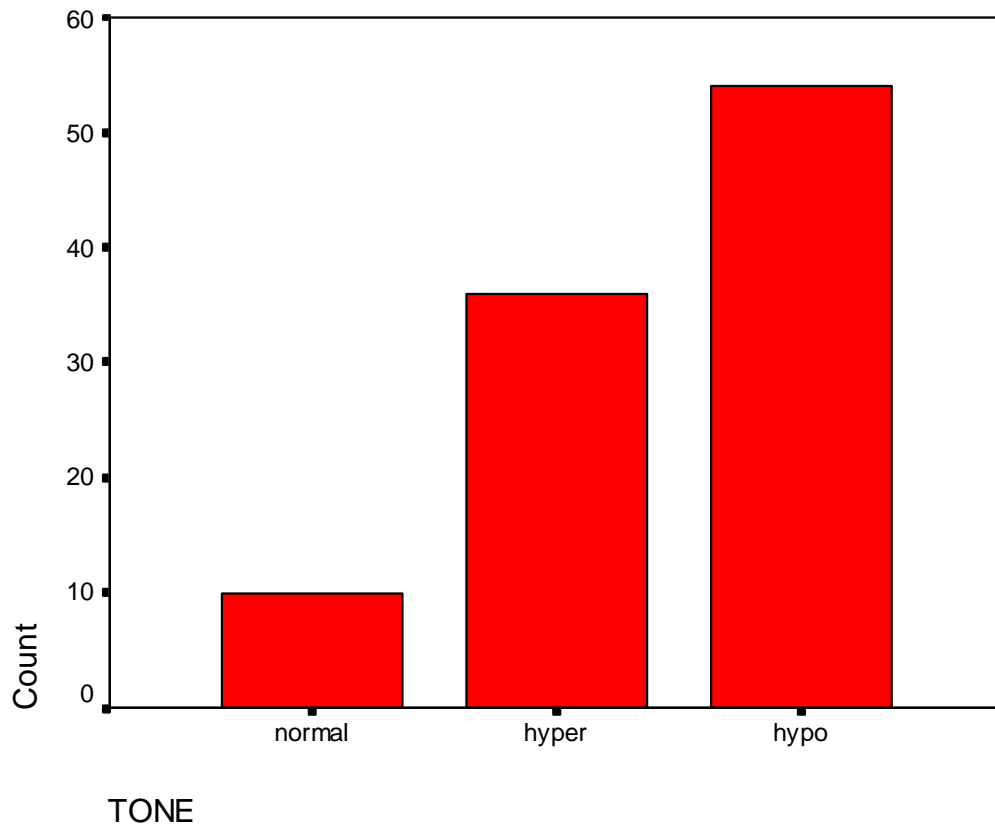


Figure No 8
The states of the reflexes

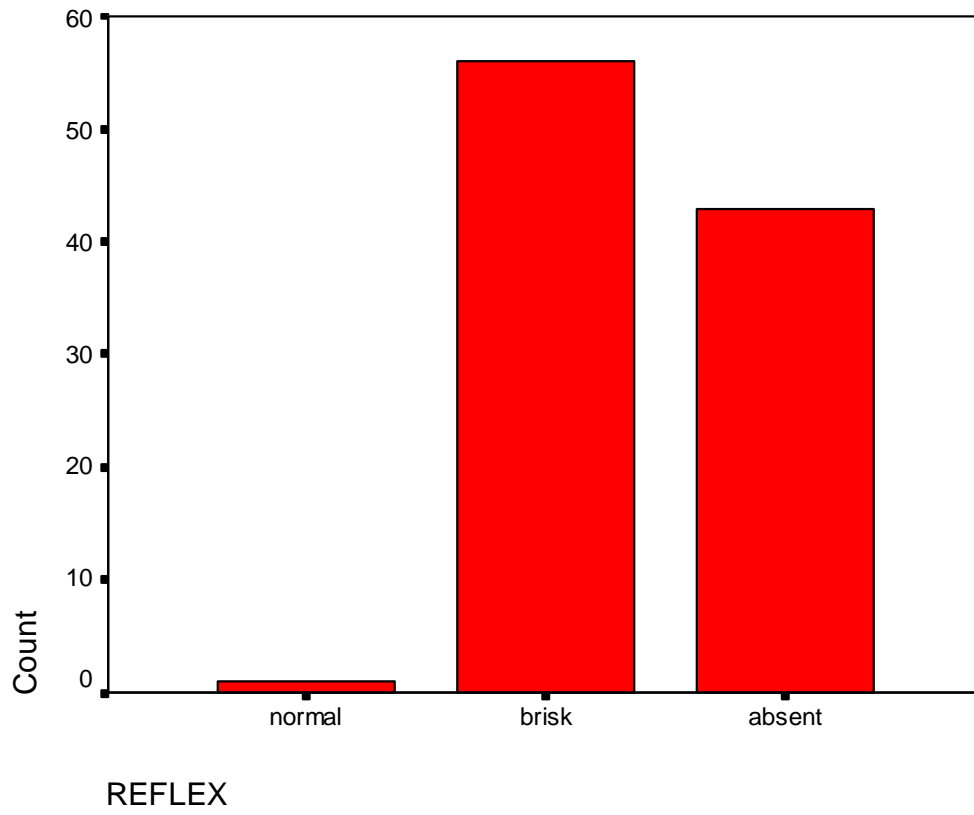


Figure No 9
The power grades

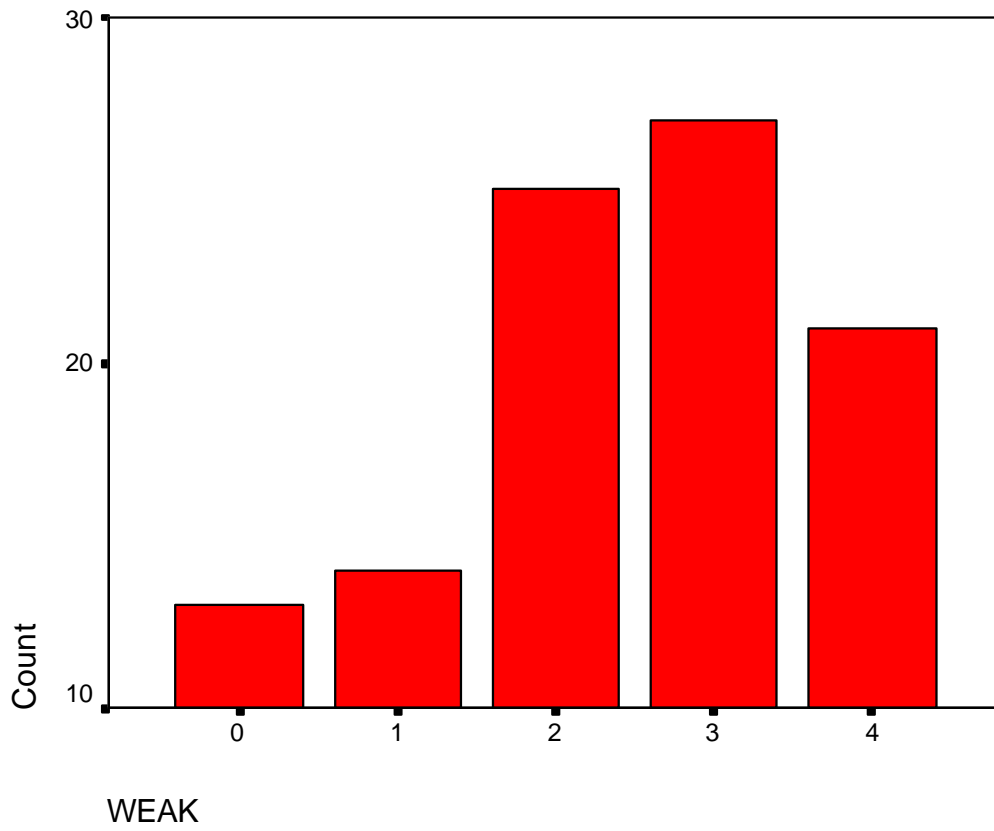


Figure No 10
The planter response

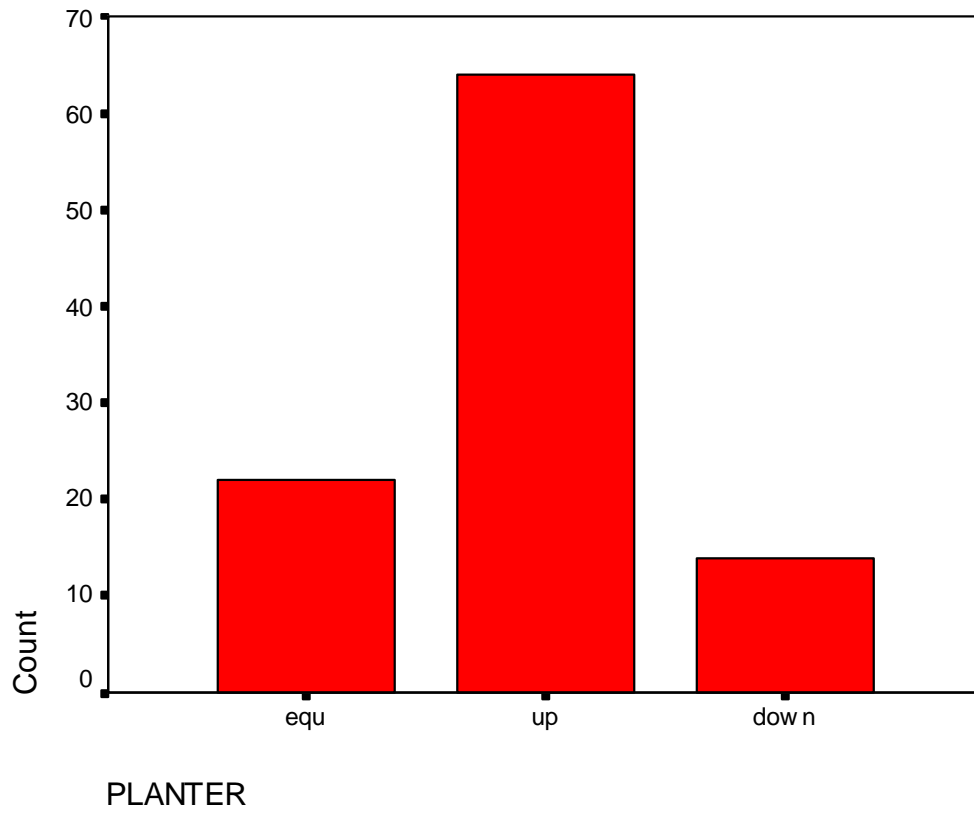


Figure No 11
The states of sensation

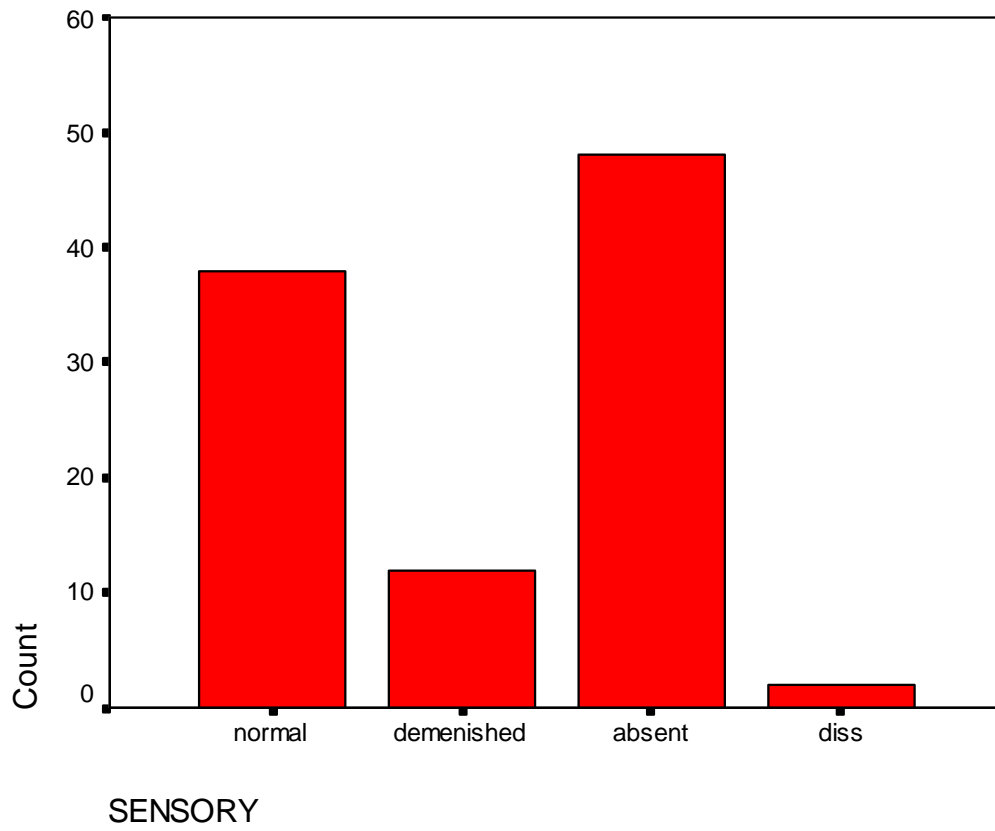


Figure No 12
The states of the abdominal reflex

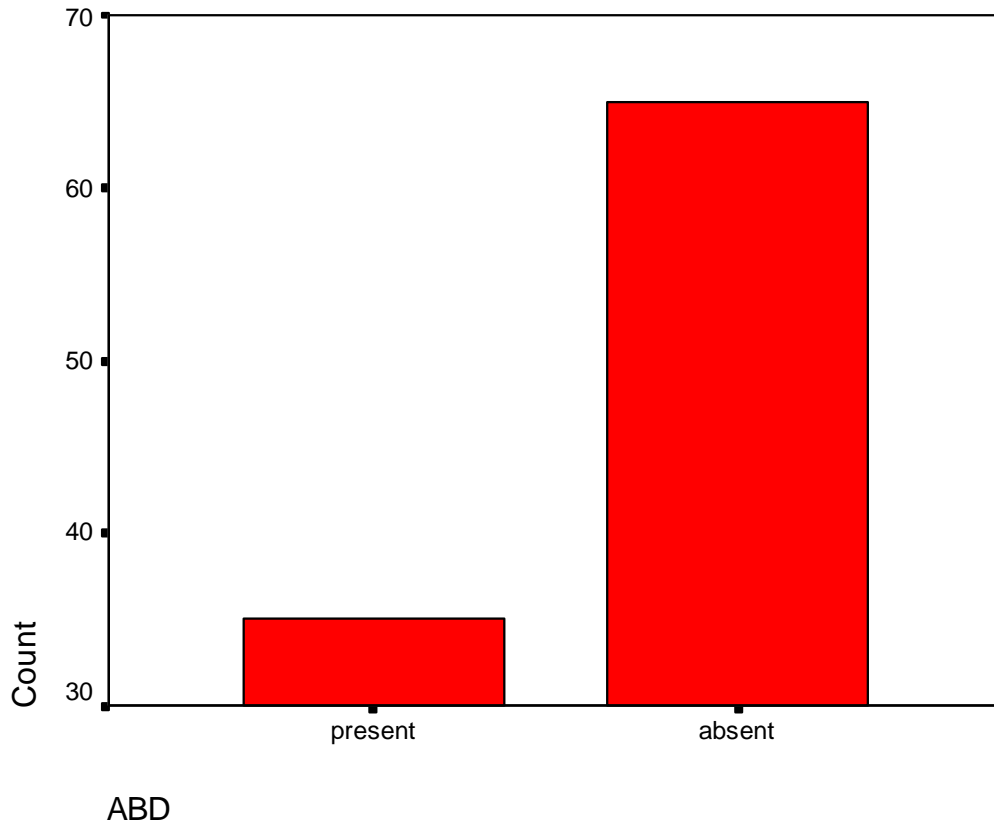


Figure No 13
The degree of disability

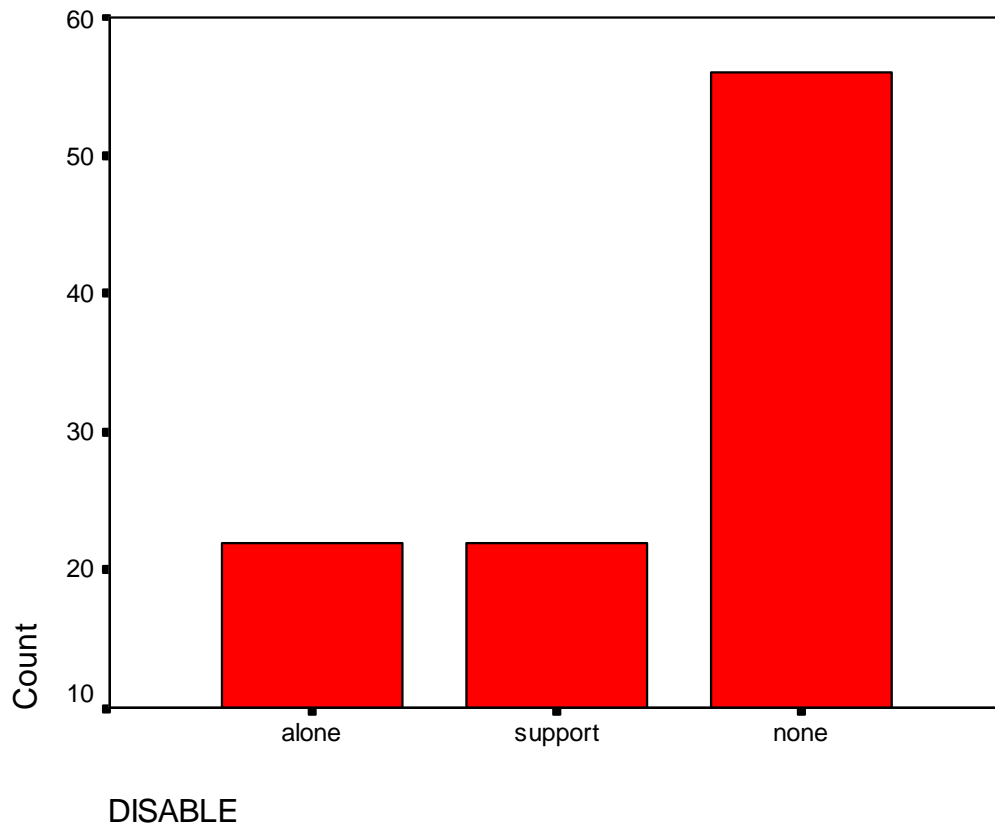
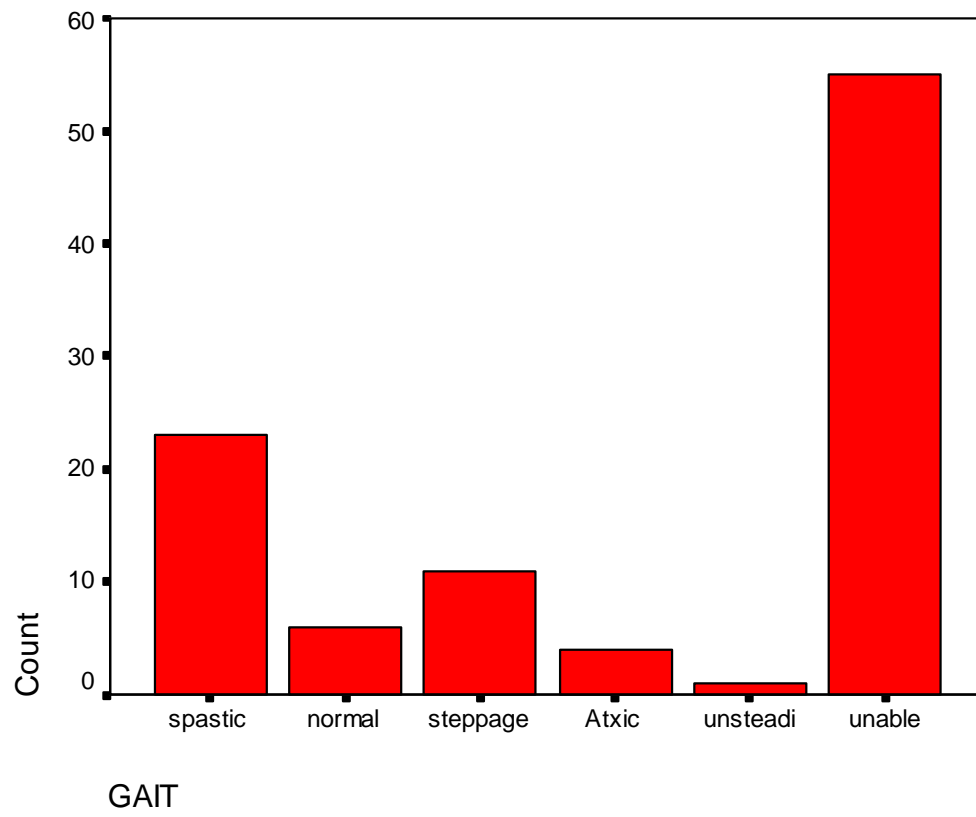


Figure No 14
The gait



Discussion

Many studies were carried out in respect to individual etiologies of paraplegia through out the world .Infectious and demyelinating etiologies constituted the bulk in most studies . 23% of the cases in the study lie in the age group 16-25, and the same percent in the age group 25-35 followed by 22% lie in the age group 35-45% , 15% in the age group 55-65 , 8% in the age group 45-55 , 7% in the age group 65-75 and 2% in the age group 75-85 yrs . In conclusion , approximately half of the patients(46%) lie in the age range 16-35 , who are the most active sector of the community . Males (56%) are more affected than females (44%) . Table No 1 .

The majority of patients from Khartoum and the surroundings , 40% followed by Al Jazira 19% , the north province 14% , the east 13% , the west 12% and the north 2% . This can be explained by the recent migration toward Khartoum in addition to the civil war in the south , but no significant geographical etiologic factor is suspected .

Weakness is the presenting complains in 92% of pts , followed by sensory symptoms in form of tingling or numbness in 38% but examinations showed 48% had absent sensation , 12% had diminished sensation and 2% had dissociated sensation . Table 3 & Fig 11.

Urinary sphincter dysfunction was reported in 46% , while bowel sphincter dysfunction is reported in 28% .

Backache was reported by 45% and root pain by 21%.

Concomitant upper limb weakness was reported by only 9% but actual weakness was detected in 40% . Only 2% present with cortical symptoms (convulsions and confusion) , and 10% had cranial nerves symptoms while cranial nerves signs were seen in 19% . This can be explained by the approximating incidence of the etiologies (demyelinating diseases) . The facial nerve is the commonly affected one (13%) , followed by the optic 6% , the 6th , 9th and 10th 2% and the 11th and 12th 1% for each .

P.H of neurologic illness was reported in all pts who had M.S (9%) , family history was reported in 5% , 4% had F.S.P and 1% had M.S . No history of recent vaccine was reported .

Systems concomitantly involved were ; the respiratory 4% C.V.S 3% , abdomen 5% (liver , spleen , ascites and masses) , and other systems 9% (thyroid , breast , skin and M.S.S) . This is due either a systemic disease manifested by LL weakness or a malignant disease causing metastatic compression to the cord .

Wt loss is reported in 69% of pt with weakness due to secondary tumors and 36% in pts with Pott's disease of the spines .

The power varied from 27% in grade III , 25% in grade II , 21% in grade IV , 14% in grade I and 13% in grade 0 . This is fitting with the degree of disability which was 22% in patients who experienced no disability , the same as patients who are able to

walk supported and 56% of patients are wheel chair bound . Fig 9 &13 .

The tone was spastic in 36% , normal in 10% and flaccid in 54% . The percentage of brisk reflexes is a little bit higher (56%) , absent in 43% and normal in 1% . This is because some patients still had brisk reflexes despite hypotonia . Fig 7 .

The planter response was up going in 64% , mute in 22% and down going in 14% . This reflects the high incidence of U.M.N lesions among the study populations (69%) . Fig 10 .

Romberg's sign was positive in 3% , all had S.C.D.C and cerebellar ataxia was an accidental finding in 2% of patients , both had an M.S .

In this study , A.I.D.P constitute the major cause of paraplegia (19%) , this goes with the increasing incidence of acute demyelinating poly-neuropathy in many countries . ⁽³⁵⁾

As in many other studies no age group is exempted . No role for gender in the incidence of the disease . ⁽³⁵⁾

Antecedent febrile illness was reported by 68% of patients , this high event may reflect a playing role for malaria (the commonest febrile illness in Sudan) as an etiologic agent . But antecedent gastroenteritis is reported in only 21% (4/19) , and respiratory infection in 10% (2/19) .

Concomitant upper limbs was detected in 79% of pts (15/19) this reflects the relatively late presentation , since upper limbs involvement occur late (90% by the end of the 4th week). 52

Facial weakness had occurred in 47% , while 6th nerve weakness in only one pt , so for the 9th , 10th and 11th . This bear no variation from literature reports .⁽³²⁾

C.S.F analysis was significantly abnormal in 95% of pts compared to 65-85% in other studies , this as mentioned can be explained by the late presentation .⁽³⁴⁾

A.T.M was found to be the next common cause (15 cases)

The incidence peaked in the age group 16-25 , (47%) , compared to a peak incidence in the age 10-19 in the literature , the rest was shared between other groups . No role for gender was noticed . So for geographycal distribution .

Weakness in the LL 100% , bladder disturbance 100% , bowel dysfunction 70% and sensory symptoms 70%, were the common presenting complains . Most patients (67%) were severely disabled with a power of grade III and less . The tone was equally flaccid or spastic , and the planter was up going in all patients . Sensation was absent in 80% and diminished in 20% .

Fever was reported by 73% of patients , at the same time leukocytosis was detected in 67% of patients and an E.S.R above 40mm/hr in 60% , this is further supported by an abnormal C.S.F findings in 53% , reflecting a possible infectious cause .

M.R.I was done to all patients . It was normal in one patient .

The abnormality involved the lower dorsal spines in 7patients ,

the cervical in 4 patients , the upper dorsal in 3 patients and extensive cervicodorsal in 1. High signal intensity , and more than one segment involvement was noted .

Syphilitic myelitis is nowadays is becoming a rare , the study actually couldn't pick up a case , this possibly due to the wide use of antibiotics .

Since tuberculosis is still prevalent in the developing countries , it is not surprising to report Pott's disease as the third common cause of paraplegia . 12 cases were reported .

The study noticed the disease to be rare among the age group below 25 yrs , in addition to the high incidence (9 out of 11) in male sex . Only one case came from Khartoum , this can be explained by the relation of the disease and poverty .

weakness and backache are the presenting complains in 10 of patients , whereas fever is reported by 8, sphincters dysfunction by 7 for urinary and 5 for both bowel and urinary . Numbness or tingling were reported by 6 . the Upper limbs was involved in only 2 patients . This is due to the rare involvement of the cervical spines in Pott's disease . ⁽²²⁾

These patients were noticed to have a better grade of power and a spastic tone compared to those with A.T.M . The reflexes were brisk in 9 , the planter was up going in 11 and mute in one . The sensation was absent in 9 and diminished in 3 . The gait was spastic in all pts who could walk 7 patients . Back tenderness and / or deformity were seen in 8 patients .

Mantoux test was positive in 8 , no result of a positive sputum was obtained and only one patient had a radiological evidence of an antecedent pulmonary Koch .

Plain X –ray of the spine still of help . It revealed an evidence of the disease in 10 patients ,whereas M.R.I could pick up the pathology in all . Abscess and canal stenosis were seen in 7patients , debris and inflamed cord in 3 and multiple pathology is seen in two patients .

Tumors (secondary 13cases and primary 3) constituted a major etiological factor . The secondary tumors originated from the lymphatic (2) , the WBCs (1) , the plasma cells (2) , the breast (2), the prostate (2), the thyroid (1) , the bowel (1) and undetermined primary (2) . The primary tumors were meningioma , astrocytomas and chordoma .No specific neurological features for these tumors . Weakness , root pain and backache were common especially with secondary one (all patients) and (2/3) with primary . M.R.I was the diagnostic tool used .

M.S is a clinical conundrum , we could pick up 9 cases the diagnosis based on a P.H of neurological illness in 8 cases and brain M.R.I in the ninth one who had a normal spinal M.R.I . The weakness was noticed to be mild (the power was grade III and more) , only one patient had sensory disturbance and an other one had cerebellar signs . No sphincters disturbance was

reported . Concomitant upper limbs weakness was seen in 3 of them , and cranial nerves affection was seen in 4 .

O.C.B was not done and the diagnosis was supported by a C.S.F which was abnormal in all patients , a spinal M.R.I which was abnormal in 7 and a brain M.R.I done for 2 and was found to be abnormal in one .

S.A.C.D was detected in 4 patients , the hemoglobin was 8-10 in three of them and less than this in the fourth one . The diagnosis was confirmed by bone marrow megaloblastosis . All these patients are males and lie in the age 25-55 . Weakness was the presenting complain in three and numbness in the fourth one . Three of them had gloves and stocks sensory disturbance and one had only absent vibration sense . Upper limb involvement was detected in all of them , and optic atrophy was seen in one . Diabetic amyotrophy is an incapacitating complication of D.M , it was seen in 4 patients , their history of diabetes dating 7-21 yrs , all of them had wasting , weakness and hypotonia . Pain in the legs was reported by three, and backache by one , and only one had optic atrophy . The C.S.F protein was high in all of them . Two of them had proteinuria .

Heredodegenerative disorders was seen in 7patients of the age 16-35 yrs , four of them had H.S.P (3 males and one female) , and one (female) had metachromatic leukodystrophy . A family history of the disorder was reported by three of the former group . For H.S.P the power was still maintained (3-4) and there was

marked spasticity , no sensory , cerebellar , cranial nerves or cortical signs were seen . That patient with metachromatic leukodystrophy had a history of weakness progressed over yrs , she had a flaccid tone , up going planters and intact sensation and she was wheelchair bound . Imaging studies and C.S.F analysis were normal in all of these pts . Two patients had A.L.S , with weakness in the four limbs , fasciculation was seen in both , and both patients aged above 55years .

Cord infarct was detected in three patients . weakness , up going planters in all of them , dissociated sensation in two and absent in one who had an A-V malformation possibly causing mass effect and pressing on the cord . One patient had in addition a wheezy chest , eosinophilia and a significant ANCA titer , accordingly the diagnosis of Churge Strauss was decided .The third patient had possibly arteriosclerosis , as evident by hypertension . ⁽⁶⁹⁾

Cauda equina was diagnosed in three patients , flaccid weakness , absent sacral sensation and sphincters disturbance were shared by all of the them . Dorsolumbar X- ray was abnormal in one and the MRI was abnormal in two .

C.I.D.P was the diagnosis in three patients , they bear some resemblance to the cases with A.I.D.P but the history was long and wasting was noted in these patients . The C.S.F showed albuminocellular dissociation .

Spondylosis with canal stenosis was seen in three patients aged above 50 years , in two of them the lesion was cervical and was lumbar in the third one , the sphincters were involved in all . X – ray spine could show the lesion in two and the M.R.I showed the pathology in all of them .

Conclusion

- * Male to female ratio was 1.27:1 , most of the cases from the centre
- * Approximately half of the patients present with gradual onset .
- * Weakness is the presenting complain in the majority though few instead have stiffness or sensory symptoms as the presenting complain .
- * Urinary sphincter dysfunction occurred in nearly half of the patients whereas bowel sphincter dysfunction occurred in one third .
- * Wasting was seen in less than one third of patients .
- * The tone was flaccid in more than half of the patients and spastic in more than one third of patients .
- * The planter was up going in two thirds .
- * More than half of patients are wheelchair bound , less than one third could walk with support , and the same number could walk unsupported .
- * M.R.I is of great help in picking up cord pathology , and the role of the plane x- ray cannot be ignored .
- * A.I.D.P , A.T.M , metastatic tumors and Pott's disease are the major etiologies of paraplegia .

Recommendations

- * El Shaab teaching hospital is the special hospital concerned with rehabilitation and care of the neurologic patient who is in great need to that care , so supplying the neurology unit there with the deficient equipment and diagnostic tools , will help these patients .
- * Provision of well trained nursing staff is of a critical importance to the neurologic patient .
- * Establishing an I.C.U to care for patients with complicated neurologic illness .
- * Rehabilitation centers and societies to help these patients with aiding tools , wheelchairs and air matters to the wards .
- * Provision of occupational therapy , psychotherapy and socioeconomic supports for patients with paraplegia .

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PATTERNS OF CLINICAL PRESENTATION

Name----- Age--- Sex Residence ---

Tribe----- Occu.----- Marital Status-----

-Symptoms preceding onset :

- Fever	yes	no
-Wt loss	yes	no
-Backache.	yes	no
-Cough.	yes	no
-Diarrhea.	yes	no

-Symptoms at the onset:

Gradual	Rapid	Sudden
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1-Sensory :

-Paraesthesia	yes	no
-Hyperesthesia	yes	no
-Root pain	yes	no

2-Motor:

-Weakness	yes	no
-Unsteadiness	yes	no
-Stiffness		

3-Sphincter disturbance ;

-Urinary	yes	no
-Fecal	yes	no

4-Cortical

- Consciousness	yes	no
-Seizures	yes	no

-Others –specify

5-Cranial nerves ;	yes	no
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PH

-S/C	yes	no
-Neurologic illness	yes	no
-Syphilis	yes	no

F.H :

-S/C	yes	no
-Neurological disease	yes	no
-Koch	yes	no
-Drug history	yes	no
-recent vaccine	yes	no

Physical examination :

- Fever	- Anemia
-Dehydration	-Dyspnoea
-Pulse	-BP

C.V.S:

Chest :

Abd :

-Others

Nervous system :

- Higher cerebral function

-Cranial nerves

-Upper limbs

power

tone

reflexes

sense

-Trunk

sensory level

weakness

- lower limbs

wasting

X-ray spine

M.R.I

Bone marrow