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Polyradiculoneuropathy , Anterior Horn cells , Anti Snake Venom - ASV, Acute inflammatory demyelinating polyneuropathy- AIDP .

QUADRIPARESIS - IT'S APPROACH AND MANAGEMENT IN THE ED

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ABSTRACT

Introduction : Vast majority of disease conditions are potentially reversible if recognized on time . Paresis or weakness may be a symptom of any pathology , which includes neurologic, non neurologic conditions and trauma. The clinical approach of a multifactorial disease considered one of the most common neurological emergencies where prognosis depends on early and accurate diagnosis gains importance .[3] We evaluate cases with quadriparesis in the midst of a busy shift very frequently , although it doesn't get much spot light , ED diagnosis and management of such cases requires a systemic approach based on focussed history , examination and workup as there are several easy to miss differentials leading to functional impairment resulting in long term consequences for patients , posing a great challenge to emergency physician .So what actually should be considered in the evaluation and treatment of such cases in the ED is a question to be answered .[9] This article endeavours to take few steps forward for steady clinical approach, evaluation and management of quadriparesis .

BACK GROUND :
Disorders of neuromuscular unit , processes involving brainstem and spinal cord result in clinical presentations ranging from subtle symptoms to acute respiratory failure . A clear understanding of pathophysiology , helps to determine the level of nervous system affected ,this also facilitates an approach based on signs and symptoms , findings of which direct the urgency of diagnostic testing and treatment .The clinical approach to a patient with acute neuromuscular weakness is aided by focussed clinical evaluation there by determining the location of lesion , differential diagnosis of most common disorders that affect the area in particular and diagnostic stratergies like neuroimaging , nerve conduction studies etc.[9]

Here we present a series of cases with quadriparesis having varied clinical presentation , its approach and management in the ED .

Case 1 :
A 72 year male with chief complaint of progressive weakness in all four limbs and tingling numbness since one week gradual in onset , rapidly progressive with history of fever and no other associated symptoms .
Primary Survey: Airway -patent, Breathing -RR - 14 , Spo2- 95 %, Circulation - HR -90 , BP- 130/80 , Disability -GCS – E4V5 M6, Pupils – normal size ,reaction to light - +
Ample History - nill significant .
Secondary Survey: General examination : poorly built and nourished ,mild pallor + Systems review- CVS - S_1 ,S_2 +, RS - Bilateral air entry present, no added sounds , GIT- P/A Soft , CNS - Higher mental functions - Normal, Cranial nerves - Normal ,Motor ,Tone- decreased bilateral, Power- Right – 3/5 Upper limb, 2/5 Lower limb ,Left - 3/5 Upper Limb , 2/5 Lower limb ,Reflexes – Deep tendon reflexes absent in all four limbs , Plantars – Bilateral extensor , Sensory - Normal , Bowel Bladder involvement-No ,Cerebellar signs and Meningeal signs – absent ,

Provisional Diagnosis: Guillain Barre syndrome ,Cerebro vascular accident , Potts Spine , Space occupying lesion .

Investigations: Complete blood picture, Serum electrolytes ,Computerized tomography scan of brain , Magnetic resonance imaging of cervical and dorso lumbar spine .

Final Diagnosis : Quadriparesis secondary to Koch' s Spine , ? Metastasis

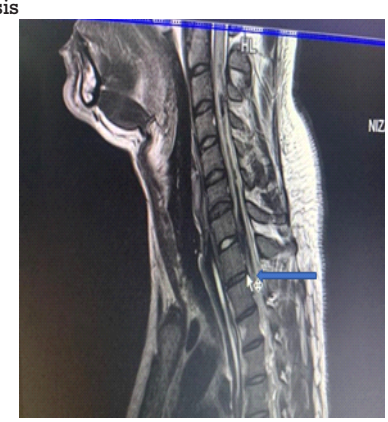

Fig -1 Retrolisthesis of C5 over C6 , lytic destructive lesion involving C5 , C6 causing cord compression - probable infective etiology , likely Koch's or possibility of metastasis .

Case 2 :
A 30 year old male patient with chief complaint of weakness of the upper limbs and lower limbs since 10 days , vomitings -3 episodes since 1 day ,shortness of breath -2 days with no other associated history.

Primary Survey : Airway -patent, Breathing -RR -30 , Spo2- 95% ,

Circulation -HR - 90, BP- 90/60 , Disability - GCS –E 4V5 M6 , Pupils -normal size , reaction to light +

AMPLE History – History of Hypertension ,Diabetes Mellitus with similar complaints in the past , history of administration of ASV in a local hospital.

Secondary Survey: General examination -moderately built and nourished ,no other signs Systems review – CVS - S1 ,S2 +,

RS - Bilateral air entry present ,no added sounds , GIT- P/A Soft ,CNS - Higher mental functions - Normal, Cranial nerves - Normal ,Motor -Tone decreased bilaterally, Power -Right -2/5 Upper Limb , 1/5 Lower Limb, Left - 2/5 Upper Limb , 1/5- Lower Limb ,Reflexes – L limbs - absent , U limb present + , Plantars – bilateral extensor , Sensory – loss below T12 present , Bowel Bladder involvement- incontinence of urine present , Cerebellar signs -absent ,Meningeal signs – absent .

Provisional Diagnosis : Cerebro vascular accident, Snake Bite ,Guillain Barre syndrome. Investigations : Serum electrolytes , Computerized tomography of Brain , Nerve Conduction studies ,Magnetic resonance imaging of spine.

Final Diagnosis : Quadriparesis secondary to Acute Haemorrhage into the spinal cord - CVA

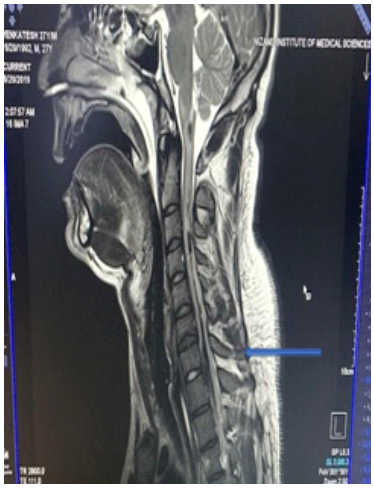

Fig -2 Meninges in the dorsal spinal region are thickened and hypointense - S/o Haemorrhage

Case 3 :
A 35 year old male with history of fall from tree , followed by inability to walk and loss of sensation in both lower limbs and abdomen below the region of umbilicus, pain tenderness in the chest region associated with history of vomiting and no other significant history .

Primary Survey: Airway – Patent, Breathing -RR- 22 ,Spo2 - 97%, Circulation- HR -96, BP – 100/70 , Disability - GCS- $E_4V_5M_6$, Pupils – normal size ,reaction to light- + Ample History- nill significant

Secondary Survey: General examination : well built and nourished , no other signs. Systems review- CVS - S1 ,S2 +,RS –Bilateral air entry present ,Tenderness + left side , No added sounds ,GIT- P/A Soft, CNS -Head - No Evidence of Injury ,C Spine tenderness – absent , Chest compression test positive ,P/A – Soft ,no tenderness ,pelvic compression test – negative , CNS - Higher mental functions - Normal, Cranial nerves - Normal ,Extremities Motor -Tone :Normal ,Power :Upper limb Right- 5/5, Left- 5/5 ,Lower limb -Right -1/5, Left -2/5, Sensory - loss Below T12 , L1, Reflexes - Upper limbs 1 + , Lower limbs absent , All peripheral pulses + , Thoracic and lumbar spine region - tenderness +, Bowel Bladder involvement - Anal tone lost , Exposure – No visible injuries .

Provisional Diagnosis : Traumatic paraplegia with Blunt Injury Chest.

Investigations : ABG , Chest Xray , Computerized tomography Brain ,Magnetic resonance imaging - Dorso lumbar Spine .

Final diagnosis : Traumatic paraplegia secondary to lumbo sacral spine injury with Blunt Injury Chest.

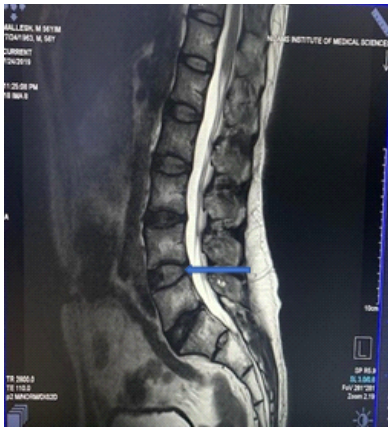

Fig -3 Posterior annular tear at L5 – S1 ;Diffuse disc bulge at L3-L4 ,L4-L5, L5- S1 , causing thecal sac indentation and neural foramina narrowing

Case 4:
Chief complaint: 52 year female being referred from a primary care hospital ,with progressive weakness of all four limbs , shortness of breath – 2 days, inability to speak since 3 days, history of present illness : weakness associated with dysphasia + , no history of headache ,vomitings , seizures ,backache ,bowel bladder incontinence ,history of fever, cough , shortness of breath + ,

Primary Survey :Airway – Protected , Breathing – RR-35 , Spo2- 95 % on ventillator , Circulation : Pulse - 116 , BP - 130/70 ,Disability GCS – E 3VT M4 , Pupils –Normal size , reaction to light + .

AMPLE History :No significant allergic history , History of administration of IV immunoglobulin in a local hospital , Past History – Hypertensive, Hypothyroid

Secondary Survey : General examination : moderately built and nourished ,Pallor present, Systems review : CVS -S1,S2 +, RS- Bilateral air entry present and GIT – P/A soft ,CNS : Higher mental functions – semiconscious ,disoriented ,Cranial nerves - Normal , Motor - Tone decreased bilateral ,Lower limbs < Upper limbs, Power- right 3/5 Upper Limb, 1/5 Lower Limb , left- 2/5 Upper Limb , 1/5 Lower Limb , Reflexes – absent , Plantars – Bilateral flexor ,Sensory - Normal , Bowel Bladder involvement- No ,Cerebellar signs – could not be assessed , Meningeal signs – Absent ,

Provisional Diagnosis : Systemic Envenomation ,Hypokalemic paralysis ,Cerebro Vascular Accident ,Guillian Barre Syndrome with respiratory failure on ventilator.

Investigations : ABG ,Serum electrolytes, Nerve conduction studies ,Magnetic Resonance Imaging of spine .

Final Diagnosis : Guillain Barre Syndrome with respiratory failure

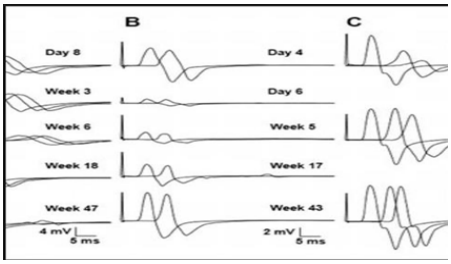

Fig -4 Nerve Conduction Studies

- When GBS is suspected ,electro-physiologic studies are essential to confirm the diagnosis and exclude its mimics.

- The finding of multifocal demyelination on early electrodiagnostic testing (or repeated a week later) is extremely helpful in confirming the diagnosis of AIDP, with high sensitivity and specificity .

DISCUSSION :

ED patients with nonspecific complaints such as weakness can be overlooked or under diagnosed so a focused history and examination following the primary survey , helps in early diagnosis and prevent the complications. So ,it is imperative to have a clinical approach rather than relying on electrophysiology , lab parameters or imaging to narrow down the differential diagnosis .

Primary Survey [9]:

- Ensure protection of airway and adequate ventilation with cervical spine stabilization (Especially if there is respiratory muscle weakness, shallow respiration, dysphagia or weak gag reflex)
- Check and support: BP and Heart Rate
- Immobilize neck if history of neck / head trauma
- Send electrolytes and get an ECG- to look for hypo/hyperkalemia

History (6):

As a first step , assess if the symptoms are acute or chronic , unilateral or bilateral , symmetrical or not . Next important thing is how the weakness manifests , Is it continuous or episodic , if it was a traumatic event , presence of systemic symptoms such as headache , muscle pain , vision loss, altered mental status , bowel bladder involvement are the clues in the history for further work up.

Secondary Survey[5]:

The following are the clinical examination clues :

Step 1: Is it an upper motor neuron (UMN) or a lower motor neuron (LMN) lesion?

- Clues to UMN lesion (mostly a spinal cord lesion) could be presence of either:
- Brisk reflexes
- Extensor plantar
- Definite sensory level
- Bladder or bowel involvement
- A LMN lesion will have absent reflexes with mute plantar.
- Frequently, an acute onset spinal cord lesion may present with absent reflexes due to spinal shock. But the presence of other differentiating features (extensor plantar, definite sensory level and bladder involvement) help to localize the lesion to spinal cord.

Step 2: If It Is A LMN Lesion What Is The Pattern Of Weakness?

- Is it proximal or distal?
- Lesions localized to the following anatomical locations cause proximal, mostly symmetrical weakness in AFP.
- Muscl
- NMJ
- Polyradiculoneuropathies

Poly neuropathy will present with distal, mostly symmetrical weakness. Lesions localized to the anterior horn cells can present with symmetrical or asymmetrical, proximal, distal or a combination of both.

The reflexes will be absent in both the conditions.

Step 3: Are The Reflexes Preserved?

Localization of lesions with proximal weakness and preserved reflexes include muscle or NMJ.

Fatigability (appearance of weakness with repeated use) is a prominent feature in NMJ disorders especially myasthenia Reflexes would be absent in:

- Anterior horn cell disorders
- Polyradiculoneuropathies
- Neuropathies

Step 4: Are Sensations Preserved?

Neuropathies present with sensory involvement. Polyneuropathy presents with glove and stocking sensory involvement while mononeuritis multiplex would present with patchy sensory loss. Polyradiculoneuropathies may or may not have a sensory loss. Some GBS variants may have sensory loss. cauda equina may present with a sensory loss in a radicular distribution.

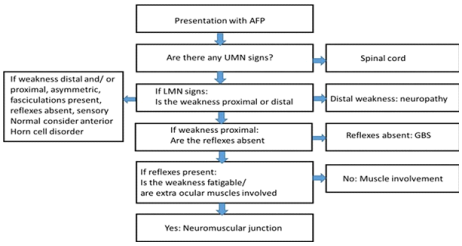

Table : 1 Flow Diagram For Clinical Examination Of Quadriparesis

The initial ED differential diagnosis of quadriparesis with or without alteration of sensorium , include [2]:

1. Systemic Envenomation :

Snake Bite , Botulism ,Tick Paralysis , Heavy metal poisoning , Alcoholic intoxication , Scorpion sting.

2. Guillain Barre Syndrome :

3. Electrolyte Disturbances :

Hypercalcemia , Hyper and Hyponatremia, Hypokalemia , Hyperkalemia Hypermagnesemia and Hypophosphatemia.

4. Central Nervous System Disorders :

Transverse Myelitis , Multiple Sclerosis, Ischemic , Vasculitic , tumours, traumatic cord lesions.

5. Disorders Of Neuromuscular Junction :

Myasthenia gravis , Lambert Eaton syndrome .

6. Muscle Disorders :

Myopathies and channelopathies .

7. Infectious Causes :

Bacterial osteomyelitis ,Spinal abscess , Poliomyelitis, Diphtheria, Dengue fever, HIV, Lyme disease.

8. Vascular Causes :

Anterior spinal artery occlusion , AV malformation .

9. Vertebral Disease :

Vertebral disc prolapse, Pagets disease , Spinal deformity, Spondylosis .

10 . Others :

Degenerative Motor Neuron disease , Decompression syndrome Hereditary spastic paresis , Epidural , Intra medullary haemorrhage

Treatment [3]:

1. Meticulous Supportive care - Respiratory, bulbar weakness ,

2. Spinal Immobilization- if required , correction of shock due to reduced vascular tone (spinal cord disease) ,autonomic instability with intravenous fluids and vasopressors .

3. Specific therapy: (initiated In -Consultation with Primary

department)

4. Hypokalaemia: Intravenous potassium correction Guillain Barre syndrome: IVIG, 2 g/kg over 2–5 days

Transverse myelitis: IV methyl-prednisolone 10–30 mg/kg, daily Compressive myelopathy & Acute traumatic : Spinal immobilization, steroids . Spinal cord injury - Neurosurgical intervention Dermatomyositis, Myasthenia Gravis: Immuno modulation

5. Prevention of nosocomial infections and complications of immobilization .

Acute Flaccid Paralysis in Adults

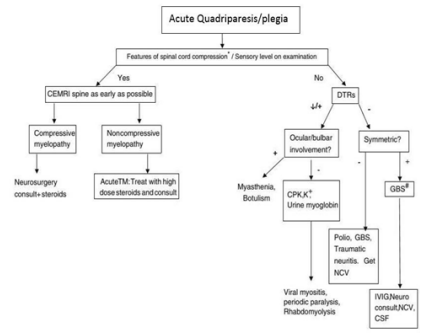

Table : 2 Algorithm for ED Management of Quadriparesis

Evidence Based Teaching Points – Key Notes

1. Quadriparesis in ED represents a diagnostic challenge with multi dimensional etiology.

2. A protocol based approach accompanied by early diagnosis ,timely intervention and stabilization would preserve the neurologic function and prevent complications.

REFERENCES

1. Winner SJ, Grimley Evans J (1993);Guillain-Barré syndrome in Oxfordshire: Clinical features in relation to age. Ageing 22: 164-170.
2. Klingon GH (1965);The Guillain -Barre syndrome associated with cancer. Cancer 8:157-163.
3. Crone C, Krarup C (2007);Diagnosis of acute neuropathies. J Neurol254: 1154-1169.2. 4.Miller FW, Leitman SF, Cronin ME, Hicks JE, Leff RL, Wesley R, Fraser DD, Dalakas M, Plotz PH(1992); Controlled trial of plasma exchange and leukapheresis in polymyositis and dermatomyositis. N Engl J Med 1992; 326: 1380-1384 [PMID: 1472183 DOI: 10.1056/ NEJM199205213262102].
5. Green DM, Ropper AH(2001);Mild Guillain-Barré syndrome. Arch Neurol 58:1098-1101 [PMID: 11448299 DOI: 10.1001/archneur.58.7.1098].
6. Young GB, Hammond RR(2004);A stronger approach to weakness in the intensive care unit.Crit Care 8:416-418 [PMID: 15566605 DOI:10.1186/cc2961].
7. Zifko UA, Zipko HT(1998);Bolton CF. Clinical and electrophysiological findings in critical illness polyneuropathy.J Neurol Sci 159: 186-193[PMID: 974140 DOI: 10.1016/ S0022-510X(98)00164-6].
8. Chiò A, Cocito D, Leone M, Giordana MT, Mora G, Mutani R.(2003);Guillain-Barré syndrome: a prospective, population-based incidence and outcome survey. Neurology 60:1146-1150 [PMID:12682322 DOI:10.1212/01.WNL. 0000055091.96905.D0].
9. Rosen's Emergency Medicine :Concepts and Clinical Practice ,7th Edition ISBN -13:978-0-323-05472-0, Volume 2,Part Number- 9996073521.
10. Reed AM, Ytterberg SR.(2002); Genetic and environmental risk factors for idiopathic inflammatory myopathies. Rheum Dis Clin North Am 28: 891-916. [PMID: 12506777 DOI: 10.1001/ jama.2010.1977].