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Review Article

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A CASE OF EPIDURAL ABSCESS IN IMMUNOCOMPROMISED BACKGROUND

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ABSTRACT

Spinal epidural abscess (SEA) is still a rare but dreaded complication of infection involving the spine especially in late presentation with immunocompromised background. Diagnosis of abscess formation is often delayed, specifically in the preliminary stages of the disease process before patients present with neurological symptoms. Classical symptoms of SEA are characterized by fever, back pain, and neurological symptoms, which are described as a classical triad, but this collection of symptoms is manifested in only about 10% of cases. Early suspicion of the disease process, initiation of appropriate and early treatment depending on the clinical finding and radiological investigations can often improve the situation even in late presentation where paralysis has already set in.

INTRODUCTION

Spinal epidural abscess (SEA) is an uncommon disease featured by the accumulation of pus in the epidural space along different areas of the spinal cord in continuity or in pockets causing compression of the spinal cord and spinal roots leading to sequel of abscess formation. During the 1970s, its incidence was found to be around 0.2 to 2.0 cases per 10,000 hospital admitted patients.[1] However, with the improved and appropriate use of magnetic resonance imaging (MRI), the detection rate of abscess along with the suspected aetiology is improving, and consequently, the number of reported cases is increasing day by day.[1] Lower limb weakness and/or bladder and/or rectal dysfunction often signifies SEA and may often be the presentation of the cases as in our case was.[1]

A 33-year-old male PLHIV, non-hypertensive, nondiabetic, resident of Aurangabad, Bihar, jewellery worker by occupation previously employed at Mumbai presented with gradual onset bilateral lower limb weakness for last 3weeks, urinary incontinence for last 10 days and gradual onset upper limb weakness for last 7days associated with low grade intermittent fever with shortness of breath for last 2 months.

Patient has been diagnosed recently as having immunocompromised status with HIV seropositive status started on ART from Katihar Medical College ART centre with TLE (Tenofovir, Lamivudine and Efavirenz) from 20/8/2020.Patient is addicted to smoking and alcohol for last25 years. H/o high risk sexual behaviour present.

NO history of loose stool, pain abdomen, recent travel, headache, loss of consciousness, convulsion, recurrence of such episode in the past, throat pain, burning micturition, chest pain, palpitation, joint pain, rash, difficulty in smelling, double vision, blurring of vision, loss of sensation over face, drooling of salivation, difficulty in chewing foods or pooling of food in the mouth, difficulty in deglutition of solid or liquid food.

CLINICAL FINDINGS

On examination patient was found to be conscious oriented with time place and person lying in helpless attitude on bed unable to move, severely malnourished and cachectic. Patient was pale without any cyanosis, clubbing, icterus, oedema, engorged neck veins or enlarged neck lymph nodes.

BP-90/60 mmHg
Pulse-80/min RR-24/min, spo2-97% on room air

Patient was having oral candidiasis with severe wasting of muscles of both upper and lower limbs present. Chest findings revealed bilateral vesicular breath sounds with left sided infrascapular crepitation, audible heart sounds without any adventitious component. Abdomen was soft, non-tender with just palpable liver without any shifting dullness.

Detailed CNS examination revealed normal higher functional state, cranium with no signs of external injury. All cranial nerves were normal on examination. Cerebellar signs could not be evaluated. No neck rigidity, Kernig or Brudzinski sign present.

Spine was normal in position without any external deformity, gibbus, kyphoscoliosis, discharging sinus or

step-up deformity. Gait could not be evaluated as the patient was having quadriparesis and lying helplessly on bed.

Patient was having Power of 0/5 in all four limbs and cannot lift his head nor can move his trunk with hypotonia in all four limbs. No involuntary movement present. Coordination could not be evaluated. All superficial and deep tendon reflexes were absent with unresponsive plantar reflex B/L.

Sensory system examination was normal without any specific sensory level found.

Postural drop in BP could not be evaluated.

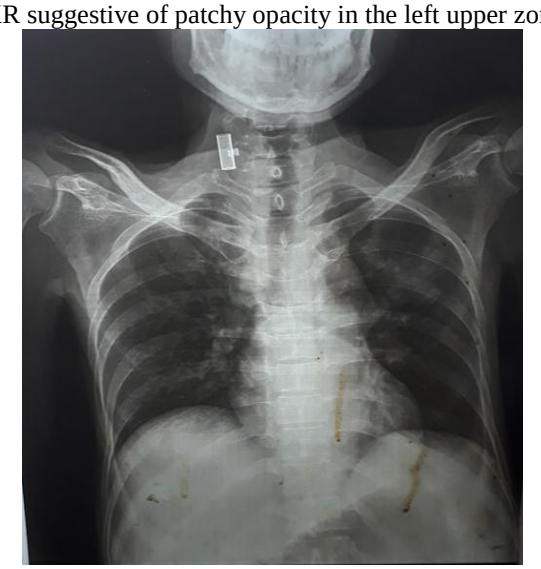
INVESTIGATION REPORTS

DATE	TLC	RBC	Hb/HCT	PLATELET	T.BIL	SGOT/SGPT	ALB/GLOB	UREA/CR	ALP
2/10/20	3400	2.97	9/28	1.54	0.6	58/54	4.8/3.2	24/1.0	273
5/10/20	4100	3.28	10.1/30.9	1.94	0.3	59/46	5.6/3.5	15/0.7	289
14/10/20	5200	3.63	10.9/34.1	2.97	0.3	45/37	4.7/4.0	20/0.7	319

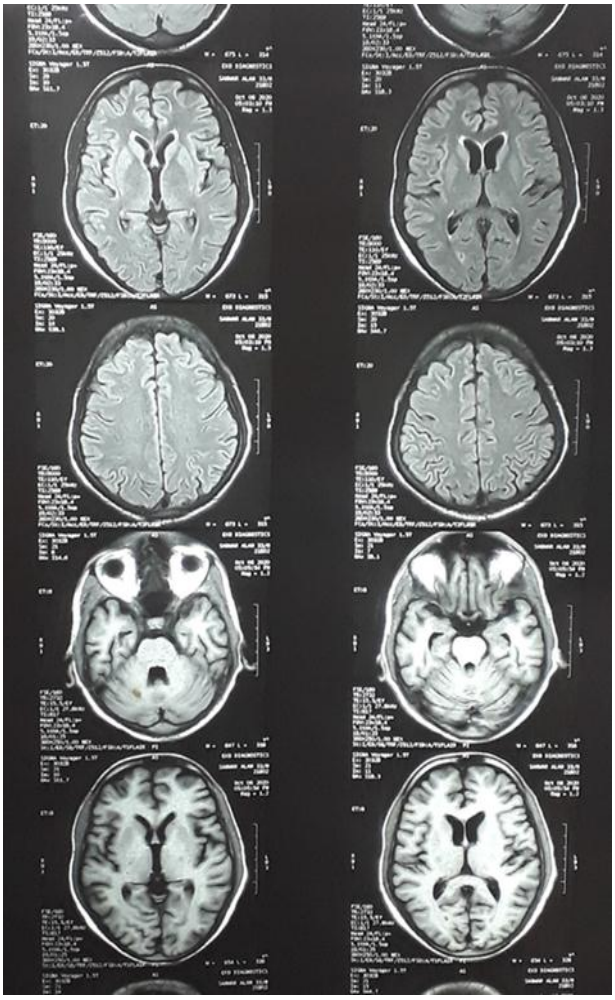
Urine R/E, M/E report
Pus cells-1-2/HPF
Epithelial cells-0-1/HPF
Albumin -2+, sugar, acetone, bile pigments and salts absent.
Urine C/S shows growth of E. coli, colony count>10^5 CFU/ml
Sputum CBNAAT-MTB not found.
No bacterial or fungal growth found.

CSF study-cell count-5 cells, all lymphocytes.no budding yeast cells or cryptococcus seen.
No encapsulated budding yeast in India ink preparation.
CSF bacterial culture-Enterococcus casseliflavus growth, fungal culture-no growth.
CSF glucose-36mg/dl, LDH-287 U/L, protein-1285 mg/dl, ADA-3.41
CSF VDRL-nonreactive
CSF CRAG- nonreactive.
CSF CBNAAT-MTB not detected.
Serum VDRL-nonreactive.
Serum Toxoplasma IgG-positive (>200 IU/ml)
Blood C/S shows no growth.
CD4 count-169 on 7/10/2020.Previous records not available.

RADIOLOGICAL INVESTIGATIONS
CXR suggestive of patchy opacity in the left upper zone.

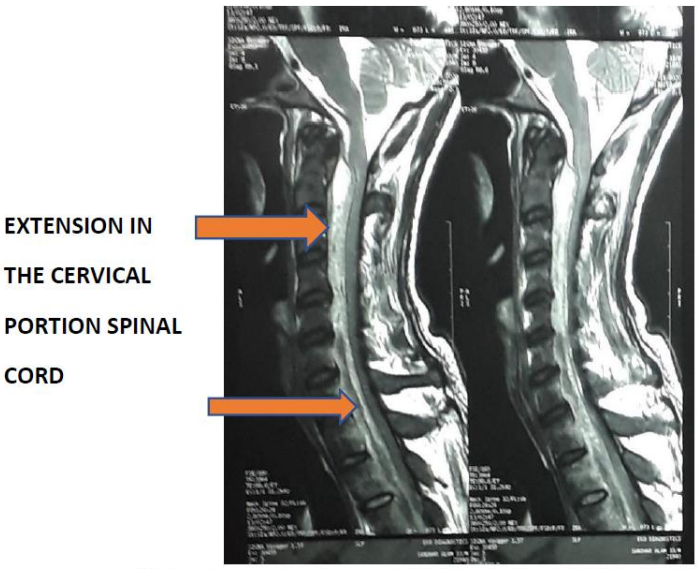


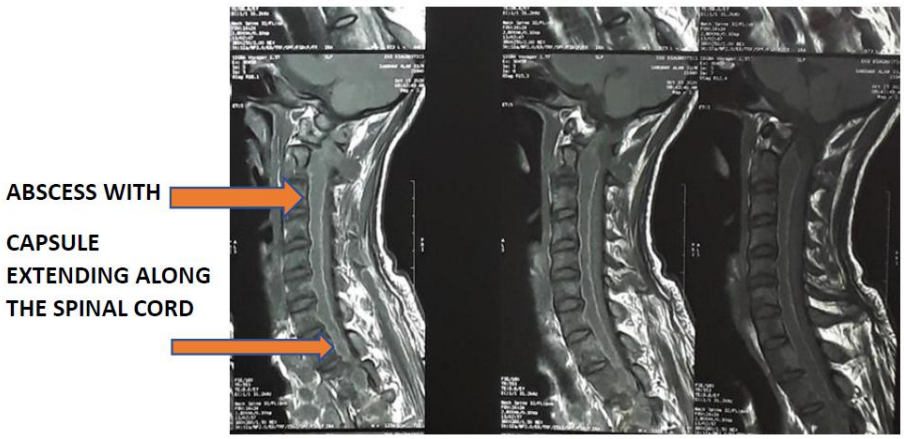
MRI BRAIN suggestive of mild prominent cortical sulci and cerebellar folios with small hyperintensity seen in right side of brain, could be ischaemic.

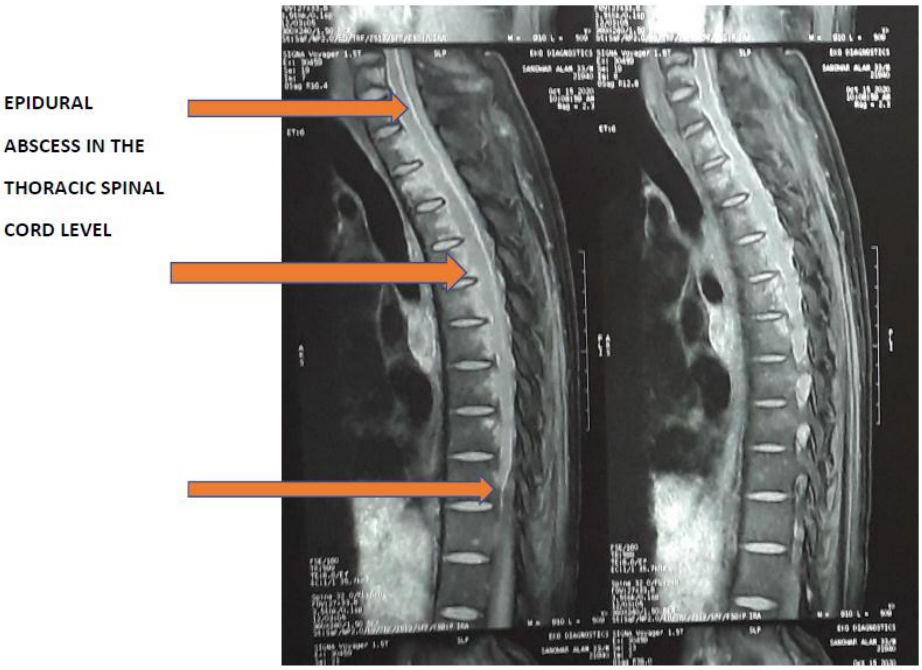


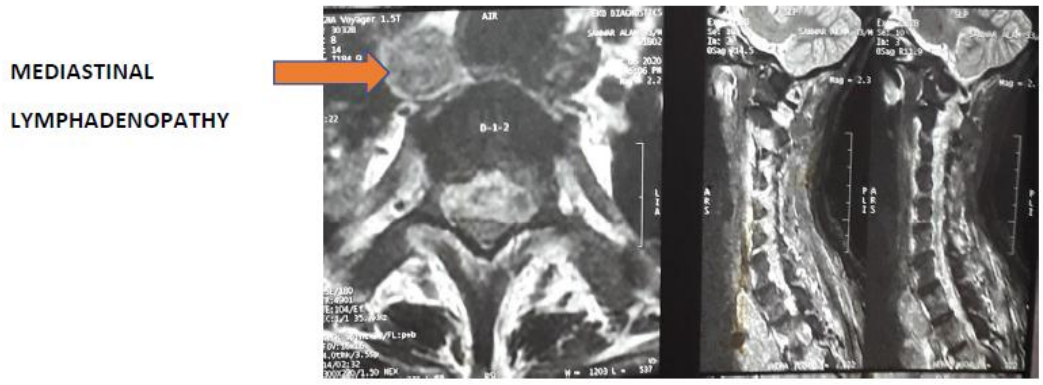
MRI CERVICAL AND DORSAL SPINE (P+C)
T2 weighted FSE and STIR images is showing signal changes from anterior epidural region and extending from C2 level downward with involvement of dorsal region is also seen. Inferior extension is noted up to D10 level. Post contrast study is showing enhancement there. Signal changes are seen from cervical and dorsal vertebrae and extending from C6 level downward up to

D9 level with enhancement. Focal areas of signal change is also seen in left lung upper and mid zone with mediastinal lymph nodes are also seen. Post compressive signal changes are also seen in visualised cervical and dorsal cord. All features are suggestive of inflammatory condition, tubercular with extension, anterior epidural collection, lung involvement and Para mediastinal lymphadenopathy.









Referral Opinion Taken

Ophthalmology: direct fundoscopy suggestive of peripheral scarring with suspected toxoplasma with no features of papillitis or papilledema.

Neurology: with a suspicion of HIV myelopathy, caries spine, GB syndrome with supportive evidence of cervical and dorsal spine MRI (P+C) neurology gave opinion of quadriparesis due to compressive myelopathy due to anterior epidural abscess extending fromC2-D12 associated with arachnoiditis.

Neurosurgery: gave opinion that no drainage of abscess required at present and to continue conservative treatment.

Treatment Summary

Initial liquid diet later shifted to semisolid and lastly solid diet.

IV fluid initially later omitted.

Tab.Cotrimoxazole(toxoplasma dosing)

Tab.Fluconazole

Inj.Pantoprazole and ondansetron

Inj.Polymixin B

Inj.Linezolid.

ATD 4FDC @3PILLS/DAY

ART-TLE

TAPERING dose of Inj..Dexamethasone starting at 0.4mg/kg/day later shifted to Tab.Dexamethasone as per protocol.

Reasons Behind Initiation of ATD

1) History of fever for 2 months with SOB.
2) Third most common cause of epidural abscess.
3) Immunocompromised background.
4) Endemic zone of TB.
5) Involvement of bladder and bowel going against GB syndrome or AIDP.
6) Raised ALP.
7) Mediastinal lymphadenopathy in MRI.

Course During Hospital Stay For 84 Days

Gradually patient had improvement in appetite.

Fever subsided.

Single breath count improved from 5 to 22 per single breath.

Gradual healing of multiple pressure sores and the patient was put on physiotherapy and incentive spirometry.

Upper limb power improved from 0/5 to 3/5 bilaterally.

Lower limb power improved from 0/5 to 1/5 bilaterally.

Incontinence of stool and urine persisted during his 84 days of hospital stay.

DISCUSSION

The clinical presentation of epidural abscesses is related and hence dependent on the anatomy of the spinal canal and dural tube. As normal anatomical variation the epidural space is narrower in the cervical region and wider in the lumbosacral region. The formation, localization and spread of SEA are related to the presence of a real epidural space. Anatomically the dura mater attaches superiorly at the foramen magnum and inferiorly at the sacrococcygeal membrane. The anterior epidural space is mostly encroached by the duramater, posterior longitudinal ligament, and periosteum of the corresponding vertebral body and are tightly adherent; so, because of this anterior obliteration most SEAs occur posteriorly.[2] But in our case, we had extensive involvement of the anterior epidural space which makes this a unique presentation. The epidural space composed of fat, arteries, and venous plexus. SEAs are frequent in the thoracolumbar area, where the epidural space is wider and contains more infective nidus of fat tissue. Low pressure of the venous plexus can frequently cause reflux from the venous plexus within the abdominal and pelvic cavities in the adjoining areas.[3][4]

Infection can be a direct extension of a local nidus, haematogenous spread or a case of iatrogenic inoculation, but some cases show no obvious source of infection, and several are associated with immunodeficiency in the form of HIV.[5][6]

As the pyogenic inflammatory process progresses and the abscess spreads longitudinally within the epidural space, damaged of cord may be due to the following mechanisms: (i) direct pressure and compression, (ii) thrombosis and thrombophlebitis of adjoining veins, (iii) obstruction of arterial blood supply, and (iv) toxin mediated inflammation.

Clinical presentation is classified into four stages: back pain in stage I, nerve root symptoms in stage II, muscle weakness and paraesthesia in stage III, and complete paralysis in stage IV.[7][8]

MRI is a non-invasive, highly sensitive (91%) and specific imaging modality that can well delineate the extent and location of the abscess.[9]

Aim of treatment is to reduce abscess volume and eventually eliminate the abscess and hence the causative organism. Surgery mainly involves laminectomy, degranulation, and pus drainage. Relative contraindications to surgery are as follows: (i) serious medical illness, (ii) lack of spinal cord compression symptoms, and iii) presentation of more than three days from the onset to complete paralysis.

Generally, the postoperative recovery in the symptoms of paralysis is poor in stage IV cases.[10][11]

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