ORIGINAL RESEARCH PAPER
Volume - 9 | Issue - 7 | July - 2020 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

SPINAL EPIDURAL ABSCESS – HYPERACUTE PARAPLEGIA : A Case Report

General Medicine

Dr. Sanjeevkumar N Bentoor Professor, Department of Medicine, Shri B M Patil Medical College, BLDE (Deemed to be University), Vijayapura.

Dr. K Sugunakar Reddy* Resident, Department of Medicine, Shri B M Patil Medical College, BLDE (Deemed to be University), Vijayapura.*Corresponding Author

ABSTRACT

Spinal epidural abscess is rare in occurrence and most often it is missed diagnosis leading to considerable morbidity and mortality which could be easily prevented with timely diagnosis and intervention. Here we present a middle aged male patient presenting with sudden onset of paraplegia which is secondary to spinal epidural abscess.

KEYWORDS

Spinal Epidural Abscess, Paraplegia, Immunocompromised state

INTRODUCTION

Presence of spinal infections can be dated back to 7000 BC, and is found in remains of prehistoric humans. Spinal epidural abscess (SEA) was first described in the medical literature in 1761(Barkas, Chatzidakis, Zogopoulos, & Fratzoglou, 2016). Since its original postmortem description more than 250 years ago by Giovanni Morgagni, SEA has often evaded timely diagnosis, with up to 75% of cases misdiagnosed on their first contact with the health care setup(Bond & Manian, 2016). Spinal and paraspinal infections include "vertebral osteomyelitis, spondylodiscitis, infectious facet arthropathy, epidural infections, meningitis, myelitis and infections of paraspinal soft tissue and musculature"(Article, 2014). SEA is a "very rare disease and occurs at an incidence rate of 0.2 to 2.8 cases per 10000 hospital admissions" (Report, Sales, Tabrizi, Elmi, & Soleimanpour, 2013). Surgery to spine, instrumentation, trauma, immunosuppression, infection, intravenous drug use, vascular access, diabetes mellitus and alcoholism are the possible etiology for spinal epidural abscess. In 20% of cases there will not be any identifiable cause. Back pain and fever are two common complaints with subtle and misleading clinical features which most probably lead to diagnostic delays causing irreversible paralysis and making this case a diagnostic minefield(Report et al., 2013). Only 50% of the cases will present with the "classical clinical triad of back pain, fever and neurological deficit", adding to diagnostic difficulties–(Kumar, Kumar, Dhatt, Lal, & Bahadur, 2017).

SEA though rare, it is a potential life threatening condition and has potential ability to cause permanent neurological disability if it is not diagnosed in time and treated. 50% of the cases of SEAs are either misdiagnosed or a delayed diagnosis is made. Combined with the rarity of condition with non specific and non sensitive clinical features are responsible for missing the diagnosis–(Kumar et al., 2017).

SEA is a suppurative infection of the epidural space, which can cause injury to the spine by direct compression by mass effect or secondary to local ischemia. SEA presents with the "classic triad" of pain, fever, and neurological dysfunction which are nonspecific and could lead to a high rate of misdiagnosis.15% of patients with SEA show a poor prognosis (with paralysis in 8% and 7% mortality rate) (Yang et al., 2019). Gadolinium-enhanced magnetic resonance imaging (MRI) has 90% sensitivity and specificity and is the gold standard to diagnose SEA. As SEA is generally ignored in the preliminary differential diagnosis when patient presents with back pain and the nature of illness demanding early diagnosis and intervention, is the major issue when dealing with a case of SEA. With antibiotics and surgical decompression being mainstay of the treatment, only in timely intervention can only improve the morbidity and mortality(Barkas et al., 2016).

Spinal epidural abscess may jeopardize the spinal cord or cauda equina through compression and by vascular compromise. Without intervention, SEA expands into the spinal epidural space compressing on the spinal cord, leading to sensory symptoms and signs, motor dysfunction, which ultimately leads to paralysis and death, with radicular symptoms patients presents with complaints of chest pain or abdominal pain(Huff & Editor, 2019).

Spinal epidural space is not a uniform space; anteriorly it is a potential space where dura firmly adherent to vertebral bodies and ligaments and posteriorly, the epidural space consist of fat, small arteries, and the venous plexus. SEA frequently present in posterior epidural spaces in thoracic region, and infection may spread over several vertebral levels in both cephalic and caudal directions. Review of several reported cases is suggestive that weakness and neurological deficit is more likely presentation in posterior epidural abscess when compared to anterior(Huff & Editor, 2019).

Though source of infection for epidural abscess could not be identified in majority of cases, hematogenous spread with infection of the epidural space is the suspected source of infection in many. Reported possible sources of infection may include "bacterial endocarditis infected indwelling catheters, urinary tract infection, peritoneal, retroperitoneal infections, and direct extension of infection from vertebral osteomyelities, epidural catheters and injections". Spinal epidural abscess may involve the vascular supply to the spinal cord and may lead to infarction which can cause clinical significant neurological deficit rather than direct compression. Most commonly reported pathogen in SEA is "Staphylococcal aureus, less commonly other organisms like Pseudomonas species, Escherichia coli, Brucella, and Mycobacterium tuberculosis". Unusual bacterial and fungal organisms may be isolated from SEA in immunocompromised patients(Huff & Editor, 2019).

SAE can rapidly and unpredictably deteriorate to stage of irreversible neurologic injury. With diagnostic delays, the presence and duration of neurologic deficits appear to correlate with severity of morbidity and mortality(Artenstein et al., 2015).

CASE REPORT:

A 38 year old male farmer and tractor driver presented to our casualty with sudden onset weakness of both lower limbs 8 days back. Patient 10 days back developed upper backache which is sudden in onset, continuous, dull aching type of pain, not related to posture and no history of radiation of pain and not relieved with rest. There is no history of trauma and lifting heavy weights while at work prior to onset of pain. There is no history of fever, trauma, loss of appetite and weight and no history of cough, joint pains, and stiffness in lower back. On the same day patient consulted his village primary care physician and was given injectable analgesics and patient returned home and slept. When patient woke up after two hours of sleep, patient noticed that he is unable to move his legs on bed and felt lower abdomen discomfort and is unable to pass urine. And patient consulted his primary care physician and then catheterized and was relieved with discomfort and fullness of lower abdomen.

Following 8 days patient was brought to our hospital casualty with Foleys catheter insitu with paraplegia. On examination, patients all higher mental functions are intact, and all cranial nerve examination

are unremarkable. On motor system examination it is found that both lower limbs there is hypotonia and power in muscles acting around hip joint, knee joint and ankle joint is found to 0 (zero) out of 5 grade, and superficial reflexes like corneal, conjunctival are intact and abdominal reflex is absent on both the sides, and plantar reflex on both sides is mute. Deep tendon reflexes, both biceps and triceps are of grade 2, knee and ankle jerk are absent on both sides. On sensory system examination touch, pain, temperature and sense of vibration and joint position are absent up to $T_{6\,level}$ on both sides. All cortical sensations are intact above T_6 level and absent below it. There are no head titubations, no nystagmus, no impaired coordination in both the upper limbs and speech is normal. There were no signs of meningeal irritation and no neurocutaneous markers are found.

Based on the history of hyper acute onset of weakness and examination findings, a provisional diagnosis of transverse myelities and possibility of vascular pathology was considered and patient had undergone MRI dorso-lumbar spine which revealed epidural abscess extending from $D_5 – D_8$ (figure 1,2). Investigations revealed total WBC count of 8880 cells/cmm with neutrophil predominant with 90% and lymphocytes of 6.8%, with serum ADA of 33 U/L, C reactive protein of 90mg/L, with liver function test being within normal limits and urine examinations revealing plenty of pus cells, and patient tested to be positive for HIV 1 Antibodies and coagulation profile within normal limits.

Patient was posted for dorsal laminectomy with evacuation of collection on the next day and unfortunately patient could not be survived and succumbed to sudden cardiac arrest with acute pulmonary embolism.

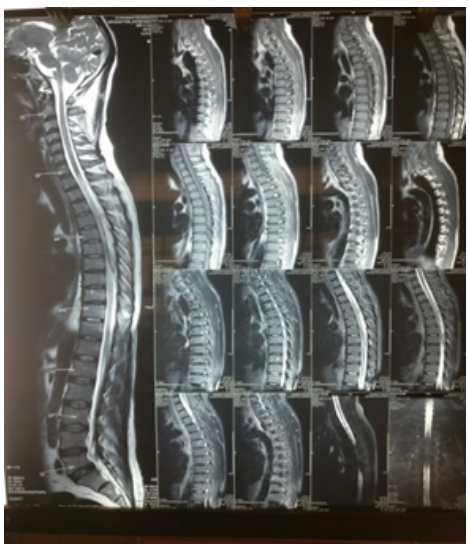

Figure 1: MRI Spine sagittal section - pyriform shaped SEA extending from D4 to D8 vertebrae.

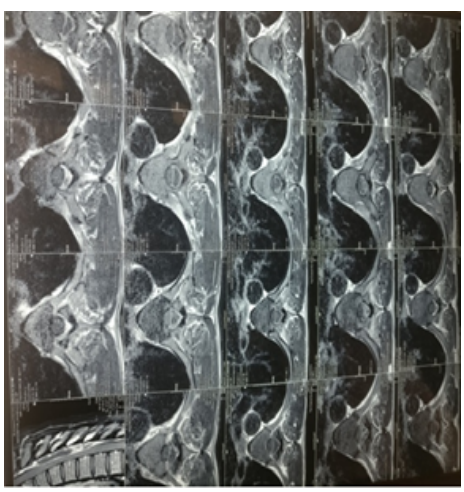

Figure 2: MRI Spine- Coronal section, showing SEA compressing spinal cord.

DISCUSSION

SEA though a rare condition where without timely intervention, it may lead to several complications leading to paralysis and even death with rapid and unpredictable course of progression. “Clinical evolution of SEA encompasses 4 stages: pain, radiculopathy, weakness and paralysis”.

Though there is no consensus on the preferred modality of treatment in cases of SEA and it is reported that emergency surgical intervention with antibiotics results in better outcomes. There is still a paucity of the data regarding size of epidural abscess in relation with neurological deficit and size of epidural abscess for which antibiotics without surgical intervention could be tried and size of abscess which requires surgical intervention.

In our case in present discussion the question was how does a epidural abscess (a infective mass) resulted in the hyper acute presentation of paraplegia with no evidence of infarction in spine and no history of prior ongoing backache? On studying MRI of whole spine it is found that there is extensive edema extending above and below the epidural abscess, because of edema that developed within hours, patient had developed sudden onset paraplegia.

The pathological basis of cord damage is an admixture of direct compression by mass effect & secondary infraction due to thrombophlebitis of the local venous channels and spinal cord edema that is found surrounding the compression.

Though the spinal epidural abscess is a rare cause of paraplegia, “clinically accurate diagnosis is almost impossible and the most frequent suspected clinical diagnosis is transverse myelitis. Only in those patients where there is a clear infection risk and in medical conditions such as diabetes, subacute bacterial endocarditis & septicemia and in patients with immunocompromised state this condition would immediately enter into differential diagnosis”. An MRI scan is the only way to make a definitive diagnosis and investigation modality of choice which can both localize the lesion and show the extent of lesion(Patten, 1996).

CONCLUSION:

Though the cases of spinal epidural abscess are rare in the literature, now the incidence is in rise because of increasing incidence of acquired immunological deficiency in population. In the present case in discussion, patient presented with sudden onset of backache followed by weakness of both the legs secondary to the edema developed in the spinal cord which was secondary to space occupying mass effect of spinal epidural abscess. From experience it is prudent to include the space occupying lesions like spinal epidural abscess in the differential diagnosis while dealing with the patients of sudden onset paraplegia which could be a reversible cause of paraplegia with timely intervention.

REFERENCES

1. Artenstein, A. W., Friderici, J., Holers, A., Lewis, D., Fitzgerald, J., & Visintainer, P. (2015). Spinal Epidural Abscess in Adults : A 10-Year Clinical Experience at a Tertiary Care Academic Medical Center. Open Forum Infectious Diseases, 3(4), 1–8. https://doi.org/10.1093/ofid/ofw191
2. Article, R. (2014). Pyogenic and non-pyogenic spinal infections : emphasis on diffusion-weighted imaging for the detection of abscesses and pus collections. British Journal of Radiology, 87(June). https://doi.org/10.1259/bjr.20140011
3. Barkas, K., Chatzidakis, E., Zogopoulos, P., & Fratzoglou, M. (2016). iMedPub Journals Spinal Epidural Abscess Causing Cauda Equina Syndrome : A Case Report. IMedPub Journal, 2(1:13), 1–3.
4. Bond, A., & Manian, F. A. (2016). Spinal Epidural Abscess : A Review with Special Emphasis on Earlier Diagnosis. BioMed Research International, 2016(october), 0–5. https://doi.org/10.1155/2016/1614328
5. Huff, S., & Editor, C. (2019). Spinal Epidural Abscess (pp. 1–13). pp. 1–13. Retrieved from https://emedicine.medscape.com/article/1165840-print
6. Kumar, A., Kumar, V., Dhatt, S. S., Lal, H., & Bahadur, R. (2017). Journal of Clinical Orthopaedics and Trauma Spontaneous spinal epidural abscess in a normoglycemic diabetic patient – Keep it as a differential. Journal of Clinical Orthopaedics and Trauma, 8(2), 178–180. https://doi.org/10.1016/j.jcot.2016.09.007
7. Patten, J. P. (1996). Neurological Differential Diagnosis 7.1 (2nd editio). midhurst, sussex GU29 0BL, UK: Springer-verlag Berlin Heidelberg New York.
8. Report, C., Sales, J. G., Tabrizi, A., Elmi, A., & Soleimanpour, J. (2013). Adolescence spinal epidural abscess with neurological symptoms : case report , a lesson to be re-learnt. Medical Journal of Islamic Republic Of Iran, 27(1), 38–41.
9. Yang, X., Guo, R., Lv, X., Lai, Q., Xie, B., Jiang, X., … Zhang, B. (2019). Challenges in diagnosis of spinal epidural abscess : A case report. Medicine, 98(5), 3–5. https://doi.org/10.1097/MD.0000000000014196