IDCases 13 (2018) e00438

Contents lists available at ScienceDirect

IDCases

journal homepage: www.elsevier.com/locate/idcr

Case report

Spinal epidural abscess caused by a community acquired extended spectrum beta lactamase producing *Klebsiella pneumonia*

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ARTICLE INFO

Article history:
Received 8 June 2018
Received in revised form 7 August 2018
Accepted 7 August 2018

Keywords:
Abscess
Antimicrobial
Epidural
ESBL
Klebsiella
Stewardship

ABSTRACT

Spinal epidural abscess (SEA) can be a medical and surgical emergency. It is encountered in patients with epidural catheter placement, paraspinal injections, diabetes mellitus, alcoholism, HIV infection, trauma, contiguous bony or soft tissue infection, intravenous drug use, hemodialysis, or overt bacteremia, but may occur spontaneously associated with a presumed silent bacteremia. We report here, a case of extensive SEA due to a community-acquired extended spectrum beta lactamase (ESBL)-producing *Klebsiella pneumoniae* in a diabetic patient. This case highlights the importance of stringent antimicrobial stewardship and also the need for prompt diagnostic evaluation, and early surgical decompression in order to prevent morbidity and mortality.

Introduction

The incidence of spinal epidural abscess (SEA) has increased over the last several decades owing to the improved sensitivity and specificity of magnetic resonance imaging (MRI) and also partly due to the growing number of spinal intervention procedures performed in the elderly [1–3]. *Staphylococcus aureus* accounts for about two-thirds of cases of SEA and in rare instances it can also be caused by gram negative bacteria [4]. Most of the gram negative bacterial spinal infections are related to an immunocompromised state, recent spinal intervention, intravenous drug use or poorly controlled diabetes. There have been very few cases of extended spectrum beta lactamase (ESBL) producing *Klebsiella pneumoniae* spinal infections and almost all of these were hospital acquired [5–7]. Recent case reports in literature of community acquired ESBL producing strains has caused concerns for emergence of multidrug resistant bacteria [8]. Here, we present an unusual case of full axial spinal epidural abscess caused by community-acquired ESBL-producing *Klebsiella pneumoniae* in a diabetic patient along with a brief review of pertinent literature.

Case report

A 55-year-old woman presented with fever, dysuria and malaise for a week. She was also noted to have urinary incontinence and difficulty ambulating due to worsening back pain. She denied to any history of recent illness or hospitalization, back trauma, paraspinal injections, skin infection, weight loss, or night sweats. Her past medical history included poorly-controlled diabetes mellitus type 2, hypertension, hyperlipidemia, and chronic low back pain. She also denied to any history of tobacco, alcohol, or illicit drug abuse. On physical examination, she appeared acutely ill and diaphoretic. Her temperature was 38.3 °C, blood pressure 186/82 mmHg, pulse 114 beats per minute, respiratory rate 20 breaths per minute, and oxygen saturation 98% on room air. Chest and abdomen examination showed no abnormalities. She appeared mildly confused and lethargic but rest of the neurological exam was normal. Electrocardiography tracing showed poor R-wave progression and left ventricular hypertrophy. Chest X-ray did not show any acute process. Routine laboratory analysis showed white blood cell count was 15,600 per cubic millimeter, with 87% neutrophils. Comprehensive metabolic panel and coagulation studies were normal. Urine analysis had 3+ bacteria. Blood and urine cultures were obtained and she was admitted for sepsis secondary to a urinary tract infection. Initial antibiotic of choice was ceftriaxone, which was switched to piperacillin –tazobactam a day later when patient started to have persistent fever and worsening mentation.

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https://doi.org/10.1016/j.idcr.2018.e00438

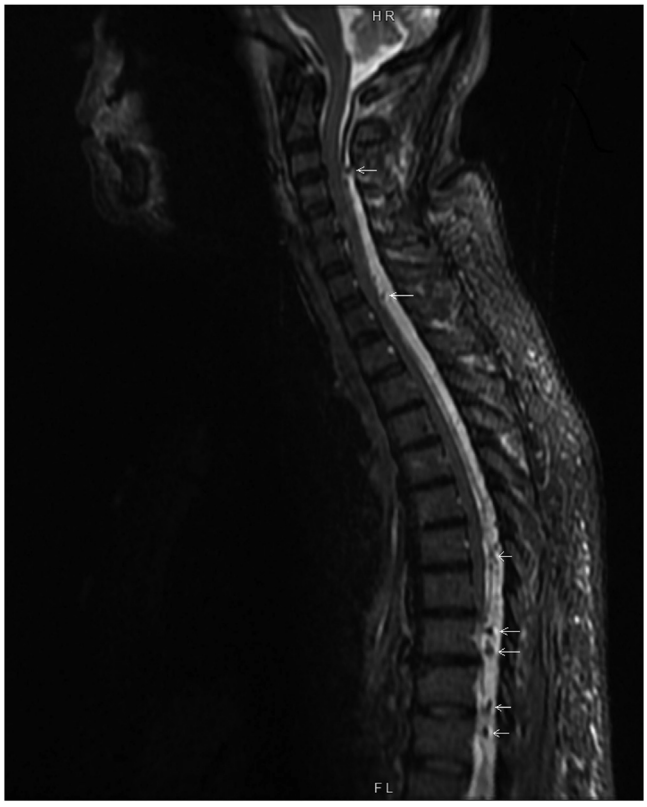

Fig. 1. Sagittal view of gadolinium-enhanced magnetic resonance imaging of the cervical and thoracic spine showing multiple peripherally enhanced fluid collections (arrows) extending from C3 to T12 spine in the dorsal epidural space.

She was transferred to the medical intensive care unit on account of encephalopathy. Repeat neurological examination showed generalized weakness with diminished strength, tone, and sensation in all extremities. Patella tendon reflexes were absent bilaterally but rectal tone was normal. There was nuchal rigidity with moderate tenderness along the entire spine. Blood and urine culture from the day of admission showed gram negative bacilli. Antimicrobials were widened to vancomycin and meropenem to provide central nervous system coverage. An emergent MRI of the spine with contrast was obtained which showed extensive posterior epidural fluid collection with internal air locules extending from C1 to L4 spine with mass effect on the cord and severe thecal sac stenosis in the low cervical and mid thoracic levels (Fig. 1). The patient was immediately taken to the operating room where she underwent cervical, thoracic, and lumbar decompression with evacuation and aggressive irrigation of the epidural abscess. Blood, urine and abscess cultures isolated ESBL-producing *Klebsiella pneumoniae* which was sensitive to fluoroquinolones, gentamicin, and meropenem (Table 1). Fungal and acid fast cultures were negative. Recovery was painstakingly slow despite intensive physical and occupational therapy. Patient was subsequently discharged to a skilled nursing facility for continuation of physical therapy rehabilitation. She remained on meropenem for a total of 12 weeks following which a repeat MRI of the entire spine showed complete resolution of epidural abscess.

Discussion

Spinal epidural abscess is a rare life threatening condition that is misdiagnosed on initial presentation as it often manifests with non-specific symptoms such as low back pain [1]. Prognosis is dependent on early diagnosis and prompt therapeutic intervention before any neurological complications occur [2].

Table 1
Culture and sensitivity of the abscess drained during surgery.

Antibiotics	MIC interpretation	MIC dilution (μg/mL)
Amoxicillin/Clavulanate	Resistant	<8/4
Ampicillin	Resistant	>16
Cefazolin	Resistant	>16
Cefotaxine	ESBL	>32
Ceftriaxone	ESBL	>32
Cefuroxime	Resistant	>16
Ciprofloxacin	Sensitive	<1
Gentamicin	Sensitive	<4
Levofloxacin	Sensitive	<2
Meropenem	Sensitive	<1
Sulfamethoxazole/Trimethoprim	Resistant	>2/38
Ticarcillin/Clavulanate	Resistant	<6

The risk factors for developing SEA include diabetes, intravenous drug use, chronic liver or kidney disease, recent spine procedures/fractures, indwelling vascular catheters, other sites of infection, or an immunocompromised condition [2]. Hematogenous spread from distant sites including urinary and respiratory tract or skin and soft tissue infection accounts for nearly half the cases of SEA [3]. Other ways by which organism gain entry into the epidural space include direct inoculation during a spinal procedure or contiguous spread from a nearby foci like psoas abscess or vertebral osteomyelitis. The leading bacterial pathogen causing SEA is *S. aureus* accounting for about 66% of cases while gram negative bacilli makes up 16% of infections [3]. Among the gram negative bacilli, *Klebsiella pneumoniae* accounts for approximately 1% of SEA [4]. The most common location for a SEA is the lumbar spine in about 48% followed by thoracic 31% and lastly cervical approximately 24%; although, it can involve several segments of the spine simultaneously [2].

Classic presentation with fever, spinal pain and neurological deficits is seen in less than 50% of cases [3]. Nonspecific complaints of low back pain is the predominant symptoms seen in about 75–80 % of patients, though some patients describe severe circumscribed tenderness. Heusner summarized the progression of SEA in four stages, stage1 wherein the patient has only back pain with occasional fever and local tenderness, in stage 2 disease, nuchal rigidity, radicular pain and loss of reflexes can be noted. Stage 3 is characterized by sensorineural and motor weakness including bladder dysfunction and lastly stage 4 leads to paralysis. The duration of symptom and rate of neurological deterioration is variable and this unpredictable course of the disease is often the reason for delayed diagnosis [2].

Inflammatory markers such as C reactive protein and sedimentation rate is often elevated and should be considered in anyone presenting with low back pain to differentiate potentially infectious etiology from degenerative processes [2,4]. MRI with gadolinium enhancement is considered gold standard for diagnosis of SEA and in patients in whom MRI with contrast is contraindicated, myelography followed by computerized tomography (CT) scan is a preferred alternate [2–4]. Lumbar puncture has limited value in diagnosis of SEA, however CT guided aspiration of the abscess can be performed early to identify the microorganism accurately [2]. Empirical antimicrobials covering common pathogens should be commenced immediately and antimicrobial coverage should be adjusted once final cultures and sensitivities are obtained [1,3]. Surgical decompression remains the treatment of choice in patients with neurological symptoms. Post-operatively parenteral antibacterial therapy is continued for 6–12 weeks; however the optimal duration for complete eradication of the infection is unclear [1–3]. Our patient presented initially with

stage 1 SEA which rapidly progressed to stage 2 involving cervical, thoracic and lumbar vertebral segments. In addition, she grew ESBL producing *K. pneumoniae* in the abscess cultures making her case both interesting and challenging.

Extended spectrum beta lactamase (ESBL) producing pathogens have long been recognized as a common cause of nosocomial infections and hospital outbreaks, although recent data suggests increased incidences of ESBL-producing strains in urinary tract infections in the community settings [8]. Several studies from China and Japan have shown that ESBL producing *E.coli* and *K. pneumoniae* infections are on a rise creating concerns for emerging antibacterial resistance [9]. Very little is known with regards to the epidemiology of these community-acquired infections and no specific risk factors have been clearly identified. Studies have indicated that rampant use of antimicrobials may be a source for fecal transmission of these multidrug resistant bacteria in the community [9,10]. Genetic bacterial studies has revealed that a majority of these community acquired ESBL infection harbor the CTX-M allele, particularly the M-15 type which is responsible for widespread dissemination and transfer of drug resistance among different coliform bacteria in the reservoir pool [11,12]. Individuals who have been exposed to cephalosporins recently and nursing home inhabitants seem to be at an increased risk of contracting the ESBL producing strains of the gram negative bacilli [8,11].

These new multidrug resistant organisms present a unique challenge as they may limit the initial approach to effective and proper antimicrobial therapy. Most ESBL-producing gram negative bacteria are resistant to β-lactam drugs and carbapenems remain drugs of choice [6]. Recently, carbapenemase resistance also seems to be emerging, increasing the need for a stringent antimicrobial practice [8,13]. ESBL-production is associated with higher mortality in *K. pneumoniae* infections. It is also associated with a high rate of treatment failure even if the organisms test susceptible to antibacterial in vitro [8–10]. Hence, we should be extra vigilant in treating these infections.

It is noteworthy to mention that our patient had an ESBL producing strain *of Klebsiella pneumoniae* that caused her SEA. She has no discernable healthcare-associated risk factors such as recent hospitalization or prior antibacterial use. Hence, her infection can be considered as truly community-acquired. The portal of entry was most likely due to a hematogenous spread from pyelonephritis as both blood and urine isolated the same organism that caused the SEA. Poorly controlled diabetes and degenerative disease of the spinal column may have been other predisposing factors contributing to epidural infection. The diagnosis in this case was challenging and relied on a high index of suspicion. Lumbar puncture and cerebrospinal fluid analysis would have been the appropriate test of choice in this patient with encephalopathy and nuchal rigidity, however, in view of loss of deep tendon reflexes, an MRI of the spine was obtained confirming the diagnosis of SEA. Our suspicion for multi-drug resistant organisms was low and empiric therapy with a carbapenem and vancomycin was selected based on the patient's rapidly deteriorating neurological status and excellent central nervous system penetration of the drug. The finding of gram negative bacteria as a cause of SEA was not surprising, however the ESBL production was unexpected. The fact that this was less likely to be a nosocomial infection but more a community-acquired ESBL-producing organism was also quite alarming.

In conclusion, spinal epidural abscess is a rare but serious infection. A high index of suspicion is needed to make a timely diagnosis. Management includes immediate surgical drainage and antimicrobial therapy to avoid catastrophic permanent neurologic sequelae. Antimicrobial stewardship program in both hospital as well as outpatient setting would help to reduce the emergence of multidrug resistant bacteria. Primary care physicians need to be aware of the increasing incidence of community acquired ESBL producing infections and should adopt a diligent practice of appropriate antibiotic use to curb the spread of these superbugs.

Funding

This paper did not receive any specific grant from funding agencies in public, commercial or not for profit sectors.

Patient consent

Written informed consent was obtained from the patient's legal authorized representative for publishing this case report

Declarations of interest

None.

Author Statement

Dr. Vinh Dang, M.D; Conceptualization, original draft of manuscript, image acquisition; Aarthi Rajkumar, M.D; F.A.C.P; Review of draft manuscript, editing, final approval of manuscript & corresponding author.

Acknowledgement

The authors wish to acknowledge Ms. Heather Lyons, Administrative assistant, Canton Medical Education Foundation, for her help with the images used in manuscript.

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