

Pyogenic cervical spondylodiskitis: case report and current literature review

Abstract

Background: Cervical pyogenic spondylodiskitis accounts for less than 11% of all infectious spondylodiskitis. Its main origin is hematogenous, its most common etiological agent is *Staphylococcus aureus*, and however, different microorganisms can be found to cause this infection. Its insidious course, aggressiveness, mortality and high risk of sequela make timely diagnosis and early treatment a fundamental part of the patient's prognosis.

Case presentation: 47 year old woman, with type II diabetes mellitus and arterial hypertension with quadriplegia of three months evolution. A diagnosis of pyogenic spondylodiskitis C4-C5 ASIA C due to *Staphylococcus aureus* was made, cervical debridement, decompression and fusion were performed and intravenous vancomycin and meropenem were administered for nine days.

Results: She had a satisfactory outcome in the postoperative period, leaving the hospital with oral levofloxacin for three months, presenting recovery of general neurological status and gait recovery (ASIA E).

Conclusion: There are several reports on elevated probability of neurological disease and its sequela, as well as on its high mortality rate (21%); however, there are few studies with an adequate level of evidence regarding the diagnosis and treatment of cervical ED.

The current guidelines suggest the early empiric broad spectrum antibiotic administration within the first contact in aggressive cases, and posterior surgical treatment in addition to specific antibiotic therapy.

Keywords: Cervical pyogenic spondylodiskitis, Cervical infection, Neurological condition, Conservative treatment, Surgical treatment, Sequelae, Mortality

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Introduction

Pyogenic spondylodiskitis (PS) or vertebral osteomyelitis is a bacterial infection of the spine that accounts for 2-7% of all hematogenous osteomyelitis. It most commonly affects the intervertebral disc, adjacent vertebral bodies and perivertebral tissues and may extend to the posterior elements of the spine. Cervical spondylodiskitis (SD), although less frequent compared to that which affects other spine segments, has a more aggressive clinical course and more severe complications.

Case presentation

We present the case of a 47-year-old female patient, housewife, with history of hypertension, diabetes mellitus type 2 and sulfa allergic, who is assessed for intermittent cervicalgia of 3 months evolution, thoracic extremities irradiated, exacerbated with daily life activities; After a brief period of analgesics and non-steroidal anti-inflammatory drugs, cervical pain became constant, pungent with an intensity of 8/10 at rest, that extends to thoracic and pelvic extremities, with concomitant paresthesias, loss in strength and gait inability. No fever or weight loss was documented.

Patient Clinically conscious, 15 points Glasgow scale, lying in bed. Diffuse pain upon palpation of cervical region, limitation of mobility, positive Spurling test, negative Jackson test, bilateral hypoesthesia in dermatomes T2-T12. Hypotrophic thoracic limbs on sight, limited active range of motion with passive preserved, negative Hoffman test, positive Lhermitte test, C6-C7 decreased osteomuscular reflexes (OMR), C5-T1 myotomes with strength 2/5 on Daniels scale, bilateral C4-T1 dermatomes hypoesthesias. Hypotrophic pelvic limbs

on sight, negative Babinski test, L2-S1 myotome decrease in OMR (+ / +++) with strength decrease 2/5 on Daniels scale, L1-S1 bilateral hypoesthesia and uresis through Foley catheter.

Cervical spine X-rays with cervical lordosis rectification, C4-C5 intersomatic space diminished, inferior C4 and superior C5 platform osteolysis. Cervical spine CAT scan with presence of C4-C5 intervertebral disc bulging and vertebral cervical canal invasion. Cervical spine MRI with C2-C5 anterior soft tissue edema, C4-C5 intervertebral space diminished, superior C5 and inferior C4 platform destruction, intervertebral disc absence replaced by heterogeneously composed image with surrounding hyperintensity and central hypointensity, protrusion and invasion towards the medullary canal of 50-70%, posterior longitudinal ligament integrity and C3-C6 myelomalacia.

Initial laboratory results with 10.4 K / dL leukocytosis, 63% neutrophilia, 44 mm / hg erythrocyte sedimentation rate (ESR), 39.8 mg / dL C-reactive protein (CRP). Urine test positive nitrites, 3-5 erythrocytes, and 25-28 leukocytes per field with positive bacteria and results negative uroculture.

5 point JOA score (A1, B1, C1, D2), ASIA C stratification. C4-C5 pyogenic spondylodiskitis diagnosis is made. Hospitalization is required and intravenous antimicrobial treatment on an amikacin and cefotaxime based scheme is initiated. 8 days after antibiotic treatment is initialized, a C4-C5 anterior abscess drainage, C4-C5 corpectomy and C3-C6 mesh and plate fusion are performed. Biopsy culture reports *Staphylococcus aureus* vancomycin and meropenem sensitive, antibiotic scheme changes are performed based on these results. Patient is discharged 8 days after surgery and 2 weeks after

intravenous antibiotic therapy with an oral levofloxacin-based regimen, which was suspended after 12 weeks administration.

Follow-up is given through outpatient consultation, and after 5 months after surgery patient is found with independent gait, C5-C8 and T1-S1 4/5 muscle strength, adequate bladder and anal sphincter control, Hoffman, Negative Lhermitte, Openheimer and Babinski tests, ASIA E scale stratification and a 15 point JOA score (A4, B5, C3, D3).

Discussion

Pyogenic spondylodiskitis (SD) or vertebral osteomyelitis is a bacterial infection of the spine that affects the intervertebral disc and may spread to adjacent vertebral bodies and even to surrounding soft tissues.^{1,2}

Epidemiology

SD represents around 1-7% of all hematogenous osteomyelitis, with an estimated incidence of 1: 250,000 to 1: 400,000 inhabitants. It is believed that its incidence and complexity have increased due to demographic factors (higher survival rate, prevalence of chronic-degenerative diseases, etc.), a rise in the number of invasive procedures that cause secondary bacteremia and the appearance of multidrug-resistant organisms.³ Chronic degenerative diseases (diabetes mellitus type 2, hypertension, etc.), rheumatoid arthritis, liver cirrhosis, intravenous drug use, malnutrition, infection by the human immunodeficiency virus (HIV), kidney disease. Chronic cancer and use of steroids for long periods of time are considered risk factors and have associated them with up to 50% of all cases. Commonly it affects individuals over 50 with a male gender predilection.⁴ Its most frequently located in the lumbar region (48-60%), followed by the thoracic region (26-35%) and finally the cervical region (6.5-13%).^{1,4}

Etiology

According to their origin they are divided into hematogenous, by contiguity and direct inoculation (traumatic, iatrogenic). Currently, an increase in minimally invasive procedures, cervical and pharyngeal sharp injuries secondary pyogenic SD has been reported, and also a case secondary to prosthetic voice device placement has been described.⁵ Three types of SD are recognized according to their etiological agent: pyogenic, parasitic and granulomatous including tuberculous, aspergillar and fungal (representing the latter less than 24%).⁵ The majority of pyogenic SD (>90%) are those of a monomicrobial flora and most frequently caused by Gram-positive microorganisms (69.3%), being *Staphylococcus aureus* the most commonly isolated etiologic agent (10-12% methicillin-resistant), followed by *Streptococcus* and coagulase negative *Staphylococcus*.³⁻⁶ Also but minor in frequency Gram negative bacilli (21.5%), anaerobic bacteria (*Propionibacterium*) and fungi have been isolated (especially in immunosuppressed individuals).⁴

Clinical presentation

SD has an insidious course, with variable degree of neurological affection, occasional rapid progression and is highly aggressive.^{1,5,7} Diagnosis is usually late, more often in early chronic stage (more than 3 months) and is diagnosed in the acute stage (less than 3 weeks) less than 30% of the time. More commonly it affects a single level (63.2%), C5-C6 mainly, followed by C6-C7 and C4-C5.^{1,8,9} It can also be multisegmentary (<20%) and less frequently present itself in tandem lesions.¹⁰ The clinical diagnosis requires high clinical suspicion, complete anamnesis and a thorough neurological examination. The first most common clinical manifestations are pain (lumbar and dorsal

in 91% of cases) and fever (68%); however in cases of cervical disease symptomatology is less clear, with presence of pain in less than 10% of all occasions.⁸ Neurological affection may be absent in up to 69% of all patients, although some authors have described it in 35% to 60% of cases at the time of diagnosis.^{1,8}

Imaging

Simple radiology: Diagnosis by means of conventional radiology (simple radiographs) in the acute stage is difficult, because at this time no significant radiographic findings are commonly observed. An early appearance sign is an intervertebral disk space increase; however, a 30%-40% bone loss is usually required for radiological changes to be detectable, this often happens after 2 weeks onset.¹¹

In the sub acute phase (3 weeks-3 months), progressive radiological osteolysis, loss of bone definition, vertebral platforms irregularity and osteopenia in adjacent vertebral platforms (secondary to the inflammatory process) begin to appear.^{10,11} The presence of a paraspinal mass, observed as a retropharyngeal space increase (cervical SD), parietal pleura rejection (thoracic SD) and psoas effacement (lumbar SD) may be visible.¹⁰ In the chronic phase (more than 3 months) extensive bone destruction can be observed with alterations of sagittal and / or coronal alignment (scoliosis, kyphosis), reactive sclerosis, ankylosis and spondylolisthesis.¹⁰⁻¹²

Computed axial tomography: Computed tomography scan (CAT) has a high sensitivity for SD, but low specificity, which is why it isn't considered a first line study for the diagnosis and it does not represent an advantage over conventional radiography in the setting of acute stage SD diagnosis.^{10,11} In the sub acute stage, a greater number of radiological changes can be observed, such as an intervertebral space height decrease, osteopenia, vertebral platforms fragmentation and sub-periosteal defects. Gas detection within the lesion or in surrounding tissues is likely, as well as edema and obliteration of the fatty tissue, however these findings can also be found in noninfectious and degenerative conditions.¹⁰ In the chronic phase, a CAT scan shows the same changes described for simple radiographs, but with a higher image resolution.

The use of CAT scan, however, is a very powerful tool as biopsy and as percutaneous abscesses drainage auxiliary.

Magnetic resonance imaging: Magnetic resonance imaging (MRI) has a sensitivity and specificity of up to 96% and 94% accordingly, with a diagnostic certainty of 94%.¹¹

The earliest detectable radiological findings by this method are hypointensity of cancellous bone, vertebral platforms and pre-vertebral structures on T1 weighted images and hyperintensity on T2 and STIR sequence, with reinforcement of the vertebral region upon contrast administration.^{10,11} It is common that in the early course of the disease these changes can be easily confused with MODIC changes, neoplasias or Schmorl's nodule, which is why they are considered nonspecific findings. In subacute phases however, an epidural abscess that exerts a mass effect towards the epidural region can be detected, with hyperintense images at the center of the lesion on T2 weighted sequences and hypo to isointense T1 weighted images. T1 hypointense and T2 hyperintense images with homogeneous lesion reinforcement can be seen upon contrast administration.¹²

Chronic changes observed in MRI images are decreased vertebral intensity on T1 and T2 images, sclerosis, minimal reinforcement upon contrast administration and deformities (kyphosis, scoliosis, ankylosis, spondylolisthesis).^{11,12} MRI can be useful in outpatient follow-up, however its use is not recommended in at least a period of up 2 months after beginning treatment.¹⁰

Laboratory findings

The most significant findings are the presence of leukocytosis usually under 12,000 U / dL (sensitivity 27.2%).^{13,14} Considered of greater diagnostic value although paradoxically with less diagnostic specificity, are ESR and CRP. Elevation of ESR over 40 mm / hr is present SD diagnosed patients in up to 73-100% of the time; Pereira Da Silva Herrero et al.¹⁴ reported 100% sensitivity for ESR and 90.4% for PCR while Orso et al. reported 100% sensitivity for ESR and CRP.^{14,15} Serial blood cultures (2 blood cultures, in 2 different times) with antibiogram is a practical tool in the setting of pyogenic SD pharmacological treatment.^{15,16} Positive cultures are reported with isolation of the causative agent only in 50% of cases.¹³ Uroanalysis with uroculture is mandatory in the case of hematogenous SD suspicion, since the urinary tract it is the most common originating site.¹⁶ Fine-needle aspiration CAT scan guided biopsy is considered at the time to be the goal standard for isolation of the causative agent upon clinical suspicion of pyogenic SD and negative radiology and blood cultures studies, being positive in up to 63% of all cases.^{13,16}

Treatment

Antibiotic therapy

Intravenous antibiotic therapy is the cornerstone in the treatment of pyogenic SD. Depending on the patients clinical situation, two main antibiotic regimens can be established¹⁶⁻¹⁸

- A. Hemodynamically stable patient: no data of sepsis or septic shock, absent neurological alterations and pyogenic SD suspicion or diagnosis.
- B. Hemodynamically unstable patients: sepsis or septic shock signs and severe or progressive neurological alterations with pyogenic SD suspicion or diagnosis.

In the first group, withholding empirical antibiotic therapy is recommended until a microbiological diagnosis is obtained. On the contrary, in group 2 patients, immediate broad-spectrum antimicrobial drugs administration is recommended. Broad-spectrum recommended drugs are vancomycin and ceftriaxone (methicillin-resistant *Staphylococcus aureus*, *Streptococcus* and *Enterobacteriaceae* coverage), as an alternative in cases of pseudomonal infection suspicion ceftriaxone can be replaced by ceftazidime.^{16,18} In penicillin allergy cases a cephalosporin change can be made for meropenem or aztreonam; Vancomycin allergic patients can be switched to linezolid.¹⁶ Generally, a minimum duration of 4-8 weeks with intravenous antibiotic therapy is recommended based on a variable number of studies. This scheme is associated with a lower recurrence rate.¹⁶⁻¹⁸ However, there are other authors who advise antibiotic change to an oral administration if the blood culture reports good sensitivity for said drug after 2 weeks intravenous therapy.^{5,18}

Surgical treatment

Surgery indications vary accordingly to different publications; however, there are well-established situations in which immediate surgery is necessary. Immediate surgical drainage and debridement with or without stabilization is indicated in patients with neurological deficit and epidural abscess, in those with neurological deficit, progressive deformity and vertebral instability despite antimicrobial treatment,¹⁷ as well as in those patients with radiological and asymptomatic spinal abscess. Surgery may be open or percutaneous.¹⁶ The approach and the use or not of metal implants is controversial, being the anterior approach the logical one. Shousha & Bohem reported in 2012 a retrospective study that included 495 patients with

cervical pyogenic SD with 30 patients undergoing surgical treatment in 4 modalities (intersomatic fusion 18, corpectomy 10, transoral approach 2 and posterior percutaneous decompression 1) combined with posterior instrumentation in 17 cases and anterior plate placement in 5 cases. Of the total, 2 cases were treated by anterior corpectomy and plate fixation, these developed SD in the adjacent segment. One case warranted surgical reintervention with mesh and anterior plate replacement and another one was treated by posterior decompression and instrumentation which later developed surgical wound dehiscence that required multiple debridements, finally achieving infection control and adequate fusion.¹⁹ Gobrial et al.¹⁹ reported in 2017 a retrospective study of 59 patients with cervical pyogenic SD with neurological deficit, those who were treated surgically by decompression showed an improvement in ASIA score of 6.9 points ($p < 0.05$) and 1 point of the American Spinal Injury Association Impairment Scale (AIS).

Follow up

A useful tool in the evaluation of treatment response is ESR and CRP determinations; these are recommended during antimicrobial treatment and up to 6 months after suspension.¹⁸ The absence of fever and pain, as well as a weekly decrease of 50% of the CRP values is considered a good response to treatment, these will continue to decline until its normalization in approximately 4 weeks after treatment initiation.^{18,19}

Prognosis

The overall mortality rate reported for lumbar and thoracic pyogenic SD is 3.6-12%, with varying results according to different studies and treatment methods; however, the mortality reported for pyogenic cervical SD is as high as 21.1%. Neurological improvement occurs in approximately 60% of cases (improvement in 1 point for cases with initial assessments in Frankel D and C) with up to 6.9 points recovery in ASIA scale with a low probability of recovery for patients with Frankel A and B stratification,²⁰ The risk of sequela is as high as 50%, being the most common persistence of local or residual root pain, paresis, neurogenic bladder and musculoskeletal reflexes alterations of the lower extremities.¹⁷ The recurrence rate has been reported as high as 17%.¹⁷

Conclusion

Cervical pyogenic SD presents itself with a lower rate than thoracic and lumbar SD, it represents a diagnostic challenge due to its varied and nonspecific clinical manifestations, late radiological signs and because a delay in its accurate diagnosis and specific treatment are associated with an increased sequela risk and higher mortality.

A high suspicion of this entity must be present in individuals with risk factors. Likewise, it is very important to understand the specific role that each auxiliary diagnostic study plays in its course. The hemogram, although with a low sensitivity and specificity, is an adequate first line study for ruling out other types of pathologies, uroanalysis and urine culture are mandatory since urinary tract infection are the main origin for the development of hematogenous SD. The identification of the causative microorganism by mean of percutaneous CT guided biopsy in the absence of blood culture isolation has a positive result for micro-organic isolation in around 65% of all cases. The ESR and CRP, although nonspecific, play an important role in the diagnosis and response to treatment follow-up. First line antibiotic treatment either in its broad spectrum or specific microorganism scheme, achieves a mortality reduction in up to 55% of cases and although its duration is still a matter of debate, its absolute effectiveness leaves no room for doubt. Surgical treatment, for which absolute indications are already

established improves the patient's neurological status and decreases overall mortality as well as morbidity and risk of sequela.

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Conflicts of interest

The authors declare, have no conflict of interest in this work.

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