INFECTIONS OF THE SPINAL COLUMN

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ABSTRACT:

The rate of spinal infections are increased in parallel to the increase in immune suppressive diseases and intravenous drug abusers. At the same time new radiological techniques led the clinicians to diagnose these infections easily. In this report spinal infections which can be observed in various forms and clinical onsets are reviewed and discussed under the view of literature.

Key words: *Spinal column, infection.*

INTRODUCTION

The incidence of spinal infections appear to be rising during the recent years. On the other hand because of the variable clinical manifestations, non-specific laboratory findings and various radiographic changes, they are difficult to diagnose. Unfortunately it has been reported that diagnostic delays were common (21), and could result in serious complications, such as neurological compromise. At this point early diagnosis and treatment gain importance for the patients' life (21, 22, 64).

This paper is prepared to review the types of infections of the spinal column, their clinical manifestations, diagnostic methods and treatment procedures which can be faced.

Pyogenic vertebral osteomyelitis is an infection of vascular vertebral corpus end plate and much more seen in developing countries. The infectious agents spread via hematogenous way in the systemic infections in the basis of diabetes mellitus and long time steroid therapy (2, 12, 13, 34). Male are affected mostly and the disease is most often seen in thoracolumbar vertebra. Staphylococcus aureus is the most commonly isolated organism (12, 13, 60), but gram negative organisms and anaerobes have also been implicated (22, 29, 50). Pseudomonas aeruginosa is the most common organism responsible for the disease in intravenous drug users (34, 61). Infection progresses rapidly and the systemical features occur in the first week. The spinal symptoms starting with the low back pain and progressing to neurological deficits appear within two weeks-two years (12, 13, 50).

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During the early period, diagnosis can be made by computed tomography (CT) and magnetic resonance imaging (MRI). MRI is the most sensitive diagnostic tool and vertebral osteomyelitis is seen as low intensity in T1-weighed series, high intensity in T2-weighed. T1-weighed series give detail information such as pus and granulation tissue (6, 17, 30, 53). After 2-8 weeks, histopathological changes can be seen on X-rays (6, 11, 54). Peripheral leukocytosis is absent in approximately 50% of the cases (32, 69). The erythrocyte sedimentation rate is usually elevated and may be used as prognostic guide during treatment (32). The definite diagnosis of vertebral osteomyelitis rests on the isolation of the organism (12, 13). At this point a bone biopsy is required because blood cultures are usually sterile (40).

Therapy is ideally done with the sensitive antibiotic according to the antibiotic susceptibility test applied to the cultured bacteria from debridement, biopsy material and blood for at least six weeks (12, 13). Decompressive surgery can cause instabilization so should not be done (2, 22). Traction and fixation methods can be used in case of other pathologies such as kyphosis (2, 12, 13).

Pyogenic spinal epidural abscess, first identified by Morgagni in 1769, is written as case reports for a long time. These pathologies are seen in relatively low rates and seen with heavy neurologic findings (17, 31, 43, 58). Elsberg reported a pyogenic spinal epidural abscess seen with transverse myelitis, and identified this infection as pachymeningitidis externa (13, 17, 30).

These pathologies are seen at a rate of 2-12/100.000 and the most commomly recovered

organisms are S. aureus, aerobic and anaerobic Streptococci, gram-negative organisms especially Escherichia coli and P. aeruginosa (31, 36). In the presence of diabetes mellitus, agents from the skin infections spread hematogenously or disseminate directly from vertebral osteomyelitis (17). Traumatic vertebral lesions and epidural hematomas as well as spinal catheter applications predispose these infections (1, 17, 25). The pyogenic spinal epidural abscess are frequently seen at the thoracolumbar region while localizing to the posterior because of the posterior epidural fat tissue (17, 30).

Destruction and sclerosis of the bone structure, erosion of the vertebral end-plates and narrowed disc space are observed on X-rays (1, 13, 17). MRI shows epidural mass as iso and hyperintence signal intensity on T1 and nonhomogeneous hyperintensity on T2 weighed series (30, 43, 53). MRI may not give any information in case with meningitidis (30, 53). Gram stain of cerebrospinal fluid is usually negative while positive cultures are detected in 12-25% of cases (19).

Successfull therapy of the spinal epidural abscess is decompressive laminectomy, drainage of abscess plus 4-6 weeks of intravenous antibiotherapy (17, 31, 48, 58). Garrido and Rosenwasser (26) suggested 4 weeks of oral antibiotic therapy proceeding 2 weeks of intravenous antibiotic therapy after surgical procedures to shorten hospitalization. Nussbaum et al (48) preferred antibiotic therapy for four weeks in the absence of osteomyelitis and a longer therapy as 13 weeks in the presence of it. Before the decompressive surgery, lumbar punction should be done to diagnose the presence of meningitis (13, 31, 48). It should be performed under operation conditions because of the progression risk of neurological status. In the previous years, series cured without surgical intervention have been reported (17, 30, 43).

The morbidity rates were found to be directly related to the size of abscesses in the reported series. In the first series of epidural abscess, Dandy's mortality rate was 83% which decreased with years. The rate was lately reported to be lower than the 10% (13, 17, 31, 48, 58).

Spinal subdural abcess and empyema are rarely seen infections and reported as cases (13, 20, 23). Hematogeneous spread often causes them, but sometimes direct invasion of organisms from epidural infections, trauma and lumbar puncture, congenital lesions like dermal sinus are etiological factors for these infections (13, 20, 65). Diabetes mellitus was found to be a predisposing factor (13, 20). Etiologic organisms are usually Staphylococcus aureus and, less often, Streptococci, gram negative organisms, or Staphylococcus epidermidis (13, 20). Infection has a tendency to spread to whole subarachnoid space because of the thinning of arachnoid layer. Although pus take place in the subdural space, granulation tissue invades both spinal cord and dura (13, 38, 53).

Clinical symptoms in the acute stage are attributed to the compression of the purulent material, but in subacute and chronic stages, compression of the granulation tissue is hold responsible. In the chronic stage of the disease, compression of the intradural and extradural vessels cause venous thrombosis and heamorrhagic infarction resulting in severe clinical pathologies (13, 23, 38). X-ray studies don't help in diagnosis but myelography shows irregular thinnig of structural shape. If a block is detected in myelography which shows an epidural abscess or granulation tissue adherence, surgical intervention must be done (13). MRI is reported to be sensitive in diagnosis (10, 53).

Treatment is laminectomy, opening of the dura, drainage of the pus and taking out the granulation tissue if possible. In case of thick granulation tissue, dura can be left open and antibiotic and steroid therapy should be given after surgery (13, 23, 72).

Intramedullar pyogenic spinal cord abscesses are seen rarely and reported at a rate of 1/40.000 in the autopsy series (13, 72). The disease is usually detected in the 1st and 3rd decades; 25% of the cases are observed below age 5(13). Most commonly, lesions take place in the throcal region and 80% of the patients have multiple spinal lesions. 80% of the cases have respiratory infections, spinal infections (together with fractures morely), valvular infections of the heart and spinal disraphism (9, 13, 46). Staphylococcus and Streptococcus species are mostly grown in bacterial cultures of abscesses, however 25% of them are sterile (13). Bacteria infects the medulla by direct invasion, as secondary to trauma, hematogenous spread and lymhatic spread in the Wirchow-Robin spaces of the spinal nerves by the way of retropharyngeal, abdominal and mediastinal anastomoses (9, 13).

Symptoms differentiate according to the localization and fever and transverse myelitis findings are seen in the acute stage. In the subacute and chronic stages pain, urinary incontinance, motor and sensory

findings depending on the localization of the lesion are observed (13, 46, 72).

Myelography, CT and MRI help to diagnose the infection (6, 53). During the recent years the localization of the abscess has been determined by perioperative ultrasonography so the myelotomy place (24, 47, 53).

After the diagnosis, broad spectrum and high dose parenterally antibiotherapy should be started, and continued for a long time (13, 72). If surgical drainage is done, the patients should be followed up for long periods of time after this procedure because of high risk of recurrence (9, 13, 46). On the other side minor or major neuropathological deficits are seen in most patients after surgery. Steroid therapy should be added to treatment because it helps the regression of clinical symptoms (72).

Brucellar spondylitis is a complication of chronic brucellosis and present itself as epidemics in Latin America, Middleeast and Mediterannean regions (15, 41, 55). The most common etiological agent is Brucella melitensis however B. abortus and B. suis can also cause the disease (5, 33). The microorganisms spread hematogenously and cause spondylitis in 10-50% of the cases (5, 15, 55). Brucellosis usually localizes in lumbosacral region (15, 55).

Paravertebral abscess must be differentiated from discal hernia because of causing the same symptoms (3, 16, 33). The definite diagnosis is made by the isolation of the organism from blood or abscess cultures. Wright and Rose-Bengal agglutination tests are also important (41, 55). On the x-rays, osteophytic formation called "parrot's beak" can be observed and CT scan shows diffuse enlarging of the disc material resembling discal hernia, Schmorl nodules and vacuum phenomena with the degeneration of the Sharpy fibers (8). Changes in signal intensities of the vertebral corpuses without morphological changes, increased signal intensity of the disc in T2 weighed and contrast injected series, involvement of the soft tissue and facet joints are the main findings in MRI (35).

The treatment lasts for 6 weeks with 2 or 3 antibiotics (5, 8, 15, 68). If rapid progression is observed in neurological findings, decompressive surgery can be done. The recurrence rate is 20% in the first year, so the patients should be followed up by serological tests (41, 68).

Paraspinal tuberculosis is seen in two forms (Tuberculosis spondylitis and spinal tuberculosis) which are different from each other clinically and radyologically. Tuberculosis spondylitis (Pott's paraplegia) is seen mostly in throcolumbar vertebra corpuses in the first decade of life (39, 49). While more than one vertebra is affected generally, posterior parts of vertebra are rarely involved in the disease. 5-47% of the cases are complicated neurologically (16, 28, 39, 49). PPD test is done for diagnosis but negative results are seen in 15% of the patients (15). On X-rays, decalcification of the corpus is prominent at the anterior parts and sclerotic changes are also detected (13, 63). Paravertebral soft tissue abscesses (psoas=Brody abscess) can be seen on x-rays which are observed as blocking in myelography at the abscess localization (13, 63). CT is diagnostic in the early period showing lysis and destruction of the corpus (13, 16, 63). Aftar contrast injection, abscess became prominent which can be mixed with metasthasis (16, 45).

CT guided percutanous drainage of the abscesses can be done for both diagnosis and treatment (16, 63). C1 and C2 involvement are rare and cause subluxations. Retropharyngeal abscesses which treat life can also be seen (39). The main treatment is against Mycobacterium tuberculosis and in case of abscesses causing neurological symptoms, decompressive surgery can be done. If vertebral column instability and pathologies such as kyphosis and scoliosis are present, corrective operations are indicated (39, 45).

Spinal tuberculosis can be seen in the epidural space, duramatter, arachnoid and spinal cord without involvement of the bone structure (7, 49). Out of these rare pathologies, intramedullary tuberculoma is seen 30-100 times less than the cerebral tuberculoma (13). Arachnoidal involvement causes transverse mlyelitis by affecting vascular structure (7, 13). Mostly subdural involvement does occur diffusely and steroid should also be given in the treatment (7, 13, 45). Epidural space tuberculosis causes discal hernia like symptomps and must be differentiated (7, 13). At this point the most sensitive diagnostic tool is MRI (66). Treatment consists of laminectomy, exision of tuberculoid granular structure and classical antituberculoid therapy (7, 13, 45).

Fungal infections of spine are seen sporadically. Diabetic and immune deficient people are

predisposed. Patients having lymphoma, leukemia, aplastic anemia, chemotherapy and immune supressive therapy are more affected (13, 28).

Actinomycosis, nocardiosis, aspergillosis, coccidiomycosis, blastomycosis and candidosis are mostly seen spinal fungal infections. They are important because of severe clinical results although seen rarely. Treatment is done with antifungal drugs and decompressive and drainage procedures are applied in the presence of abscess (13, 44, 73).

Parasitic infections can be seen up to a rate of 5% among spinal infections in the developing countries. The main parasitic infections are shistosomiasis, echinococcosis and cysticercosis (37, 71).

Cysticercosis, caused by the larva stage of tenia solium, frequently involves central nervous system (CNS) (71). This infection clinically manifests itself in a wide range from meningoencephalitis to obstructive hydrocephalus (Brun's syndrome). Spinal column is involved in 2-5% of the cases (37). Caudal involvement is frequent and causes low back and leg pains as well as sphincter problems (71). On the other hand intramedullary involvement occurs multifocally (37).

Diagnosis is made by certain laboratory tests like indirect hemagglutination, agar gel precipitation, immunelectrophoresis, indirect immunoflorescence and blastoid transformation (37, 71). Enzyme linked immunoelectrotransfer blot test used during the previous years for the diagnosis of cysticercosis was reported to be 100% specific and 98% sensitive (70). Treatment is done with praziquantel and complete improvement is detected in up to 50% of the cases. Albendasole is the second choice and steroid is used if arachnoidal involvement is present. Decompressive surgery is indicated in cases of compression (37, 70, 71).

Echinococcosis is sporadically seen in our country and cerebral involvement is frequently seen instead of spine (28, 52). The agent, Echinococcus granulosa involves liver and lungs and spreads hematogenously. CNS is affected in 2-3% of the cases (13, 27). Bone involvement occurs at a rate of 2% especially at the lumbosacral region causing paraparesia (52). In 50% of the cases, vertebrae corpus is affected at first and there after pedincules and laminas are involved. The parasite spreads through periost and ligaments so the disc is spared. Generally, the infection disseminates to epidural space however intradural and extramedullary localizations are also seen (13, 28). Casoni skin test, Weinberg Ghendini complement fixation and immunoelectrophoresis tests are used for diagnosis. These tests became negative after 6-18 months of succesfully therapy (28). Hydatic osteopaty and multifocal lacunar osteolysis is typical on X-rays. CT shows lesions as hypodence regions in the corpus (13). Treatment is surgical excision plus antihelmitic drugs. The mortality and recurrence rates are 3-4% and 30-40%, respectively, even if surgical procedures are successfull (13, 28) because of the risk of rupture related to the local invasion ability of parasite (14).

Shistosomiasis is a disease caused by Schistosoma hematobium, S. mansoni and S. japonicum. They usually habitate in the urinary tract and spinal dissemination occurs by penetration (62). They also spread to the brain and spinal column hematogenously (13, 62). S. mansoni localizes in the lumbosacral region and causes myelopathy. Conus medullaris is affected in 75% of the cases and granulamateous lesions are seen in the chronic stage of the disease. Hematological examinations are no help for the diagnosis however CT and MRI show mass lesions of granulomas at T12-L1 levels (13). Treatment consists of antihelmintic therapy with praziquantel and oxamiquine and surgery in case of compressive mass lesions (13, 62).

Postoperative infections are reported as 0.2-5% after lumbar disc surgery. These infections can also occur after lumbar punction, myelography, lumbar symphatectomy, chemonucleosis and spinal epidural anesthesia (1, 18, 21, 56, 57).

Clinical symptoms generally appear after 3 days-8 months proceeding the surgical intervention and 80% of the infections occur within 3 weeks after the operation (18, 28, 56). The initial symptom is low back pain which increases with movement and disseminates to buttock, scrotum, perinea, abdomen and legs (18, 56). Fever is seen in 30-50% of the patients (56, 57). In physical examination, paravertebral muscle spasm preventing movement is detected (18, 56). 10-12% of the cases have wound infections while 0-8% of them have pus drainage (42).

Sedimentation rate increases over 20 mm/hour most of the time and 18-30% of the patients have leukocytosis (>10.000/mm^3) (18, 32, 56, 69). PPD test should be applied to all patients to rule out tuberculosis (39). There is a tendency to follow patients with C-reactive protein during the recent years (21).

Many signs can be detected radiologically however 1 to 8 months are needed to observe them which seems to be a disadvantage. The first finding is the demineralization of the end-plate which can be detected after 2-4 weeks. 4-12 weeks later, sclerosis is observed causing increased density on X-rays. Irregularity of the end-plate, pedincular and corpus destruction, increased disc space (balloning), intradiscal and/or intraosseus gas appearance, circumferential bony formation and proceeding spur formation occuring after 6-8 months are the main signs on X-rays. The latest sign is spinal fusion and needs a 2 years time (8, 18, 28, 56). CT shows abscess formation and other X-ray findings however MRI differentiates the infection from fibrosis and recurrent discal hernia sensitively (59, 67). Nuclear sintigraphy is 85% sensitive for the early stages and can detect infection by using Technetium 99 after 7 days proceeding clinical symptoms (4, 28, 67). This time is 14 days with gallium (67). The main finding is increased focal activity of vertebral end plates. It can certainly be differenciated from vertebral osteomyelitis but differentiation from tumor and fracture healing is hard (28, 67).

The etiological agents of post-operative infections can be isolated in only 50% of the patients (13, 18, 28, 56). The agents were tried to be isolated with blood cultures formerly but nowadays the specimens are aspirated by needles or nucleotom and cultured. The diagnosis of the infection is done with the material collected by the nucleotom after the disc surgery (18, 26, 51, 56). In this way the appropriate antibiotic can be started in the early period. Evacuation and debriding the disc space is also an advantage (51).

Treatment with bed rest and antibiotherapy is reported to be successfull. Bed rest prevents pain rather than infection healing and helps returning to work early. Braces and trunc casts are other alternatives to shorten the bed rest interventing pain. Antibiotherapy can be done in two different ways (18, 28, 42, 51, 57). First and most used is 4-6 weeks parenteral application. Second is parenteral therapy until the sedimentation rate returns to normal and a 2-3 week peroral antibiotherapy afterwards (18, 28, 42, 51). If the agent is not isolated, antistaphylococcal drugs such as Vancomycin and a broad spectrum antibiotic such as third generation cephalosporins are combined (13, 18, 28, 57). Additionally analgesic and muscle relaxant drugs must be used. Some authors have reported using steroids to decrease the pain (28). Surgical intervention is restricted and done only when a diagnostic difficulty is faced, in cases of abscess presence and instability (18, 28, 56). Succesful results have been obtained with early diagnosis and appropriate treatment (13, 18, 28, 42, 51, 57).

Spinal infections should not be thought as rare anymore and should be suspected in the patients showing appropriate clinical symptoms. Early diagnosis and therapy can prevent most of the complications which can result from untreated infections.

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