

Infectious Spondylodiscitis After Appendectomy for Perforated Appendicitis: Case Report

Perfore Apendisit Nedeni ile Yapılan Apendektomi Sonrası Enfeksiyöz Spondilodiskit

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ABSTRACT We described an infectious spondylodiscitis case after appendectomy to indicate the importance of clinical suspicion for the diagnosis. A 24-years-old female referred with low back pain. She had an appendectomy surgery 3 months ago. She had no neurodeficitis and fever. Sedimentation and CRP were slightly elevated. After contrast administration, enhancement in vertebral bodies and disc at the L5-S1 was detected in T1-weighted MRI. We hospitalized the patient as having infectious spondylodiscitis and treated with antibiotics. The diagnosis of spondylodiscitis is difficult and often delayed due to non-specific physical, laboratory and radiographic findings. A high clinical suspicion is necessary for the early diagnosis.

Keywords: Appendectomy; low back pain; spondylodiscitis; rehabilitation

ÖZET Tanı için klinik şüphenin önemini belirtmek adına, apendektomi sonrası gelişen bir enfeksiyöz spondilodiskit olgusu tanımlıyoruz. Yirmi dört yaşındaki bir kadın olgu, bel ağrısı ile başvurdu. Üç ay önce apendektomi cerrahisi geçirmiş idi. Nörodefisiti ve ateşi yoktu. Sedimentasyon ve CRP hafifçe yüksekti. Kontrast madde sonrasında T1-ağırlıklı, MRI'da L5-S1'de vertebra korpuslarında ve diskte tutulum saptandı. Enfeksiyöz spondilodiskit olduğu düşünülen olgu hospitalize edildi ve antibiyotik başlandı. Spesifik olmayan fiziksel, laboratuvar ve radyografik bulgular nedeni ile spondilodiskit tanısı zordur ve sıklıkla gecikmektedir. Erken tanı için yüksek klinik şüphe gereklidir.

Anahtar Kelimeler: Apendektomi; bel ağrısı; spondilodiskit; rehabilitasyon

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Infectious spondylodiscitis is an infection of vertebral bodies, endplates and discs. The most common causative organism is staphylococcus aureus.¹ The incidence has been estimated to be 0.4 to 2.4 per 100.000/year. In adults, it usually starts at the vertebral endplates and affects two adjacent vertebral bodies with the intervertebral disc. Lumbar spine is the most affected area. It may also spread to posterior elements of the spine, the paravertebral area and the epidural space.¹⁻⁴

The symptoms are non-specific and diagnosis is often delayed. The most common complaint is back pain. Fever is detected in less than 20% of patients. Localized spinal tenderness, paraspinal muscle spasm, limited spinal movement and radicular pain are common.^{2,3} Neurological deficit may be seen in 10-50% of patients.^{1,4}

Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels are usually elevated. They are correlated with activity of infection, but not specific for infectious spondylodiscitis.^{1,3} The infectious agent can be identified by CT-guided percutaneous biopsy.⁴ Magnetic resonance imaging (MRI) is the most specific imaging modality.⁵

The treatment of infectious spondylodiscitis includes the use of intravenous antibiotic therapy followed by oral antibiotic therapy. The optimal duration of antibiotic therapy is unclear.^{1,2}

We describe a case of infectious spondylodiscitis that occurred after appendectomy for perforated appendicitis.

CASE REPORT

A 24-year-old woman was referred with a 1 month-history of low back pain that is aggravated by movement and not relieved by neither rest nor analgesics. She reported no pain that radiates into the buttock or leg. Her pain did not increase by valsalva maneuvers. She had an appendectomy surgery for perforated appendicitis 3 months before. She had no history of trauma or any systemic disease.

On physical examination, the lumbar range of motion was severely limited. There was localised tenderness at L5-S1 level. The straight leg raising test was positive at 45° in both legs. The neurologic examination was normal. She had no fever. Clinical examination of the cardiovascular and respiratory systems revealed no abnormality.

The laboratory data revealed elevated ESR (30 mm/h) and CRP (31.3 mg/L) levels. Complete blood cell count (white blood cell count: 5650 cells/mm³, hemoglobin: 12.6 g/dl, platelet count: 256.000 cells/mm³), biochemical tests and urine analysis were normal. Brucella agglutination test was negative. Urine culture and three sets of blood cultures were negative. The x-ray images of the lumbar spine, pelvis and chest and chest CT scan were normal.

Lumbosacral MRI showed decreased signals in L5-S1 vertebral bodies and disc on T1-weighted images and a slight increase on T2-weighted images. After contrast agent, enhancement in the

same areas and also paravertebral area were detected (Figure 1). Sacroiliac MRI was normal. Although MRI clearly identified the infectious disease, we did not perform bone scan.

Based on these findings, we hospitalized the patient as having infectious spondylodiscitis due to perforated appendicitis. The patient did not accept to undergo invasive diagnostic procedure and, therefore empirical broad-spectrum intravenous antibiotics were prescribed. Intravenous ciprofloxacin (2x400 mg/day) and ampicillin sulbactam (4x1.5 gr/day) were given for 4 weeks and then switched to oral ciprofloxacin (2x500 mg/day) and sultamicillin (4x750 mg/day). At the end of the therapy, pain intensity and lumbar spine movements were improved. ESR and CRP levels were normal.

DISCUSSION

We described a 24-year old woman with a history of low back pain 2 months after perforated appendicitis. She was afebrile, and remained afebrile in our clinic. White blood cell count was normal. The only abnormal laboratory test results were mildly elevated ESR and CRP. The MRI showed abnormalities consistent with a L5-S1 infectious spondylodiscitis.



FIGURE 1: Contrast-enhanced T1-weighted sagittal imaging shows enhancement in the vertebral endplates and the anterior side of the intervertebral disc at the L5-S1 (arrow at the left), and also high signal intensity in the paravertebral and epidural space is seen (arrow at the right).

Infectious spondylodiscitis can develop from hematogenous spread of bacteria, direct inoculation and infections in adjacent structures.¹ Hematogenous way is the most important spreading way usually from genitourinary, respiratory or gastrointestinal tract.³ We believe that the patient may have had an infectious spondylodiscitis due to a transient bacteraemia after perforated appendicitis. Our patient rejected the diagnostic biopsy. For this reason, we started empirical antibiotic therapy coverage for staphylococci and gram-negative bacilli for 8 weeks. To our knowledge, this is the first case of spondylodiscitis after perforated appendicitis in the literature.

The diagnosis is difficult because of non-specific symptoms and negative blood cultures.^{3,6} Gadolinium dimeglumine (Gd-DTPA) enhanced T1-weighted MRI is an essential part of the diagnosis.^{5,7} The infectious agent can be identified by CT-guided percutaneous disc biopsy.^{4,8} In our patient, MRI showed findings suggestive of infectious spondylodiscitis. However, the infectious agent could not be detected because the patient did not want to undergo a biopsy procedure.

Degenerative disc disease (DDD), inflammatory spondylodiscitis, and vertebral tumors may simulate infectious spondylodiscitis.^{2,5} Infectious spondylodiscitis may mimic type 1 Modic DDD. Low signal intensity in endplates on T1-weighted imaging and high signal intensity in the same areas on T2-weighted imaging may occur in both conditions. Contrast enhancement in the disc and endplates may also occur in both conditions. In contrast to DDD, the disc is typically hyperintense on T2-weighted imaging in spondylodiscitis. Also, eroded or destroyed endplates, presence of paraspinal/epidural involvement and elevated CRP levels are usually detected in spondylodiscitis rather than DDD.⁹ Another differential diagnosis is inflammatory spondylodiscitis such as spondyloarthropathies and SAPHO syndrome. Multiple foci of spondylodiscitis are more common in inflammatory conditions and paraspinal/epidural involvement is not observed in inflammatory spondylodiscitis.¹⁰ Sacroiliac joint involvement that is commonly seen in inflammatory spon-

dyloarthritis could be useful to differentiate this pathology from spondylodiscitis.

Infectious spondylodiscitis should be also distinguished from vertebral malignancies. The disc is relatively preserved and vertebral compression fractures may be seen in malignancies.³ MRI is a useful method for differentiating infection and malignancy.¹ In our case, we observed hyperintense disc and paraspinal/epidural involvement on T2-weighted images, normal sacroiliac MRI findings, single focus of spinal inflammation. As a result of these findings, we did not consider degenerative disease, inflammatory spondylodiscitis or vertebral tumors.

Tuberculosis and brucellosis may also be considered the cause of spondylodiscitis.⁸ Tuberculous spondylitis involves mainly thoracic vertebra and it is more associated with neurological deficit. Relatively preserved disc and multilevel involvement are more frequent in tuberculosis than in pyogenic spondylodiscitis.^{1,5} High-grade fever is detected more frequently in brucellosis than in pyogenic spondylodiscitis.⁸ Epidural/paravertebral abscesses may be more frequent in tuberculosis or brucellosis.⁵ The case we present here had involvement of two adjacent vertebra and intervertebral disc and she revealed no neurodeficit. The chest X-ray and chest CT scan revealed no signs of pulmonary tuberculosis. Brucella agglutination test was negative.

Our patient did not agree to undergo biopsy and therefore we started empirical broad-spectrum intravenous antibiotics. A meta-analysis of randomized trials of antibiotic therapy for bone infections showed no significant differences in the outcome when comparing with the specific antibiotic therapy. Similarly Lora-Tamayo et al. found no significant difference between the empirical therapy and specific therapy.^{6,7} We treated the patient with antibiotics for 8 weeks. At the end of the therapy, improvements in pain intensity, lumbosacral range of motion and CRP level were observed.

CONCLUSION

In conclusion, the diagnosis of spondylodiscitis is difficult and often delayed due to non-specific

physical, laboratory and radiographic findings. A high clinical suspicion is necessary for the early diagnosis.

Conflict of Interest

Authors declared no conflict of interest or financial support.

REFERENCES

1. Yu SH, Kim DH, Kim HS, Nam KH, Choi BK, Han IH. Infectious spondylodiscitis by uncommon pathogens: a pitfall of empirical antibiotics. *Korean J Spine* 2016;13(3):97-101.
2. Sobottke R, Seifert H, Fätkenheuer G, Schmidt M, Gossmann A, Eysel P. Current diagnosis and treatment of spondylodiscitis. *Dtsch Arztebl Int* 2008;105(10):181-7.
3. Cheung WY, Luk KD. Pyogenic spondylitis. *Int Orthop* 2012;36(2):397-404.
4. Spira D, Germann T, Lehner B, Hemmer S, Akbar M, Jesser J, et al. CT-guided biopsy in suspected spondylodiscitis--the association of paravertebral inflammation with microbial pathogen detection. *PLoS One* 2016;11(1): e0146399.
5. Longo M, Granata F, Ricciardi K, Gaeta M, Blandino A. Contrast-enhanced MR imaging with fat suppression in adult-onset septic spondylodiscitis. *Eur Radiol* 2003;13(3):626-37.
6. Lora-Tamayo J, Euba G, Narváez JA, Murillo O, Verdaguer R, Sobrino B, et al. Changing trends in the epidemiology of pyogenic vertebral osteomyelitis: the impact of cases with no microbiologic diagnosis. *Semin Arthritis Rheum* 2011;41(2):247-55.
7. Luzzati R, Giacomazzi D. The empirical antibiotic therapy of pyogenic vertebral osteomyelitis. *Semin Arthritis Rheum* 2012; 41(4):e9.
8. Skaf GS, Kanafani ZA, Araj GF, Kanj SS. Non-pyogenic infections of the spine. *Int J Antimicrob Agents* 2010;36(2):99-105.
9. Rahme R, Moussa R. The modic vertebral endplate and marrow changes: pathologic significance and relation to low back pain and segmental instability of the lumbar spine. *AJNR Am J Neuroradiol* 2008;29(5):838-42.
10. Kubaszewski Ł, Wojdasiewicz P, Rożek M, Słowińska IE, Romanowska-Próchnicka K, Słowiński R, et al. Syndromes with chronic non-bacterial osteomyelitis in the spine. *Reumatologia* 2015;53(6):328-36.