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Case Report

Cervical myelopathy caused by vertebral osteomyelitis after below-knee amputation for diabetic foot: A case report on early diagnosis and rehabilitation

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ABSTRACT

Vertebral osteomyelitis (VO) usually results from hematogenous spread, while diabetic foot osteomyelitis (OM) spreads contiguously. Vertebral OM secondary to diabetic foot OM is rare. Herein, we reported a rare case of a 62-year-old male with multiple comorbidities, including diabetes mellitus, end-stage renal disease, and cardiovascular disease, who developed cervical VO four months after undergoing below-knee amputation due to diabetic foot OM. The patient presented with neck pain, progressive limb weakness, and respiratory insufficiency. Imaging confirmed cervical spondylodiscitis with compressive myelopathy. Due to a high Charlson Comorbidity Index score of 9 and poor general condition, surgical intervention was contraindicated. Conservative treatment, including intravenous antibiotics, cervical orthosis, and comprehensive rehabilitation, was initiated. Although the patient's condition initially deteriorated to American Spinal Injury Association Impairment Scale Grade A, he showed gradual neurological improvement to Grade D with a neurological level of C6 after one year of continuous rehabilitation. This case highlights the need to consider VO as a potential complication in patients with high-risk diabetic foot OM and suggests that early conservative management combined with structured rehabilitation can lead to favorable neurological outcomes, even in patients with severe comorbidities.

Keywords: Charlson Comorbidity Index, diabetic foot, nonsurgical treatment, rehabilitation, vertebral osteomyelitis.

Osteomyelitis (OM) is an inflammatory condition characterized by bone destruction caused by infection. It is classified into two primary types: contiguous spread and hematogenous dissemination.[1] Contagious OM occurs due to direct extension from adjacent infected soft tissues or following direct inoculation. A classic example is diabetic foot infection, where severe cases are associated with a high prevalence of underlying OM reported in up to 50% of cases.[2] In contrast, hematogenous OM results from the seeding of bone by pathogens circulating in the bloodstream, originating from a distant infectious focus. Vertebral osteomyelitis (VO) is a typical manifestation of this form.[1] These distinct pathogenic mechanisms of OM associated with diabetic foot infections, and VO should be considered when evaluating at-risk patients.

Vertebral osteomyelitis can affect patients with diabetes mellitus, those receiving intravenous medications, or those who are immunocompromised, and it predominantly affects the lumbar spine; cervical involvement remains rare.[3] Furthermore, in cases of cervical OM, clinical symptoms are often nonspecific and vague, making early diagnosis challenging.[3] Hence, magnetic resonance imaging (MRI) evaluation and initiation of intravenous antibiotic therapy are frequently delayed. Previous case reports have described cervical VO in patients with diabetes and odontoid process abscesses or in those who with a history of intravenous drug use or treatment, with progression to paralysis.[4,5] However,

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to the best of our knowledge, no cases in which cervical VO led to tetraplegia in a patient who had previously undergone below-knee amputation due to diabetic foot infection have been reported.

Although surgical treatment is often considered in cases of cervical VO, this case is noteworthy for the development of cervical myelopathy likely due to hematogenous dissemination from diabetic foot–related OM, an atypical route of infection. Moreover, despite severe comorbidities that precluded surgical intervention, the patient achieved meaningful neurological recovery through timely conservative treatment and comprehensive rehabilitation. This report presents a rare case of a patient who underwent below-knee amputation for a diabetic foot four months prior and was subsequently diagnosed with cervical VO.

CASE REPORT

A 62-year-old male with a history of diabetes mellitus, coronary artery occlusive disease, non-ST elevation myocardial infarction, peripheral artery occlusive disease, chronic kidney disease on hemodialysis, hypertension, asthma, and subclinical hypothyroidism was diagnosed with chronic OM of the right foot due to diabetic foot complications and underwent right below-knee amputation four months prior to presentation. The patient was admitted to the Department of Rehabilitation Medicine at a university hospital for comprehensive prosthetic rehabilitation therapy (Figure 1). This study was approved by our Institutional Review Board and complies with the CARE guidelines. Written informed consent for publication was obtained from the patient.

The patient complained of neck pain on hospital day (HD) 20, prompting a cervical radiograph, which revealed disc space narrowing without other significant abnormalities. However, by HD 27, the patient's neck pain worsened, increasing from a Visual Analog Scale score of 3 to 7. Although cervical MRI was initially planned, the procedure could not be performed because of the patient's inability to maintain the required position secondary to severe pain. Therefore, a cervical computed tomography (CT) was conducted, which revealed disc space destruction with endplate bony erosion at C6-7, bilateral foraminal stenosis at C6-7, and left foraminal stenosis at C5-6 (Figure 2a). Despite the absence of fever, elevated C-reactive protein levels raised the suspicion of cervical VO (Table 1). Hence, intravenous vancomycin administration was initiated, and a Philadelphia neck brace was used to minimize cervical motion. On HD 41, nerve conduction studies showed reduced amplitude of sensory nerve action potentials in the bilateral median, ulnar, and left sural and superficial peroneal nerves, consistent with diabetic peripheral

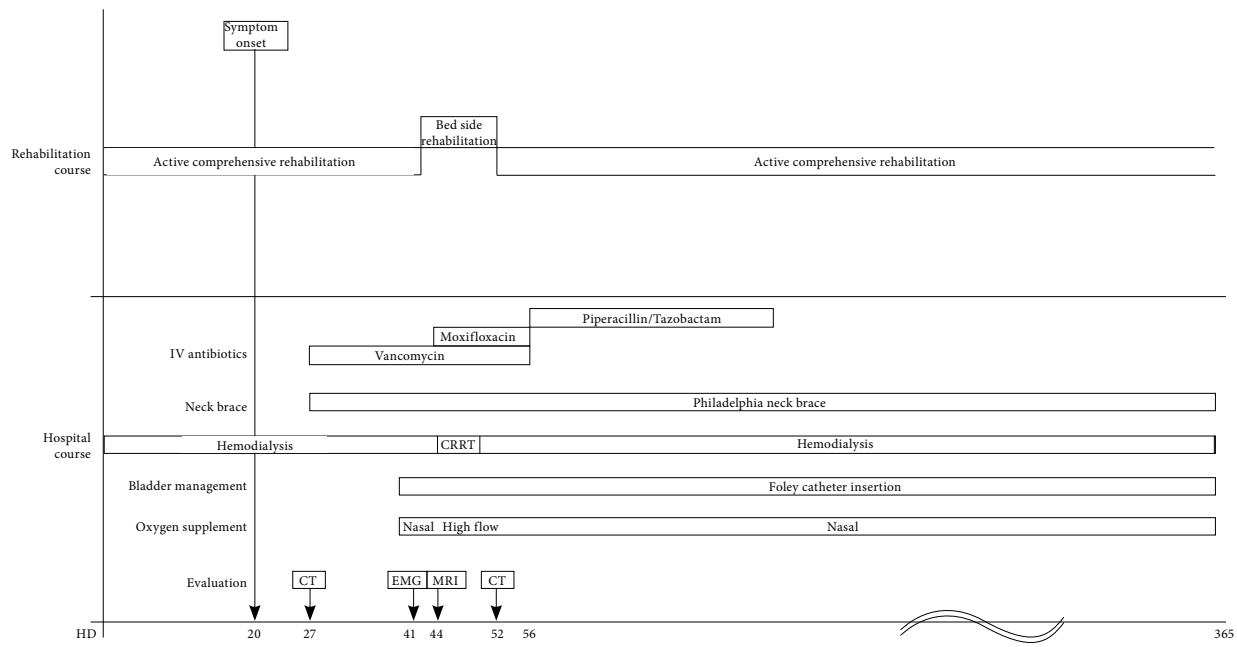

Figure 1. Hospital course of the patient.

CRRT: Continuous renal replacement therapy; CT: Computed tomography; EMG: Electromyography; MRI: Magnetic resonance imaging HD: Hospital Day.

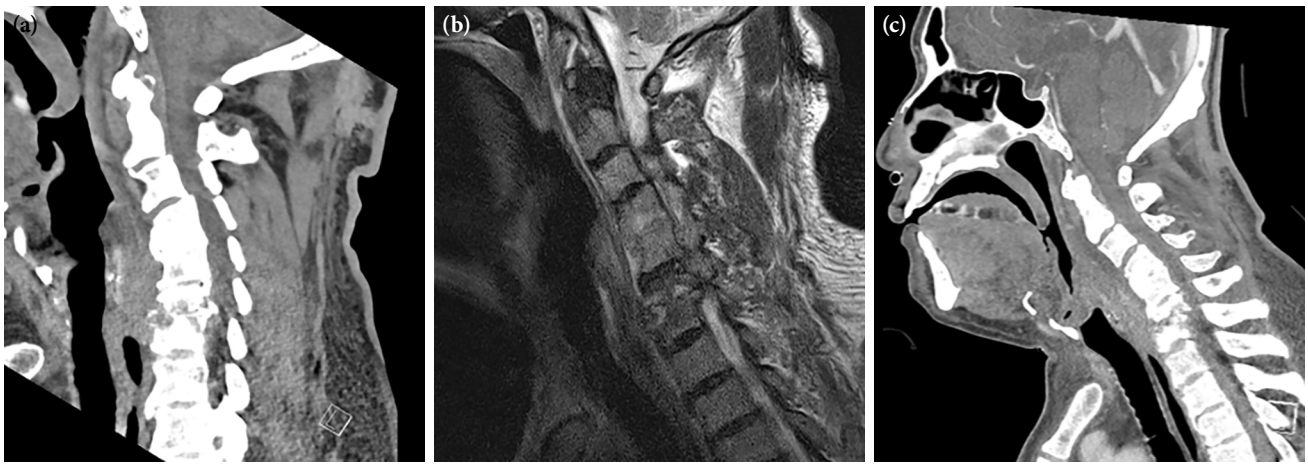

Figure 2. Radiologic progression of cervical vertebral osteomyelitis. **(a)** Cervical CT performed on HD 27 showing disc space narrowing and endplate erosion at C6-7. **(b)** Cervical MRI on HD 44 showing disc space destruction, endplate erosion, and subtle enhancement at C6-7, consistent with osteomyelitis, along with central canal and foraminal stenosis. **(c)** Follow-up cervical CT on HD 52 revealing progression of spondylodiscitis at C6-7, increased bony destruction, a prevertebral and anterior epidural enhancing soft tissue mass suggestive of phlegmon, and evidence of spinal cord compression with C6-7 subluxation.

CT: Computed tomography; HD: Hospital Day; MRI: Magnetic resonance imaging.

polyneuropathy. Somatosensory evoked potentials revealed delayed latencies and reduced amplitudes in upper extremity responses, suggesting impaired dorsal column function. Motor evoked potentials demonstrated prolonged central motor conduction time in the upper limbs and absent responses in the left lower limb, consistent with cervical myelopathy. From HD 42, the patient developed weakness in all four extremities. Neurological examination revealed the increased deep tendon reflexes and positive Babinski sign, indicating upper motor neuron involvement. Sensory examination demonstrated impaired proprioception and vibration sense in four extremities, consistent with dorsal column dysfunction. Additionally, respiratory insufficiency developed, necessitating nasal oxygen supplementation. By HD 44, further weakening and an increase in C-reactive levels led to the decision to perform cervical MRI (Table 1). T2-weighted MRI revealed disc space destruction with endplate erosion and subtle contrast enhancement at C6-7, consistent with OM. Additionally, severe central

TABLE 1
Changes in neurological function over time

	HD 1	HD 42	HD52	5 month follow-up	1 year follow-up
AIS grade (A-E)	E	C	A	C	D
Neurological level of injury		C5	C5	C5	C6
ASIA sensory score (0-112)	106	61	42	61	73
Upper Extremity Motor Score (UEMS) (0-50)	50	29	20	26	36
Lower Extremity Motor Score (LEMS) (0-50)	35	17	0	7	18
Total Motor Score (TMS) (0-100)	85	46	20	33	54
Spinal Cord Independence Measure (SCIM) total score (0-100)	73	6	13	20	20
SCIM mobility subscore (0-40)	16	0	0	3	3
CRP (mg/L)		24.07		1.28	
Neck pain (Visual Analog Scale) (0-10)	1	8	7	2	2

HD: Hospital Day; AIS: American Spinal Injury Association (ASIA) Impairment Scale; UEMS: Upper Extremity Motor Score; LEMS: Lower Extremity Motor Score; TMS: Total Motor Score; SCIM: Spinal Cord Independence Measure; CRP: C-reactive protein.

canal stenosis and bilateral foraminal stenosis at C5-6 and C6-7 were observed, indicating possible compressive myelopathy (Figure 2b). The patient exhibited minimal spontaneous urination. However, due to the high risk of neurogenic bladder associated with cervical myelopathy, Foley catheterization was performed, and a post-void residual volume of 600 mL was measured. Thereafter, the Foley catheter was maintained. In addition, the patient showed signs of respiratory insufficiency due to high cervical myelopathy, and nasal oxygen therapy was enhanced to high-flow oxygen therapy. After a neurosurgical consultation, surgical management was deemed high-risk, owing to the patient's septic condition, hemodynamic instability, and multiple underlying comorbidities. Therefore, conservative treatment was continued, and moxifloxacin administration was added. The patient was admitted to the intensive care unit for three days and managed with continuous renal replacement therapy. On HD 52, follow-up cervical CT revealed progressive spondylodiscitis at C6-7, with increased inflammation and bony destruction of the C6-7 vertebral body (Figure 2c). A large enhancing soft tissue mass was observed in the prevertebral and anterior epidural spaces, suggestive of phlegmon. In addition, there was evidence of probable spinal cord compression with progression of cervical spine kyphosis and C6-7 subluxation. Once the patient's vital signs stabilized, hemodialysis was resumed, and oxygen therapy was de-escalated to nasal oxygen. From HD 56, in consultation with the infectious diseases team, the antibiotic regimen was changed to intravenous piperacillin/tazobactam, which was continued for six weeks.

On HD 42, the patient developed tetraparesis and respiratory insufficiency, prompting high-flow oxygen therapy. Neurological examination revealed an American Spinal Injury Association Impairment Scale (AIS) C injury at the C5 level, with an initial sensory score of 61 and motor score of 46. Owing to the patient's deteriorating condition, rehabilitation was temporarily limited to bedside passive exercises. Neurological function further declined by HD 52, with AIS motor and sensory scores dropping to 20 and 42, respectively. On HD 52, the sensory examination revealed a complete loss of light touch and pin prick sensation in the S4-S5 dermatomes. Voluntary anal contraction was absent, and deep anal pressure was also not perceived. After stabilization and transition back to nasal oxygen therapy on HD 56, active rehabilitation was resumed. Active rehabilitation was provided five days a week. Physical therapy included passive range of motion exercises with gentle stretching of both lower extremities, facilitation of voluntary movements at both lower extremities, resistance training, and trunk stabilization exercises. Occupational therapy involved passive range of motion and gentle stretching exercises for both upper extremities, facilitation of upper extremities movements, and training in compensatory techniques. As part of neurorehabilitation modalities, functional electrical stimulation was applied to both quadriceps and both deltoid muscles. Over the course of one year, the patient gradually improved to AIS D with a motor score of 54 and sensory score of 73, achieving a neurological level of C6 (Table 1).

DISCUSSION

This report describes a rare case in which a peripheral infection, originating from OM following a below-knee amputation, spread hematogenously and led to cervical VO and subsequent myelopathy. While VO is most commonly associated with risk factors such as intravenous drug use, unknown sources of bacteremia, or persistent bacteremia lasting more than one day. Cases secondary to peripheral OM have not been widely documented. This case highlights the importance of recognizing that hematogenous spread from peripheral infections, particularly in patients with multiple comorbidities, can result in VO and spinal cord involvement.

While surgical management is generally associated with favorable outcomes in cases of VO,[6] patients with significant comorbidities may face increased risks with surgery. The patient in this case had a Charlson Comorbidity Index (CCI) score of 9. Previous studies have demonstrated that a CCI score ≥4 is associated with higher rates of surgical complications and reoperations.[7] For such high-risk patients, conservative treatment may be the only feasible option. In these cases, early and continuous rehabilitation can help prevent muscle contractures and atrophic changes, potentially contributing to more favorable clinical outcomes.

Known risk factors for VO include intravenous drug use, an unknown source of bacteremia, and persistent bacteremia lasting more than one day.[1] Due to differences in mechanisms, VO secondary to diabetic foot-related OM has not been previously reported. However, this case illustrates that in patients with multiple comorbidities, there may be a risk of VO developing from the contiguous spread of

existing OM. Therefore, in such high-risk patients, early diagnosis and prompt management are crucial to improve outcomes when VO is suspected.[8]

Previous studies have reported that treatment failure occurs in approximately 75% of patients with VO within 4.7 months after diagnosis.[9] Patients with a high CCI are at greater risk for treatment failure.[10] Additionally, in patients with pyogenic VO, a high CCI and a low AIS at diagnosis have been associated with an increased risk of persistent severe neurological deficits.[3] In the present case, despite a high CCI, the patient did not experience treatment failure and showed neurological improvement, achieving AIS Grade D with a neurological level of C6 at one-year follow-up. These findings suggest that even in patients with a high comorbidity burden, early and intensive rehabilitation may play a key role in preventing long-term sequelae and improving treatment outcomes and contribute to motor recovery in patients with VO.

In conclusion, this case highlights the possibility of hematogenous spread of infection from a peripheral site, such as diabetic foot OM, leading to cervical VO and subsequent myelopathy. In patients with multiple comorbidities, early suspicion and diagnosis are crucial. Conservative treatment with antibiotics, orthosis, and early, continuous rehabilitation may help avoid surgical risks and support neurological recovery even in complex cases.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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