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Case Report
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Recurrent Spondylodiscitis in a Cirrhotic Scuba Diving Instructor

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Abstract

Spondylodiscitis is a challenging infection in cirrhotic patients. Liver dysfunction makes the course of the infection more aggressive. We report the case of a 56-year-old man with a medical history of abstinent alcoholic cirrhosis and repeated infectious events including cervical abscess. He had been scuba diving instructor for 20 years. He presented to the emergency room for chills, rigor, and high temperature.

A *Citrobacter freundii* prostatitis was identified and treated. Then he reported mechanical lower back pain persisting despite analgesia. MRI of the lumbar spine demonstrated L5/S1 spondylodiscitis with an unusual signal modification of S1 vertebral body. This case enlightens the high susceptibility to bacterial infections in cirrhosis. Special attention should be given in patients who scuba dive regularly in case of bacteremia with back pain.

Keywords: Cirrhosis; Spondylodiscitis; Dysbaric osteonecrosis

Background

Challenged by these repeated spinal infections, two predisposing factors must be discussed: Cirrhosis and potential dysbaric osteonecrosis. Firstly, cirrhosis is a major risk factor for infectious events with increasing frequency of multi-drug resistant bacteria related infections. This infectious predisposition is secondary to the interaction of internal factors such as relative-acquired-immunodeficiency involving both innate and adaptative immunity, increased intestinal mucosa permeability, reduced bile flow or dysbiosis characterized by increased populations of gram-negative bacilli (Enterobacteriaceae) and gram-positive cocci (Streptococcaceae and Enterococcaceae) [1-3]. External factors as proton-pump inhibitor overuse or multiple antibiotic courses also contribute to this susceptibility [4]. Those factors lead to chronic immune system exposure to pathogen bacteria notably by translocation which results in chronic inflammation and recurrent sepsis [5,6]. By those mechanisms, cirrhotic patients are up to 10 times more likely to develop bacteremia compared to general population [7,8]. Furthermore, infectious events are a major risk factor of cirrhotic decompensation mainly by a higher risk of developing ascites and kidney failure [9]. Hence these events are independent predictors of death by deeply impacting survival of cirrhotic patients irrespective of remaining liver function or cirrhotic etiology [10-13]. However, spondylodiscitis is a rare disorder which is mainly a secondary infectious site from bloodstream infection [14]. Hematogenous spondylodiscitis preferably affects the lumbar spine due to a greater vascularization compared to cervical and thoracic spine [15]. In cirrhosis, the typical sources of bloodstream infections are primary infections and spontaneous bacterial peritonitis caused by gram-negative enteric bacilli [16]. The prevalence of spondylodiscitis in the Western population is estimated at 0.4 to 2.4 per million with cirrhosis as identified risk factor (0.05% of cases), subject to an obvious lack of data [17]. Secondly, Dysbaric Osteonecrosis (DON) is a form of Decompression Illness (DCI) characterized by aseptic osteonecrosis due to hyperbaric environment exposure, conditions met in scuba diving. The physiopathology is not well established but the main hypothesis, based on Henry's and Boyle's gas laws, is the intra-tissue loss of solubility for intra-tissue dissolved gas due to ambient pressure decrease during the ascent to the surface.

Intravascular or intra-tissue bubbles will form and embolize in capillaries and cause focal ischemia, or increase the intra-tissue pressure responsible for microvascular collapse [18]. Anatomical sites preferentially affected are joints, proximal femur, and shoulders, but all can

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theoretically be affected, resulting in bone fractures or irreversible joint damage up to bone collapse requiring orthopedic surgery. The diagnosis of DON is based on radiological signs and clinical history but challenging. Indeed, radiological signs can appear only after a few years after dysbaric environment exposure. Non-invasive therapies are mainly symptomatic (analgesics) and preventive to limit the necrosis progression (exposure to dysbaric environment avoidance mainly). Those therapies are ineffective in case of advanced osteonecrosis for which the last remaining treatment option is orthopedic surgery [19-21].

Case Presentation

We report the case of a 56-year-old man with a medical history of abstinent alcoholic cirrhosis, Child-Pugh C11 MELD (Model for end stage liver) score of 21, complicated by portal hypertension and infectious events as cervico-dorsal epiduritis, urinary tract infections, repeated cellulitis for which he is on long-term antibioprophylaxis by cefalexin. He has been a scuba diving instructor for 20 years and dives weekly at shallow depths exclusively with air tanks. His symptoms began with chills and rigor while he was on holiday. This was followed by confusion characterized by spatiotemporal disorientation. Urgent medical check-up was performed concluding at ammoniacal encephalopathy without any identified etiology. He received lactulose enemas. The situation improved, allowing his repatriation to our hospital. Physical examination showed sub-icteric status and flapping tremor. Lab tests showed grade 4 thrombocytopenia ($17 \times 10^3/mm^3$ [$150\text{-}450 \times 10^3/mm^3$]), moderate inflammation level (CRP 91.6 mg/l [≤ 5 mg/l]) with stable kidney function and liver tests. Microbiological tests were performed including blood cultures, chest X-ray and urine examination showing mild leukocyturia (290/µl [<25/µl]) and significant microscopic hematuria (5848/µl [<25/µl]) with negative urine culture. Blood cultures returned positives for multi-resistant *Citrobacter freundii*. In this context with elevated PSA (5.45µg/l [<2.5 µg/l]), it was concluded to a prostatitis with secondary bacteremia. In the meanwhile, the patient developed cirrhotic encephalopathy and liver dysfunction. Intravenous antibiotic therapy by meropenem was then administered and the evolution both clinically and biologically was initially favorable.

After one week of Intra-Venous (IV) antibiotics, a relay to oral ciprofloxacin was performed according to the antibiogram. Then, the patient complained with mechanical lower back pain but responsible for nocturnal insomnia. Considering his previous history of spinal infection, a lumbar spine MRI was performed and demonstrated ischemic changes in the fifth Lumbar (L5) vertebral body MRI signal in accordance with the diagnosis of aseptic osteonecrosis complicated by spondylodiscitis (Figure 1). PET-CT was then performed which confirmed the spondylodiscitis with L5-S1 hypermetabolic infiltration of unfavorable evolution despite IV antibiotherapy characterized by an inflammatory extension to anterior paravertebral musculature. Associated endocarditis was excluded. Surgical options were ruled out due to ascites and decompensated cirrhosis that contraindicated surgery with anterior approach. Despite clinical and biological favorable evolution (Figure 1), radiological investigations showed paravertebral muscle damages. Blood culture showed persistent *Citrobacter freundii* growth despite 2-weeks of antibiotic therapy with meropenem relayed by oral ciprofloxacin. IV antibiotic therapy with meropenem was resumed for 2 additional weeks followed by a 4-weeks course of oral ciprofloxacin for a total 6 weeks of antibacterial therapy. The patient's evolution was then favorable, and the patient was discharged after stabilization. The MRI performed 4 weeks later showed significant regression of the edema of the paravertebral musculature and resolution of anterior epiduritis but with persistent signal abnormalities of L5-S1 bone medullar which tends to confirm chronic avascular bone changes.

The retained diagnosis is dysbaric osteonecrosis complicated by spondylodiscitis considering frequent exposures to dysbaric environment, imaging results and the absence of cardiovascular disease identified during the recent pre-liver transplant assessment.

Discussion

In our case, lumbar spine was the only affected site by dysbaric lesions which is an atypical location and none of the commonly affected areas were abnormal based on FDG PET-CT images. Unfortunately, the clinical condition of our patient did not allow us to perform a bone biopsy to buttress our diagnostic hypothesis. We were also unable to objectively measure the patient's risk of vertebral compression due to the lack of validated scale to evaluate the risk of bone collapse in DON. The Ficat system which evaluates the risk of articular surface collapse for avascular osteonecrosis of femoral head is by default used in the literature for hip DON without any scientific data confirming its diagnostic values for those cases. Furthermore, our patient previously benefited from osteoporosis screening by bone densitometry as part of pre liver transplant evaluation which was strictly normal. Therefore, we only recommended stopping scuba

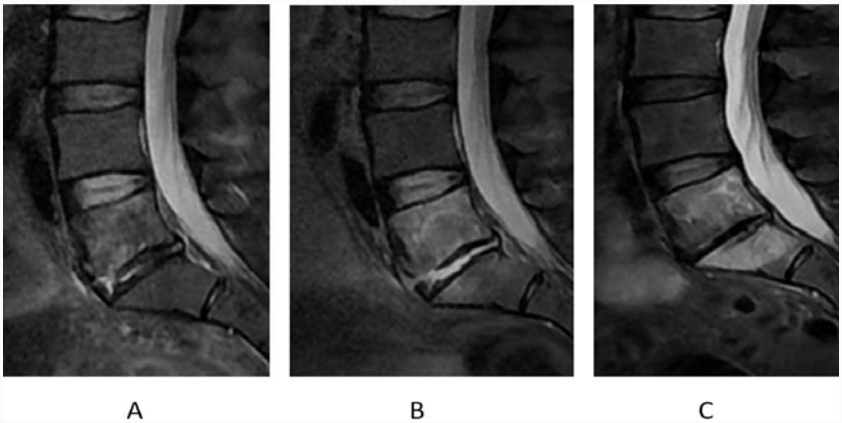

Figure 1: Lumbar MRI in sagittal plane (T2) showing necrotic changes in MRI signal of bone medullar at diagnosis (A), after 3 weeks of antibiotics (B) and 1 month after antibiotic treatment completion.

diving to avoid further bone lesions due to dysbaric environment exposure, limitations in weight bearing activities and physical therapy to reinforce paravertebral musculature. No oral bisphosphonate therapy was initiated. To our knowledge, only one similar case was published in a 54-year-old non-cirrhotic female scuba diving instructor with persistent lower back pain and secondarily abdominal pain concluding with the diagnosis of Salmonella spondylodiscitis of L2/L3 on land of DON [22].

Conclusion

This case illustrates the extreme susceptibility of cirrhotic patients to bacterial infections mainly due to global immune dysfunction, dysbiosis and the atypical potential presentation of sepsis in this specific population.

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