CASE REPORT

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Alveolar Hemorrhage, Intracranial Bleeding, and Infective Endocarditis in A Young Case of Brucellosis

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ABSTRACT

Brucellosis is a common zoonosis, caused by facultative intracellular, gram-negative rod-shaped bacteria. More than 500,000 new human cases are reported annually, and unreported cases are estimated to be 10 to 25 times more. It's a treatable disease, mortality rates are low even in untreated patients (2%). Here we present three fatal conditions due to brucellosis in a case: severe aortic valve insufficiency related to infective endocarditis, hemorrhagic cerebral embolism, and alveolar hemorrhage. In this case, brucellosis, known as a chronic, treatable disease, resulted in death weeks after the first admission to the outpatient clinic in another hospital with the first symptoms associated with infective endocarditis. We present this case as it exemplifies the urgency and severity of the timing of valve surgeries required for infective endocarditis.

Keywords: Brucellosis, infective endocarditis, alveolar hemorrhage, cerebrovascular hemorrhage, surgery timing

Introduction

The incidence of brucellosis in the world varies between 0.4-160 /100 000 population (7.99/100 000 population, Turkey 2017) (1). *Brucella spp*. infection is known to be transmitted by eating infected dairy products or un-well cooked infected meat. Also, it can survive in aerosols and resist drying, and this property caused the usage of *Brucella spp*. as a biological warfare agent (2). 10-100 microorganisms via aerosol are highly contagious. It usually infects laboratory workers and butchers, by way of airborne transmission. Pulmonary involvement of brucellosis is rare (1%) even in patients who are infected with airborne transmission (2,3). Although bronchopneumonia and bronchitis are the most characteristic findings of fetal abortion materials in animals. The bacteria can multiply in the lung tissue in postnatal *Brucella spp*. infections, and surprisingly, histopathological changes may not be seen despite the presence of *Brucella spp*. in the lung tissue (2,4). These findings suggest that the adult lung, unlike the fetal lung, serves as a niche rather than a target organ for *Brucella spp.* infection. 80% of brucellosis-related mortality is with infective endocarditis (1% of all cases) and meningitis (4% of all cases). Furthermore, primer alveolar hemorrhage in brucellosis is not an expected finding as a direct effect of infection and has been not reported before in a brucellosis case according to our literature research.

Case

A 32-year-old male patient with no comorbidities was admitted to another hospital outpatient clinic on May 20 with oedema, discolouration and tenderness in the right arm. He was admitted to the hospital with swelling and purpuric rash on his feet, cough, hemoptysis, and visual impairment symptoms. Roth spots were observed on fundus examination, and computerized tomography (CT) revealed hepatosplenomegaly and diffuse consolidations in the lung. Brucella tube agglutination test was positive on June 14th and has been treated with rifampicin, doxycycline and daptomycin. Hemoptysis and oxygen demand increased gradually during treatment, and he was referred to our hospital for bronchial artery embolization on 23 June.

Extravasation from the right lung middle lobe arteries and peripheral bronchial artery end branches was detected during bronchial artery angiography, and embolization was performed in the interventional radiology unit. The patient's vital signs suddenly deteriorated during the angiography procedure, and consciousness changes were observed. Aorta and brain CT angiograms were taken for detecting possible vascular complications. There was no vascular dissection on aortography, no bleeding, pulmonary or brain arterial embolism on CT angiography. Regarding, hepatosplenomegaly and spleen infarction were present.

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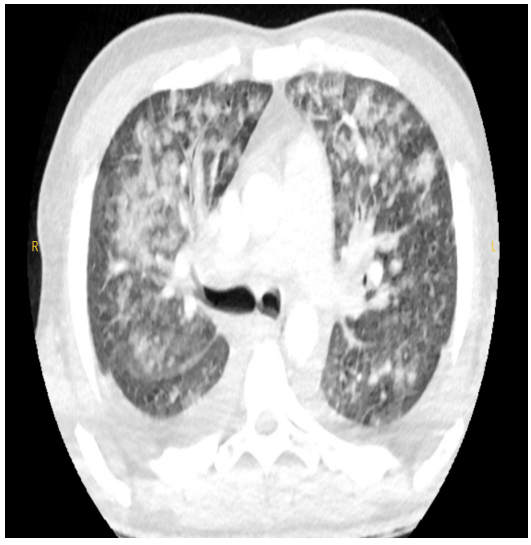

Figure 1. Day 1 thorax tomography: represent alveolar hemorrhage

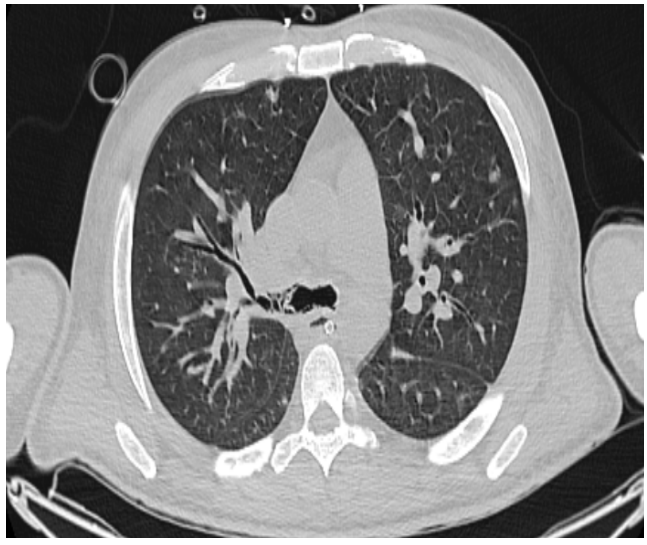

Figure 2. Day 6 thorax tomography: regressed alveolar filling, bronchial plugs

He was transferred to the intensive care unit on June 23rd. On admission, he was tachypneic, tachycardic and unconscious. His heart rate was 135 beats/min, arterial blood pressure was 163/75 mmHg, fever was 36.6 °C, respiratory rate was 40/min, and $SatO_2$ was 95% under the nasal oxygen support of 10 l/min. He was agitated and lack of orientation and cooperation. Glasgow Coma Score (GCS) was 8. Widespread rales and rhonchi were detected in the auscultation of both lungs. Murmurs were present in all cardiac regions. The abdominal examination was normal. Bilateral petechial eruptions were present in the lower extremities. At admission laboratory parameters resulted in C-reactive protein 173 mg/L (0-5), procalcitonin 0.86 µg/L (<0.16), ,LDH: 527 U/L (120-246), troponin I :122 ng/L(<45), CK-MB 1.64 µg/L (<5), D-dimer: 3.19 mg/L (< 0.55), Brucella agglutination positive at 1:5120 titer, Brucella IgM: 9.32 (>=1.1 Positive), Brucella IgG: 127.90 RU/mL (>= 22 Positive), Erythrocyte Sedimentation Rate: 44mm/hr (0-15). Other laboratory parameters were normal. Bedside echocardiography was revealed, and the aortic valve was tricuspid and destructive, 2–3 mm calcified vegetations on all cusps, 13x10 mm calcified vegetation on non-coronary cusps, severe aortic insufficiency (regurgitation jet/LVOT diameter >65%), moderate mitral insufficiency, moderate tricuspid insufficiency, pulmonary artery pressure: 52 mmHg.

The patient was intubated due to severe cognitive deterioration. Bradycardia and cardiac arrest developed during intubation, and he was successfully resuscitated. Cardiopulmonary resuscitation (CPR) time was 10 minutes. After the cardiac arrest, vasopressor or inotrope was not needed, and urine output continued.

Brain and thorax CT was performed on day 6th. Patchy areas of ischemia with hemorrhagic transformations and subarachnoid hemorrhage (SAH) were observed in bilateral cerebral, cerebellar hemispheres, and mesencephalon. It was observed in the lung the alveolar filling defects due to bleeding disappeared and a plug formed in both main bronchi (Figure 1 and 2). The plugs were removed by bronchoscopy.

He was evaluated by cardiology and cardiovascular surgery, and surgery was not considered because the ongoing active hemorrhages, low GCS, 4 weeks treatment for brucellosis had not yet been completed, and SAH was reported in brain CT scans.

He has several cardiac arrests on June 29th and 30th. At last, he was unresponsive to CPR on the 30th of June.

Discussion

The patient was a shepherd and had many cows which experienced abortus. He wanted to investigate dead calves himself, and his relatives tried to explain his effort as “he tried to autopsy the calves” and “He tried to give rescue breaths to calves”. We find it helpful to include this detail for researchers studying airborne transmission. The gold standard in the diagnosis of brucellosis is to determine the agent in blood cultures. No causative agent was detected in the blood cultures of our case, and the diagnosis was made based on clinical and serological findings.

Diffuse alveolar hemorrhage occurs histopathologically by three different mechanisms, pulmonary capillaritis, diffuse alveolar damage, and bald pulmonary hemorrhage without damage to the respiratory membrane. Its clinical response to the embolization procedure and fast recovery of lung parenchyma at CT and oxygenation, supports the idea that the respiratory membrane and its components are not affected by inflammation and the lung is not primarily affected by brucellosis. The functionality of bronchial artery embolization in bleeding control seems to be important in cases of infective endocarditis with alveolar hemorrhage in the presence of emergency valve surgery indication. Immunosuppressive therapies used in the treatment of pulmonary hemorrhage when the etiology is diffuse alveolar damage or pulmonary capillaritis carry potential risks in the presence of infection. In our case bronchial artery catheterization and embolization were performed before the etiology of massive hemoptysis was determined. The effects of embolization on bleeding control and oxygenation do not appear to be negative. No increase in oxygen demand was observed, the alveolar filling pattern disappeared,

and the major respiratory problem was related to the plugs in the airway caused by blood clots (Figure 2). Alveolar hemorrhage is reported priorly many times in aortic valve endocarditis. Brucella-related infective endocarditis and infective endocarditis-related alveolar hemorrhage are mostly seen in the left side valves. Under these conditions our thought alveolar hemorrhage is a complication of infective endocarditis but not brucellosis.

Although the first treatment option of infective endocarditis is surgery, non-operated patients mortality rate was not so high and it has been reported as 44,8% (5). Nearly %50 of infective endocarditis patients require cardiac valve surgery in the management (6,7). In a literature-based review, Keshtkar-Jahromi et al. revealed that combining antibiotic treatment with surgery reduced mortality from 36% to 6.7% (8). Similarly, Li X et al. reported that poor prognosis in a young brucella endocarditis case report (9).

Emergency valve surgery is indicated for refractory pulmonary oedema, valvular insufficiency causing cardiogenic shock, and aortic or mitral valve endocarditis causing obstruction or fistulization. It is recommended that infective endocarditis, which causes heart failure symptoms and causes severe aortic and mitral valve regurgitations that are difficult to tolerate, should be taken to surgery in the early period. In addition, uncontrollable infection, recurrent embolization, type of causative agent and vegetation that do not regress under major antibiotic therapy are indications for valve surgery. European Society of Cardiology described the timing of surgery as emergency surgery, surgery performed within 24 hours; urgent surgery, within a few days; and elective surgery, after at least 1–2 weeks of antibiotic therapy (10). However, both the American Heart Association and European Cardiology Society guidelines recommend postponing valve surgery for up to 4 weeks in the presence of intracranial haemorrhage (10,11). While the need for intraoperative and postoperative anticoagulation of the patients raises concerns about the neurological consequences of intracranial haemorrhage, high mortality rates have left the subject controversial. There are also studies suggesting that an early surgical approach is preferable despite intracranial haemorrhage (12). Results of the different approaches are needed to guide researchers about which intracranial haemorrhage would benefit from a surgical approach.

Conclusion

The case is a remarkable example of how infective endocarditis can have a catastrophic course. Regardless of the cause, delays in diagnosis and treatment may result in increased mortality and morbidity. Thromboembolic complications are unpredictable therefore indications for emergency surgery should be met before these complications develop. We hope that this case report will help to develop better decision-making methods for surgery if indicated or not for infective endocarditis.

AUTHOR CONTRIBUTIONS:

Concept: HDM, ND; **Design:** HDM, ND; **Supervision:** HDM, ND; **Materials:** BB; **Data Collection and/or Processing:** HDM, ND; **Analysis and/or Interpretation**: HDM, ND; **Literature Search:** HDM, ND; **Writing Manuscript**: HDM, ND; **Critical Review:** HDM, ND.

Informed Consent: Subject of paper is a case report. A first degree relative was informed about the content and purpose of the article, he was given a phone number to contact, his questions were answered, and his informed consent was obtained.

Informed Consent: Written and verbal consent was obtained from the patient's father, Hüseyin Çaycı.

Peer-review: Externally peer-reviewed.

Conflict of Interest: Authors have no conflicts of interest to declare.

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