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A case report of isolated thrombocytopenia induced by brucellosis

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ABSTRACT

Brucellosis may cause a vast variety of hematologic manifestations, as one of the major infectious diseases in developing countries. Thrombocytopenia is among the very rare presentations, especially when it is the only manifestation. This report was aimed to show that how an isolated thrombocytopenia can lead to brucellosis diagnosis. Hereby, we reported a young woman with a history of recent diagnosed brucellosis who admitted to the hospital with purpuric rashes with severe thrombocytopenia that was first suspected to have idiopathic thrombocytopenic purpura. As one of the most important diseases in endemic areas such as Iran, it is important to rule out brucellosis induced thrombocytopenia in the patients which presented with thrombocytopenia along with some other differential diagnosis of isolated thrombocytopenia such as idiopathic thrombocytopenic purpura.

1. Introduction

Brucellae is mostly found in domestic animals such as sheep, goats, swine and cattle which is endemic in developing countries and even in developed countries with rural population superiority. It can transfer to human by 3 main ways including direct contact of damaged skin with contaminated patients' aborted fetuses, placenta and amniotic fluid, by overpassing mucosal surfaces and the last one is through airway[1,2]. Among the various clinical manifestations, hematologic complications include anemia, leucopenia and thrombocytopenia, pancytopenia, disseminated intravascular coagulation and purpuric thrombocytopenia. Although neutropenia has been reported only as case reports, it is another member of hematologic manifestations. Anemia is the most common presentation of patients with brucellosis while purpuric thrombocytopenia is the rarest which has been reported in only 2.6% of active brucellosis cases[3,4,5].

After autoimmune phenomenon in which thrombocytopenia is the main dominant manifestation and any other underlying causes were ruled out, immune thrombocytopenia is suspected[6]. Immune and non-immune drug-induced-thrombocytopenia (DITP) are among the conditions which should be first ruled out and may cause thrombocytopenia through drug-dependent antibody-mediated

platelet destruction and bone marrow suppression[7]. The list of drugs which may cause DITP are listed in the systemic review by Arnold *et al.*[6].

2. Case report

A 23-years-old young woman was admitted to our hospital with chief complaining of generalized purpuric and petechial rashes which had an itchy essence. Aside from the history of brucellosis which was diagnosed about 10 days before admission, she was a healthy individual. She had true vertigo along with vomiting. She also complained about few episodes of epistaxis. When we asked her about experiencing melena, hematemesis or gingival bleeding, she did not give a history of any. All these symptoms have started 1 week ago, more precisely a week after the beginning of using her anti-brucellosis drugs. When asked in detail, she revealed the medical history of co-trimoxazole and gentamycin in the last week. She had not taken any over the counter drugs, not even any pain killers or any other non-steroidal antiinflammatory drugs.

On examination, she was neither tachypneic nor tachycardia. Her blood pressure was 100/60 mmHg and was not febrile. Her heart and breath sounds were normal without any pathologic findings. She had diffused purpuric rashes on her upper and lower limbs and also on her trunk and her back. She said all the rashes occurred in one night and her whole body was involved with them in an itchy nature which made her recourse to the hospital. The rashes were non-bleaching and plantar surfaces were spared. On her buttocks, the injection site for gentamycin was ecchymotic and non-tender in touch.

To evaluate the cause of her epistaxis and the diffuse purpuric rashes which we first suspected them to be petechial purpuric

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rashes, we asked for a complete blood count with coagulation factors. To our surprise, all the asked tests were normal apart from the platelet count. She had a severe thrombocytopenia (Plt = 3000). She was not anemic or leukopenic. Her prothrombin time, partial thromboplastin time and international normalized ratio were all normal. To confirm her thrombocytopenia, we took a peripheral blood sample which revealed hypo-chrome microcytic red blood counts with anisocytosis and granulated neutrophils with vacuole formation (toxic neutrophil). No platelets were detected. Her liver function tests, aspartate aminotransferase, alanine aminotransferase and alkaline phosphatase were all normal. When true thrombocytopenia was confirmed, we discontinued anti-brucellosis drugs and infused 6 units of platelets in 2 days and dexamethasone of 4 mg was given twice a day. However, there was no change in the platelet count, not even a slight increase. On the 5th day, hematologic consolation was made, in which they asked for bone marrow biopsy and aspiration. On that day, because of the discontinuation of antibiotics, an infection consolation was also made. The specialist suspected the drug-induced thrombocytopenia through his history taking of the disease and recommended the initiation of rifampin and doxycycline (two anti-brucellosis agents). He asked for abdominal sonography to check any possible organomegaly (such as splenomegaly) and her buttock sonography to rule out any collection or abscess formation. The results were normal.

At the 2nd day after new antibiotic therapy, platelet counts started to rise. On the 9th day, her platelet count was 130000 and she was discharged from hospital on tablet prednisolone of 25 mg daily, tablet rifampin of 600 mg daily and capsule doxycycline of 100 mg twice a day for 1 month. She was asked to visit the infectious and hematologic specialist with the result of her bone marrow biopsy and aspiration and for tapering the prednisolone dosage.

After one month when she was followed, she was alright and her bone marrow biopsy and bone marrow aspiration were normal and her platelet count had reached 246000.

3. Discussion

Brucellosis is known to be a multi-organ involving infectious disease which has a wide range of clinical presentation and a wide range of complications, while some are rare such as epididymo-orchitis and some are more prevalent such as arthritis and back pain[8]. Thrombocytopenia occurs mainly in combination with other hematologic manifestations and includes 1%–20% of cases; an isolated form of thrombocytopenia rarely has been reported. Causes of thrombocytopenia are not clear yet but there are some conjectures about them which are hypersplenism, disseminated intravascular coagulation, bone marrow suppression, hemophagocytosis and immune destruction of platelets[2,5,9,10]. It is documented that an immune response is activated in the course of brucellosis which alternatively may cause an autoimmune hemolysis and destruction of platelets. A series of actions that may finally lead to the hemorrhagic purpura have been detected in some cases[9,11,12]. This purpuric thrombocytopenia is reported to be severe enough to even cause mucosal bleeding, epistaxis, gingival bleeding and even hematuria[5]. Other etiology which is important enough to be kept in mind, especially in our case, is the drug-induced thrombocytopenia. Due to it is as uncommon as the brucellosis-induced-thrombocytopenia, finding an exact etiology behind the platelet destruction in these patients is a challenging problem. If drug-induced thrombocytopenia is suspected, it may occur 5 to 10 days after the exposure to the drug, when sensitization is produced[6]. As in our case, sulfonamides were also suspected. According to the underlying etiology of thrombocytopenia, the treatment may be different. If DITP is suspected, the best way is to discontinue the suspected drug. If brucellosis is suspected, the best medical plan is a full course of anti-brucellosis antibiotic therapy. If the immune reactions are suspected,

adding a corticosteroid such as prednisolone may bring the best possible results[12]. We presented a known case of brucellosis which had severe isolated thrombocytopenia. First, we suspected DITP and so we discontinued the drug co-trimoxazole and infused platelet units. However, no change in the platelet count was detected. So we suspected we misdiagnosed idiopathic thrombocytopenic purpura and asked for a bone marrow biopsy and aspiration. Meanwhile, a consultation with infectious ward was made. To our surprise, a new regimen of anti-brucellosis antibiotic therapy caused a dramatic increase in platelet counts.

In conclusion, it is important to consider brucellosis-induced-thrombocytopenia in patients presenting with an isolated decrease in platelet count. Although it is rare but if be diagnosed properly, it can lead to a dramatic response to the treatment. This subject is most recommended for endemic areas such as Iran. We recommend for doing more evaluations for brucellosis in patients that have thrombocytopenia and especially history of brucellosis, even when they use other drugs which are suspected as the cause.

Conflict of interest statement

We declare that we have no conflict of interest.

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