Letters and correspondence submitted for possible publication must be identified as such. Text length must not exceed 500 words and five bibliographic references. A single concise figure or table may be included if it is essential to support the communication. Letters not typed double-spaced will not be considered for publication. Letters not meeting these specifications will not be returned to authors. Letters to the Editor are utilized to communicate a single novel observation or finding. Correspondence is to be used to supplement or constructively comment on the contents of a publication in the journal and cannot exceed the restrictions for Letters to the Editor. The Editor reserves the right to shorten text, delete objectional comments, and make other changes to comply with the style of the journal. Permission for publication must be appended as a postscript. Submissions must be sent to Paul Chervenick, M.D., Editor of Brief Reports/Letters to Editors, American Journal of Hematology, H. Lee Moffitt Cancer Center, University of South Florida, 12902 Magnolia Drive, Tampa, FL 33612 to permit rapid consideration for publication.

Immune-Mediated Thrombocytopenia Associated With Valley Fever

To the Editor: Platelets accelerate fungal death in vitro by cell wall damage, allowing increased neutrophil killing, and, perhaps, account for thrombocytopenia seen with fungal infections. We describe an unusual case of immune mediated thrombocytopenia in the setting of a coccidioides pulmonary reactivation.

Immune mediated thrombocytopenia is frequently seen after a viral or bacterial infection [1]. Antigens on infectious organisms may mimic platelet antigens, eliciting antibodies that target both infectious or granisms and platelets. Platelets then undergo destruction by splenic macrophages. Conventional therapies for immune mediated thrombo cytopenia have included steroids, intravenous immunoglobulin, and splenectomy [2]. Although fungal induced immune mediated thrombocytopenia is rarely seen, cases of histoplasmosis induced thrombo cytopenia have been reported as early as the late 1970s [3,4]. We present the first reported case of immune mediated thrombocytopenia associated with a coccidiodes infection.

A healthy 17 year old woman presented to the general medicine clinic for evaluation of sparse menses. Her primary care physician obtained routine laboratory tests, and found a platelet count of 70,000/μL. She also had a mild leukocytosis with a left shift. The white blood count was 12,000/μL with 60% neutrophils, 10% bands, 26% lymphocytes, and 3% eosinophils. Her hemoglobin was 16.2 g/dL, hematocrit 48.8%, MCV 92.5 fl.

Four days later, her platelet count was 60,000/μL. She did not have easy bruising or bleeding. However, she had been experiencing symptoms of an upper respiratory infection for 2 weeks. Her symptoms included voice loss, rhinorrhea, nasal congestion, and a deep guttural cough. She had chills and night sweats and required Nyquil for sleep. She recalled a similar illness a month prior, which did not resolve with a course of azithromycin.

The patient was born and raised in Wyoming. She lived in Arizona between the ages of 9 and 12. She had no other contributing past medical history. Her mother was diagnosed with idiopathic thrombo cytopenic purpura when evaluated for easy bruising during her late 20s.

Her physical examination revealed a healthy, lean, young woman with no evidence of bruising, hemorrhages, lymphadenopathy, or gamonogely, or skin lesions. Her peripheral blood film revealed large platelets and occasional atypical lymphocytes. There were abundant neutrophils with normal morphology. A bone marrow aspirate revealed a normocellular marrow with trilineage hematopoiesis and increased megakaryocytes.

A diagnosis of immune mediated thrombocytopenia was made, and the patient was placed on 1 mg/kg of prednisone. Two weeks later, her platelet count had increased to 120,000/μL. She felt well enough to compete in a track meet. After running, she had an episode of hemoptysis. In retrospect, on additional questioning, she had experienced hemoptysis associated with upper respiratory symptoms for the prior 9 months. A chest radiograph confirmed a suspicious shadow in the left upper lobe, which appeared cavitary by thoracic CT scan. Broncho scopy revealed evidence of reactive coccidiodes by culture. Her steroid therapy was rapidly tapered, and she was started on itraconazole for her fungal infection. She has maintained a platelet count of 100,000/μL off of prednisone.

There is no consensus for a specific target platelet count to guide the therapy of immune mediated thrombocytopenia. Generally, most consider treatment necessary when the platelet count drops below 30,000/μL [2]. Although the history of the described case suggested a familial component to the thrombocytopenia, the decreasing peripheral platelet count in the setting of elevated numbers of bone marrow megakaryocytes prompted treatment of immune thrombocytopenia. One could argue that immunosuppression from steroids reactivated a dormant coccidiodes infection. However, her symptoms of hemoptysis had pre ceded the incidental discovery of thrombocytopenia by nine months.

An interesting theory for thrombocytopenia associated with infection is suggested by studies by Yeaman and Robert. Platelets release a cationic protein, known as platelet microbial protein, which has been observed to be cytotoxic to Staphylococcus aureus, Candida albicans, and parasitic organisms in vitro [5–7]. Christin et al. also observed an increased Aspergillus fumigatus killing by neutrophils when platelets were activated [8]. It would seem plausible, then, that platelet activation with release of platelet microbial protein may also be cytotoxic to Coccidioides immitis. This intriguing theory may, in part, explain continued platelet recovery once the pulmonary infection was properly treated with anti fungal therapy.

Coccidioides pulmonary infection is an unusual diagnosis in the state of Wyoming. However, the patient did have a history of living in Arizona. We propose that a reactivation of her coccidioides pulmonary infection, manifested as intermittent hemoptysis, not only contributed to a baseline thrombocytopenia but also induced an immune mediated component of thrombocytopenia. This latter immune component was responsive to steroid treatment.

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