CLINICAL VIGNETTE

Infection and Thrombocytosis: What to Do Now?

Sarah Ahmed, MD and Michael Rotblatt, MD, PharmD

Case Presentation

A 60-year-old male gardener without any known past medical history presented to the emergency department with right hand redness, pain, and swelling that began 3 days prior.

The patient reported potting small trees in wooden boxes and gradually began feeling pain in his right palm, then noticed mild redness of the palm. He did not note any obvious injury, cut, or insect bite to the hand. Over the next 3 days, the pain and redness spread to the fingertips and distal forearm, followed by diffuse swelling, with subjective fevers and chills.

Upon arrival to the ED, the patient was febrile to 38.6 C, tachycardic to 107, and hypertensive to 175/106, with remaining vitals within normal limits. Physical exam was notable for gingival bleeding, right upper extremity erythema extending from mid-forearm to hand, and black discoloration of fingers with dusky, cool fingertips but intact sensation. In addition, a 1 cm linear wound was observed at the distal palm, with diffuse swelling throughout the palm and digits; no fluctuance or crepitus was appreciated. Labs were notable for WBC 21.9, Plt 77, INR 2.08, Fibrinogen 124, Na 126, CRP 256.2, Lactate 2.6, CK 865. A CT scan of the right upper extremity visualized extensive edema along the subcutaneous soft tissues. The patient was admitted to the ICU for further management of disseminated intravascular coagulation (DIC) in the setting of sepsis. Plastic Surgery was consulted and made a palmar incision without expressed purulence. The patient received broad spectrum antibiotics, fluid resuscitation, and supportive transfusions.

The following day, the patient had significant pain out of proportion to passive movement of digits, and was taken urgently to the OR due to concern for compartment syndrome, which was confirmed intra-operatively. Right hand and forearm fasciotomies were performed, and cultures were sent. Two of two blood cultures returned positive for Group A streptococcus (GAS), and the wound culture also grew GAS. The Infectious Disease service was consulted with concern for necrotizing skin and soft tissue infection (SSTI), and recommended narrowing antibiotics to IV penicillin G, with intravenous immune globulin (IVIG) for possible concurrent toxic shock syndrome. The patient clinically improved and was transferred to the medicine floor.

Despite continued clinical improvement of the right upper extremity exam on IV penicillin, labs were notable for worsening leukocytosis, shortly followed by worsening thrombocytosis, both of which significantly delayed the patient's discharge. WBC then peaked, while the thrombocytosis continued to increase for several more days, reaching above 600 from a baseline in the low 100s. Out of concern for the significant thrombocytosis suggesting continued severe or worsening infection, a repeat CT was obtained which did not demonstrate worsening infection, but instead showed thrombosis of the cephalic and basilic veins. This prompted discussion of the possibility of thrombocytosis resulting from extensive superficial vein thrombophlebitis, rather than as a sign of worsening infection. Given the possibility of the thrombosis causing impaired blood flow, which could impair wound healing, the patient was also started on rivaroxaban for a limited time. The patient's lab values improved, and at the time of the patient's discharge, the IV penicillin G was switched to oral penicillin, with follow-up in Infectious Disease and Plastic Surgery clinics.

Discussion

Reactive thrombocytosis is known to occur in the setting of acute infection, as well as in response to trauma or surgery. The mechanism of thrombocytosis due to infection is thought to be related to the increased production of cytokines (such as interleukin-6 [IL-6]) stimulating megakaryocyte proliferation.[1] While the exact prevalence and timing of secondary thrombocytosis due to infection is not clear, the limited literature does contain useful information .A retrospective review of 801 cases of thrombocytosis defined as a platelet count >500, found infectious causes in nearly half the cases of secondary thrombocytosis.[2] These patients also had more rapid normalization of platelet count than those with non-infectious causes, highlighting the acute nature of infection and its resolution with appropriate management. As it applies to our case, the delayed normalization of the platelet count could potentially be a clue in support of a non-infectious cause (such as a superficial thrombosis), especially given the patient's clinical improvement during the continued platelet rise. One retrospective study of 242 trauma patients showed a correlation between illness severity using the injury severity score (ISS) and delayed thrombocytosis, suggestive of delayed thrombocytosis as a marker of worse outcomes.[3] In a smaller study of 50 ICU patients who developed thrombocytosis, the peak platelet count occurred on average more than 2 weeks after the onset of the stressor, and thrombocytosis was more common among patients with surgical rather than medical stressors.[4] It should be noted

that this patient initially presented with a significantly lower platelet count while clearly having an active infection, which further suggests that the delayed onset of thrombocytosis was due to an alternative cause. We propose that clinicians use clinical parameters such as pain, tenderness, erythema and vital signs, rather than thrombocytosis to measure the progression of these types of infections, and to further investigate thrombocytosis that does not fit the clinical picture. Additional studies are needed to compare the relationship between the timing of leukocytosis resolution vs. thrombocytosis resolution, as this may provide more objective data to help evaluate the progression of infections.

REFERENCES

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