Introduction

Platelet abnormalities have been observed in a variety of infections caused by bacteria, fungi, parasites and viruses. Most often, thrombocytopenia is observed particularly in systemic viral, bacterial or malarial infection (1, 2). Thrombocytosis, defined as symptomless, transient and with a moderate elevation of the platelet count in response to an underlying disease, is rarely observed in infection.

Anemia, thrombocytopenia and DIC occurring with infection have been well described. However, thrombocytosis in response to infection is rarely appreciated or reported in the medical literature. We are describing a patient with an acute prosthetic joint infection, bacteremia and osteomyelitis who had thrombocytosis related to this infection.

Case Report

Patient: A 57-year-old housewife presented with right hip pain and a low grade temperature of eight-days' duration seven years after the insertion of a total right hip prosthesis for degenerative joint disease. Her past history was significant for its dental manipulation without antimicrobial prophylaxis. Physical examination revealed a well-nourished female in no distress and with a temperature of 100°F, BP 120/80 mmHg and a pulse rate of 80/min. A non-radiating ejection systolic murmur was heard at the aortic area. Examination of the right hip demonstrated increased local heat, some edema and no erythema. Tenderness was elicited over the posterior aspect of the joint. Otherwise the examination was normal. Laboratory studies revealed hemoglobin of 10 g/l, a white cell count of 6,400/mm³ with 79% neutrophils and 21% lymphocytes. The platelet count, measured with a Coulter Counter S Plus II, was 518,000/mm³. The erythrocyte sedimentation rate was 110 mm/h. Serum alkaline phosphatase was 708 IU, SGOT 297 U/ml, LDH 279 IU, SGPT 230 U/ml, bilirubin 0.05 mg/100 ml, gamma glutamyl transferase (GGT) 880 U/ml, serum iron 55 μg/100 ml and iron binding capacity 286 μg/100 ml.

Clinical methods: The rheumatoid factor and the hepatitis profile were negative, and a two-dimensional echocardiogram did not reveal any abnormalities. Two of five blood cultures grew gram-positive cocci in pairs, which were identified as group B beta hemolytic streptococcus sensitive to penicillin. An arthrogram done two days after admission revealed no dehiscence in the right hip structures. Culture of the synovial fluid from the arthrogram revealed the same group B beta hemolytic streptococcus. A gallium scan was compatible with osteomyelitis of the right hip. After isolation of the organisms, the patient was started on aqueous penicillin G in a dose of two million units every four hours intravenously. The organism had an MIC of 0.06 mg/l for penicillin. The initial serum bactericidal concentration was 1/1. A repeat platelet count was very elevated with a value over a million (1,378,000/mm³), while the sedimentation rate remained very elevated (Figure 1). Gentamicin was then added on the eleventh hospital day, at a dose of 30 mg i.v. every six hours, and the aqueous penicillin G was increased to 18 million U/day. On the fourteenth hospital day the bactericidal level in serum was 1/128, and 1/32 on the twenty-ninth day. The patient’s platelet count normalized as the patient felt clinically better and remained afebrile (Figure 1).

Discussion

This case of an infected total hip prosthesis with osteomyelitis and bacteremia due to group B beta hemolytic streptococcus afforded us the opportunity of studying a variety of responses to an acute infection. In osteomyelitis, the erythrocyte sedimentation rate is usually elevated and may remain so at the end of adequate therapy, especially when the adjacent joint is involved (3, 4). This nonspecific laboratory test has been used in determining the activity of infection and its response to therapy. The patient described had an elevated sedimentation rate which increased until adequate therapy was used and was still abnormal on completion of therapy because of the adjacent joint involvement. As demonstrated in Figure 1, the parameters suggesting that the patient was responding to therapy included the serum bactericidal concentration and the platelet count. When the bactericidal level was

Received: 26 July 1984/Accepted: 19 September 1984
Asst. Prof. C. Robey, M.D., Department of Medicine, Saint Barnabas Medical Center, Livingston, New Jersey 07039, U.S.A.;
Asst. Prof. H. Chmel, M.D., Infectious Diseases Section, Department of Medicine, Saint Barnabas Medical Center, Livingston, New Jersey 07039, U.S.A.
Figure 1: The figure describes the clinical course of a patient with a prosthetic joint infection. The platelet count continued to increase until appropriate antibiotic therapy was instituted, as shown by an increased serum bactericidal concentration (BC) and a decreased platelet count.

Adequate, the platelet count decreased and eventually returned to normal. In contrast, the erythrocyte sedimentation rate did not parallel the platelet count and took a longer time to return to normal, although the patient was clinically much improved and afebrile.

Thrombocytosis has numerous etiologies (2, 5–8). An elevated platelet count can be found after a recent major hemorrhage; it can occur after major surgery, including splenectomy; it is associated with iron-deficiency anemia, inflammatory bowel disease, collagen vascular disease, alcoholism and cirrhosis, malignancy and infection. Elevated platelet counts have been observed and occasionally reported in both acute and chronic infection.

Murachanin et al. (1) described 42 of 675 patients in whom a platelet count was performed and who had thrombocytosis due either to an acute or to a chronic infection. Interestingly, during the first few hospital days the platelet count was low or normal but increased by Day 3 and returned to normal during convalescence. Only one patient had osteomyelitis. In Selroos' (2) series, thrombocytosis was observed in 17 patients in whom infection was the primary illness. Acute bacterial pneumonia was the most frequent illness, and one patient with osteomyelitis was described. Selroos (9) also described a patient with amyloidosis and thrombocytosis secondary to the osteomyelitis and its activity. More recently, Gonzenback et al. (7) claimed inflammatory causes (31% of 67 patients) to be the principal etiology of thrombocytosis in their study.

Acute viral or bacterial infections associated with thrombocytosis include tuberculosis (1, 2, 5, 7), atypical mycobacteriosis (10), rheumatic fever (11), actinomycosis (12), Whipple's disease (13), brucellosis (7), malaria (7), sacral osteomyelitis (14) and mucocutaneous lymph node syndrome (15). Infantile cortical hyperostosis, an entity of presumed viral etiology, is also accompanied by a marked rise in platelet count (16). In contrast to the other etiologies, the platelet count elevation reported in infections are only slight. In retrospect, however, in the patient described in this report, the rise in platelet count was moderate and a very useful guide to therapy. Contrary to the observed thrombocytopenia in bacteremic patients (17), our patient demonstrated an elevated platelet count. The explanation for the thrombocytosis accompanying such an infection is unknown. During infection, thrombocytosis may reflect an increase in the rate of platelet production rather than a prolongation of platelet life-span.

Although thrombocytosis is sporadically reported in patients with osteomyelitis, it has received little attention in

C. Robey, H. Chmel: Thrombocytosis in Acute Osteomyelitis