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INTRODUCTION

Thrombocytosis is quite often discovered as an incidental finding during the laboratory evaluation of an unrelated medical problem. However, when it is detected, it constitutes an important diagnostic challenge. Thrombocytosis can either be due to a reactive process (secondary thrombocytosis) or it can be due to a clonal bone marrow disorder (primary thrombocytosis or clonal thrombocytosis). It is often exceedingly difficult to differentiate between the reactive and clonal types of thrombocytosis on the basis of clinical findings or laboratory test results. Yet there are fundamental differences between them in terms of cause, pathophysiological features, and clinical implications. Here we report a case of reactive thrombocytosis with an unusually high platelet count.

CASE REPORT:

A 26 yr old lady presented to us with history of fever, cough, chest pain, breathlessness of one week duration along with anorexia and weight loss of one month. Patient did not have any other symptoms. General survey revealed that she was conscious, oriented and had a temperature of 102 degree Fahrenheit. Respiratory rate was 33/min, BP was 130/80 mmHg, PR was 116/min she was pale and had no significant lymphadenopathy. Respiratory system revealed diminished breath sound in the right hemithorax with severe intercostal space tenderness. Cardiovascular, Abdomen and neurological system examination were normal.

Chest X ray taken revealed a moderate right sided pleural effusion (Fig 1).

USG guided aspiration revealed the fluid to be frank pus and an ICD was inserted subsequently. Investigations showed TLC of 18,600 (P - 88, L - 10, M - 2), haemoglobin 5.4 gms%, platelets 22.9 lakhs/cu mm, ESR > 150mm/hr, RFT, electrolytes, LFT and urine examination were normal. USG abdomen – normal study. Pleural fluid showed WBC-11000, RBC- 6000 and cultures grew Staphylococcus aureus. Manual platelet counting done revealed it to be 21.6 lakhs/cu mm. Peripheral smear showed evidence of microcytic, hypochromic anemia along with an MCV of 58. Bone marrow aspiration and biopsy done (in view of very high platelet counts) showed a reactive marrow and no evidence of a primary hematological problem.

A diagnosis of empyema with iron deficiency anemia was made along with reactive thrombocytosis. She was treated with IV cefaperazone and sulbactum, iron supplements, blood transfusions and other supportive measures. Serial monitoring of blood indices revealed a steady fall in the platelet count as the infection and anemia improved, thereby underlining the cause of the severe thrombocytosis as a combination of severe iron deficiency anemia along with a severe purulent infection in the form of an empyema. Serial blood indices are given in Fig 2 A and Fig 2 B.

ABSTRACT

Thrombocytosis is a laboratory abnormality which may be encountered on routine evaluation of an unrelated medical problem. Thrombocytosis can either be due to a reactive process (secondary thrombocytosis) or it can be due to a clonal bone marrow disorder (primary thrombocytosis or clonal thrombocytosis). It is often exceedingly difficult to differentiate between the reactive and clonal types of thrombocytosis on the basis of clinical findings or laboratory test results. Yet there are fundamental differences between them in terms of cause, pathophysiological features, and clinical implications. Here we report a case of reactive thrombocytosis with unusually high platelet count, who did not have evidence of a primary hematological problem and was identified to have two co-existing pathologies which led to the elevated platelet count.

Key words: thrombocytosis, empyema, anemia, case reports.
DISCUSSION:

The most striking feature of this case is the gross elevation in the platelet count. The degree of elevation of platelet count in this patient was such that further work up for a primary hematological problem was warranted. However, further investigations in that direction did not reveal any evidence of a primary hematological disorder. This patient had a severe infection (empyema) and severe iron deficiency anemia which are well established causes of reactive thrombocytosis. The combination of these two illnesses may well have caused a dramatic increase in platelet count.

The cause of reactive thrombocytosis has been ascribed to cytokine mediated increased synthesis of thrombopoietin, which in turn leads to increased synthesis of thrombocytes. The various causes of reactive thrombocytosis are given in Table 1.

We report this case to highlight the following current concepts in reactive thrombocytosis: Firstly, the degree of elevation of platelet count is not an indicator whether the thrombocytosis is primary or secondary. Secondly, the management of secondary thrombocytosis is purely directed towards the inciting cause which in our patient was due to iron deficiency anemia and empyema.

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REFERENCES: