

Short Communication

Demographic Profile, Etiology of Reactive Thrombocytosis and Its Association with Anemia in North East India

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Abstract

Reactive thrombocytosis is often observed in response to iron deficiency anemia, acute infections, chronic inflammatory diseases including rheumatoid arthritis, hemorrhage and sometimes in cancer. Sixty - eight non-consecutive cases of reactive thrombocytosis that visited a secondary care hospital in North East India were studied to analyse the demographic profile, aetiology and association with anemia which is very common in this population.

Keywords: Reactive thrombocytosis; anemia; North East India; demography

Introduction

This retrospective study was carried out in a secondary care centre in North -East India to study the etiology, demographic profile, relationship with anemia of patients identified to have reactive thrombocytosis (Platelet count > 500,000/ μ l) and whether it could be used as a marker of inflammation in absence of neutrophilia.

Methods

Two milliliters of EDTA blood was collected through a clean venipuncture from 68 non - consecutive. The samples were tested twice; in a Sysmex XS 800 I Haematology Analyser followed by in Sysmex KX21 3 part Haematology Cell analyser and correlated on peripheral blood smear.

Results

There was a slight male preponderance (n=36 - 52.9%). Mean age of the patients was 16.2 years (range 3 months -80 years) of which there were 50 children (73.5%). Platelet counts ranged from 507,00 - 1,290,000/ μ l with mean of 696,000/ μ l. Seven patients (10.2%) had extreme thrombocytosis (counts > 1,000,000/ μ l) as per the classification used by Chirello. et al [1]. Fifty-three (77.9%) patients were anemic (Haemoglobin range 3.4 -13.2 gm/deciliter (gm/dl) (normal > 11 gm/dl. Leukocytosis was present in 44 (64.7%) patients. Mean Total Leukocyte Count (TLC) was 19, 800/ μ l with range 5900 -52,600/ μ l. Neutrophilia was seen in 42 (61.8%), lymphocytosis in seven (10.3%) and more than one cell type in four patients. Aetiological classification of patients is shown in (Table 1).

Follow-up

One patient with metabolic encephalopathy died within a day of admission, three patients were referred and two left against medical advice. The remaining patients were treated in the hospital and have recovered.

Discussion

This study included a wide spectrum of cases which were found

Table 1: Causes of Reactive Thrombocytosis in 68 patients.

Respiratory tract infections	22
Pneumonia	3
Chronic Obstructive Pulmonary Disease	1
Empyema	1
Chronic Kidney disease	3
Nephrotic Syndrome	2
Acute Glomerulonephritis	2
Acute Renal failure	1
Acute gastroenteritis	5
Acute gastritis	1
Necrotizing pancreatitis	1
Lymphadenitis (mesenteric and supraclavicular)	2
Polyarthritis (1 juvenile)	2
Tuberculosis	
(spine -3, military 1, lung 1, intestine 1)	6
Microcytic Hypochromic Anemia	2
Sepsis	2
Abscess	2
Orthopedics (2 fractures, 1 hip dislocation)	3
Developmental delay	2
Incomplete abortion	1
Carcinoma Rectum	1
Organophosphorus poisoning	1
Drowning	1
Kernicterus	1

to have Reactive Thrombocytosis (RT). There was no correlation between severity of thrombocytosis and anemia as has been described by some authors [2,3]. Extreme RT has been reported in less than 2-3 % of patients [4]. All seven patients with extreme RT in this study had an infectious cause. None of the patients had a complication related

to thrombocytosis and all improved on treatment of underlying cause. Yohnan et al. in a study involving 663 patients detected hyper RT (counts >1,000,000/ μ l) in 2% of the children [5]. In this study four children (8%) had platelet counts exceeding 1,000,000/ μ l; higher frequency could be attributed to the small population studied and selection. Fifty –three patients had anemia along with thrombocytosis and many of them also had a focus of infection. In a study by Subramaniam et al. Anemia with coexistent infection was the commonest cause of thrombocytosis as was observed in this study also [3]. Possible mechanisms of thrombocytosis include raised levels of cytokines, such as Interleukin (IL)-6, IL-1, and Tumor Necrosis Factor (TNF), have been shown to promote *in vivo* and *in vitro* megakaryocytopoiesis [6]. However, thrombopoietin is the principal regulator of megakaryocytopoiesis [7-9]. Eleven patients with RT, of which six were anemic had normal leukocyte counts. In these patients RT was a marker for infection /inflammation. Another significant finding was that nearly a – third of the patients which included six adults and 19 children with fever had thrombocytosis as the sole abnormality. It is therefore suggested that thrombocytosis alone could be an important marker of inflammation.

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