Thrombocytosis in Iron Deficiency Anemia - Scenario in a Developing Country

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Background: In India, iron deficiency anemia (IDA) contributes to a major disease burden. Association of iron deficiency with thrombosis is increasingly being recognized. Erythropoietin (EPO) has structural similarity with thrombopoietin, which has been incriminated to cause thrombocytosis in IDA by some researchers, while others have refuted this. Therefore, actual pathogenesis is still largely unexplained. Moreover the course of thrombocytosis while on treatment with iron is not fully elucidated. As most of the studies on thrombocytosis are from developed countries, where nutritional anemia is less prevalent, these studies do not truly reflect the proportion of cases of thrombocytosis due to underlying iron deficient state. Hence the present study was planned.

Objectives: To study abnormalities of platelet count in Iron Deficiency Anemia and to relate the severity of thrombocytosis with severity of anemia and its association with EPO level and changes of platelet count with treatment of anemia.

Methods: Prospective observational study comprising of 200 children below 18 years confirmed to have IDA based on peripheral smear, serum ferritin, total iron binding capacity and serum iron level. The severity of thrombocytosis was correlated with erythropoietin level and blood counts were followed up while on iron therapy.

Results: Thrombocytosis was noted in 24.5%. In 75.5% thrombocytosis was mild. Platelet count had negative correlation with haemoglobin. EPO was elevated in 67.35% of thrombocytosis. EPO showed negative correlation with Hemoglobin and Ferritin and positive correlation with platelet. However, these were statistically non-significant. Iron therapy normalized platelet count in 92% within one month.

Conclusion: Nearly One-fourth (24.5%) of children had thrombocytosis and majority was of mild or moderate degree. Platelet count was inversely related to Hemoglobin and ferritin level. EPO was increased in two-third cases of thrombocytosis and showed positive correlation with platelet count. No thrombotic complications were noted.
Hematological, Biochemical and Molecular Protocol for Heterozygous β-Thalassemia

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Background: β-thalassemia syndromes are heterogeneous hereditary anemia’s characterized by a reduced output of β-globin chains. Heterozygous β-thalassemia represents the mild form responsible for keeping the “defective” gene (thalassemic trait) in a territory.

Objective: To elaborate a hematological, biochemical and molecular protocol for heterozygous β-thalassemia for a faster diagnosis in countries with a low or median incidence of the disease.

Methods: A multicentric study was performed to evaluate the hematological, biochemical and molecular features of heterozygous β-thalassemia. The study was conducted in three Tertiary Pediatric Clinics, between 2002-2012. We used the standard blood test (hemoglobin, erythrocytes number, erythrocytes indices, reticulocytes, blood smear, serum iron level, ferritin and hemoglobin electrophoresis. In a small number of cases it was necessary a molecular test.

Results: 450 cases were identified, demonstrating a constant decreasing of hemoglobin level (9-11g%) with moderate polyglobulia (over 5-6 million/mmc) and reduced erythrocytes indices (MCV, MCH). The blood smear showed the usual modifications, hypochromia, microcytosis, anisocytosis, poikilocytosis and target cells. The hemoglobin electrophoresis revealed a HbA\textsubscript{2} ≥ 3,5% and moderate increasing of HbF. All these alteration permitted to elaborate a “Hematological, biochemical and molecular protocol for heterozygous β-thalassemia” that comes helping physicians and clinical laboratory scientists for a better and faster diagnosis.

Conclusion: Heterozygous β-thalassemia is a current public health problem even in countries with moderate incidence, like Romania. The notion of a protocol like the one we propose will improve the diagnosis, thus reducing the number of new cases with homozygous β-thalassemia.
Ranitidine Induced Thrombocytopenia

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Background: Thrombocytopenia due to ranitidine is very rare as a few cases until now are described in literature. Herein we present a case of an infant with a severe thrombocytopenia caused by ranitidine which completely subsided after discontinuation of the medication.

Objective-Methods: An 11 month old infant with a history of premature delivery and a mild bronchopulmonary dysplasia due to prematurity was admitted in our department because of bronchiolitis. The infant was under treatment by ranitidine since 20 days to cure an esophagitis caused by allergy in cow’s milk. In clinical examination the patient presented mild tachypnea, coughing and a purpuric rush in the face in the absence of fever. No significant enlargement of liver and spleen and no palpable lymph nodes were noticed. The blood count showed severe thrombocytopenia (platelet count: 19,000) and microcytic anemia. White cells count, hepatic enzymes and coagulations markers were normal. Inflammation markers were also unremarkable.

Results: Taking in account all the above, the most probable causes of Thrombocytopenia appeared to be idiopathic thrombocytopenic purpura and thrombocytopenia due to ranitidine. We decided to cease ranitidine treatment and after a few days a gradual elevation of platelets count was observed and it returned back to completely normal in a period of 12 days.

Conclusion: An immune mechanism probably can explain thrombocytopenia after ranitidine intake, in which ranitidine functions as hapten or antigen and leads to formation of antibodies targeting against platelets. The clinicians should be alert in cases of thrombocytopenia without overt cause and always keep in mind that it could be due to a medication.
Background: Hemophilia is an heterogeneous rare bleeding disorder, due to deficiency of functional plasma clotting factor VIII. The clinical features are variable whose severity is totally dependent on the severity of the deficit.

Objective: Report an exceptional revelation’s mode of hemophilia.

Observation: We report a 9-month-old infant hospitalized for management of left upper limb edema appeared after venipuncture for blood test. He was born to non-consanguineous parents; he has two sisters in a good state of health. We note an episode of hemorrhagic syndrome at the age of 6 months. Examination on admission notes pallor, upper left flexion limb stretched by a circumferential edema from the root of the left arm extended to the fingers with presence of ecchymotic closet oozing at the elbow. Passive and active mobilization was painful and very limited with a humeral and radial pulse difficult to palpate. Doppler ultrasound has found a large and ill-defined compressive hematoma at the elbow, the forearm and the lower 1/3 of the left arm compressing vessels. The infant had an emergency transfusion of factor VIII followed by a fasciotomy with hematoma evacuation. Coagulation tests showed a severe hemophilia A. The outcome was favorable and skin closure was made after 14 days.

Conclusion: Compartment syndrome revealing Hemophilia is rarely reported in the literature. Replacement therapy associated with surgery allows a better prognosis.
Chediak-Higashi Syndrome Revealed By Hemophagocytic Syndrome

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Background: Chediak-Higashi syndrome (CHS) is a rare autosomal recessive disorder characterized by hypopigmentation, severe recurrent bacterial infections, bleeding tendency and neurological impairment.

Objective: To get a new pediatric observation of a rare disease.

Observation: A two years old child without significant medical history admitted for prolonged fever lasting for ten days. He comes from second degree consanguineous parents. On the physical examination: he is eutrophic, febrile, a blond complexion with clear hair, a preserved general state mucocutaneous pallor with the presence of diffuse petechial. The abdomen is distended with hepatomegaly 6 cm firm and painless and splenomegaly 8 cm from the costal margin. The biology revealed a bicytopenia with microcytic hypochromic anemia (5g/dl), thrombocytopenia at 19000, white Blood Cells at 9000/mm³ with presence of big basophile inclusions, a negative CRP, a hypercholesterolemia at 4,2 g/dl, a hyperferritinemia, a hyponatremia at 129mmol/l. The myelogramme demonstrated the presence of basophil cytoplasmic inclusions in lymphocytes and polymuclear cells. The serology of Epstein Barr Virus was positive and the other serologies were negatives (leishmaniose, viral hepatitis, B19 parvovirus). The abdominal echography showed hepatomégaly, splenomegaly and deep abdominal adenopathies. The diagnosis of CHG syndrome complicated by hemophagocytic syndrome was suspected and confirmed by the hair biopsy. The EBV infection was the probable precipitating factor. The child received iterative transfusions of red blood cell and platelets. The evolution was marked by clinical and biological aggravation. He was treated by antibiotics, methylprednisolone for 5 days and immunoglobulins without amelioration. He died in 30 days of hospitalization in a hemorrhagic shock table.

Conclusion: The CHS is a disease with a so severe vital prognosis. Our observation confirm that most patients enter in accelerate phase often following EBV infection. Transplantation remains the treatment of choice.