

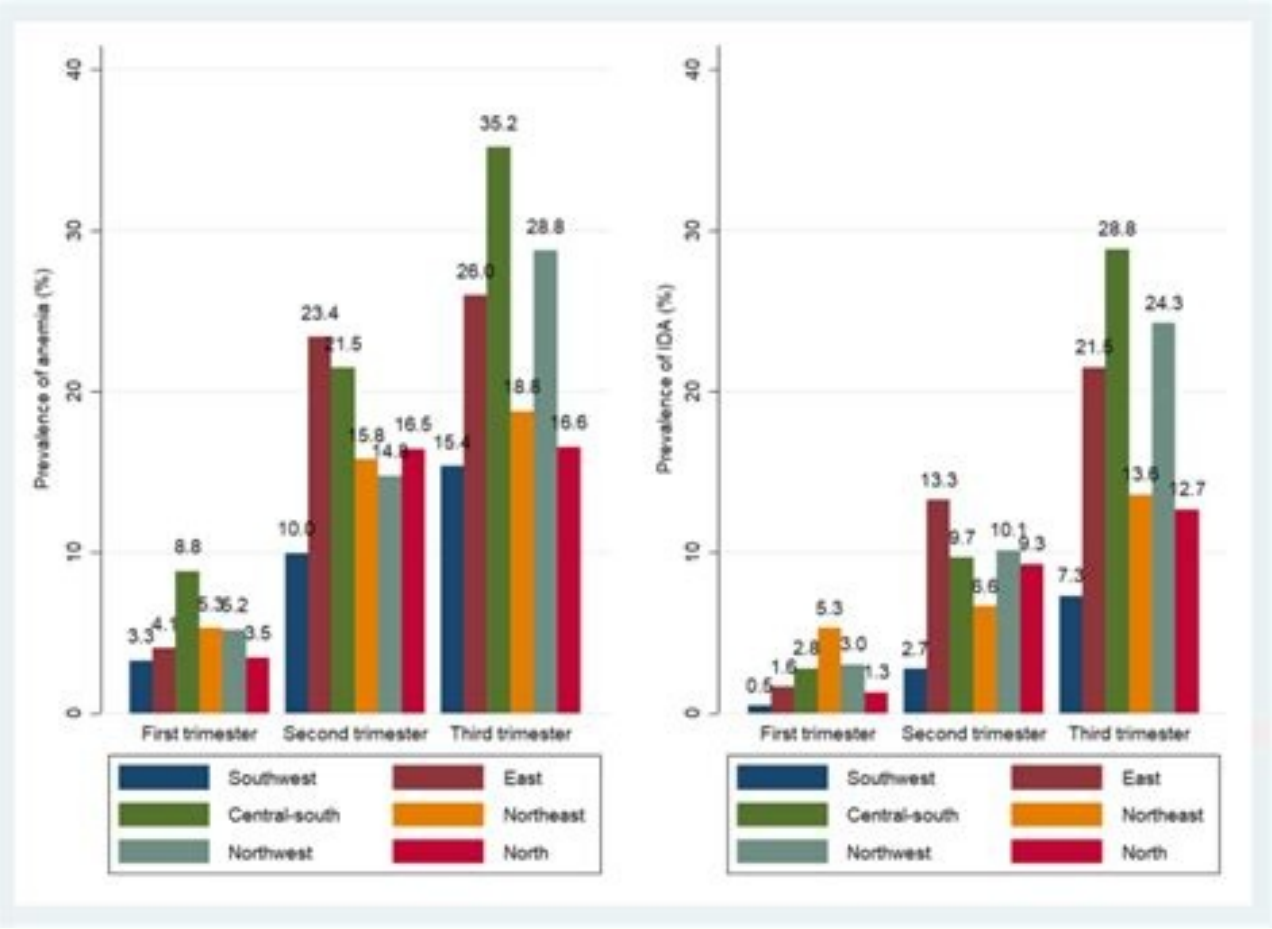
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CARDIAC COMPLICATIONS IN THALASSEMIA MAJOR

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Abstract

Thalassemia major is characterized by chronic ineffective erythropoiesis and anemia as its primary problems. These, in turn, produce physiologic adaptations in the cardiovascular system as well as pathologic/iatrogenic processes such as iron overload, splenectomy, nutritional deficiencies, chronic oxidative stress, and lung disease. This article discusses the pathophysiology of thalassemia as it relates to the cardiovascular system, the mechanisms and monitoring of iron cardiomyopathy, pulmonary hypertension, and vascular aging in thalassemia patients.

Keywords

Thalassemia; Heart complications; Iron cardiomyopathy; Pulmonary hypertension

THALASSEMIA AND THE CARDIOVASCULAR SYSTEM

Chronic Anemia

Patients with chronic anemia increase their cardiac output to maintain oxygen delivery, resulting in increased cardiac dimensions and heart rate. Anemic patients have larger hearts on CXR, echo, and MRI measurements than patients with normal hemoglobin levels, even without any other pathology. Thus, normative data generated from non-anemic patients is inappropriate for patients with hemoglobinopathies (1). The larger cardiac dimensions, stroke volumes, and heart rates carry metabolic cost; chronically anemic patients have higher resting oxygen consumption and decreased reserves. Increased resting metabolism is also a source of increased oxidative stress, independent of the free-radical effects of iron.

Patients with thalassemia have low or normal blood pressures, despite their increased cardiac output, because they have lower vascular resistance. Lower tonic vascular tone partially compensates for the increased chamber dimensions, but it leaves patients vulnerable to the endothelial toxicity of iron overload as well as making them less tolerant and responsive to the effects of afterload-reducing agents.

Splenectomy

Hypersplenism is relatively common in the thalassemias and may necessitate spleen removal. Splenectomy may also be performed to lower blood transfusion requirements. However, the spleen plays a critically important role in removing hematologic debris from the cardiovascular system. Phosphatidylserine positive platelets, platelet fragments, and red

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Pulmonary Function Tests in Sickle Cell Disease: Correspondence

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To the Editor: Prospective studies indicate that children with sickle cell disease (SCD) have progressive deterioration of lung function with increasing age [1]. We read with much interest the article titled "Pulmonary function tests in sickle cell disease" published online first in Indian Journal of Pediatrics [2], as it is the first such report from India and have the following comments to offer.

The authors conclude by stating that "pulmonary functions of Indian SCD children are statistically significantly reduced as compared to controls of similar age, however this difference was not clinically significant" only on the basis of the finding that pulmonary function test (PFT) parameters were not significantly different in children with or without history of acute chest syndrome. But the clinical parameters pertaining to respiratory system (e.g., cough, breathlessness on exertion, respiratory rate etc.) or exercise capacity [3] were not compared between the study and the control groups.

Though the children with acute respiratory illness were excluded, the past history of respiratory diseases (e.g., pneumonia, wheezing) apart from acute chest syndrome and family history of asthma/asthma, which are known to affect the PFT parameters [4] were not taken into account.

The authors posit that the children with SCD may have only restrictive pattern in PFT on the basis of a lower forced expiratory volume in 1 s (FEV1) and forced vital capacity (FVC) compared to controls. But the spirometry parameters were within normal limits in both groups which may be seen in milder cases of obstructive lung disease also, wherein forced expiratory flow between 25 % and 75 % of vital capacity (FEF25–75) would have been a more sensitive marker [5]. It would have been worthwhile to look at the spirometry parameters in light of the sickle cell disease parameters (frequency of transfusions, current hemoglobin, HbF level, use of medications for increasing HbF) of these children [6].

The purpose of comparing the hemoglobin levels of the SCD children with that of the healthy controls is not very clear as these children are expected to be anemic. Furthermore, performing a hemoglobin estimation of healthy children of this age group only for this purpose also cannot be supported.

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