

Evidence of Platelet Dysfunction in Congenital Cyanotic Heart Disease: Relevance to Palliative Systemic-Pulmonary Shunt and Haemostatic Parameters

Fatma Alzahraa Mostafa¹, Hala Agha¹, Hala Gaber², Samar Alsoda¹

BACKGROUND:

Abnormal platelet function has been hypothesized to play a role in the haemostatic abnormalities in cyanotic congenital heart disease (CCHD) patients. Blood platelets in patients with CCHD were occasionally characterized by significantly enhanced expression of P-selectin in resting circulating platelets as well as their augmented activation in response to stimulating agents.

OBJECTIVE:

To study the platelet functions in patients with CCHD by determination of P-selectin expression and the GPIIIa polymorphism and to correlate these findings with other clinical, radiological, and laboratory parameters in these patients.

METHODS:

This study included 47 patients, with CCHD with decreased pulmonary blood flow, attending the Pediatric Cardiology Clinic in the New Pediatric Hospital, Cairo University.

They were divided into two groups:

Group I: 31 patients with congenital cyanotic heart disease before cardiac surgery (23 males, 8 females age range 1m-10y). Group II: 16 patients with congenital cyanotic heart disease 3-6 months after palliative or corrective cardio surgery (11 males, 8 females, age range 2m-6y).

Group III: 30 age and sex matched normal children were included in the study as control group.

Full history taking with emphasis on thrombosis or embolism. General and local cardiac examination. Chest X-ray. Echocardiography: 2D, M mode and Doppler echocardiography. Pulse oxymetry. Flow-cytometric evaluation of platelet activation by P-selectin expression. P-selectin expression was estimated using monoclonal antibody. Typing of GPIIIa gene polymorphism

RESULTS:

There is statistically significant higher expression of P-selectin in group I and II compared with controls with significantly higher expression in young patients below 3 years. Significant higher levels of P selectin in A2A2 patients than controls with significantly higher levels in positive phenotype patients compared with positive phenotype controls. Significant negative correlation between oxygen saturation and P selectin expression was found in patients.

CONCLUSION:

Platelet activation may be an important contributor in the high thrombotic liability in CCHD patients and may be attributed in part to genetic factors. The use of platelet function or activity inhibitor in these patients needs further studies to be established.

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- 1- Pediatric department, Cairo university.
 - 2- Clinical Pathology department, Cairo university.