Congenital Syphilis and Ventricular Septal Defect

A 6600 g male infant, delivered vaginally at 38 weeks of gestation with a birth weight of 3500 g, was found to have moderate hepatomegaly, mild ascites, lower limbs edema, oliguria, hyponatremia, leukocytosis (24.8×10^3 cells mm^{-3}) and severe anemia ($5\,g\,dl^{-1}$) without apparent cause at 2 months and 17 days after birth. The mother had no antenatal care but at birth he was apparently healthy until day 45 when began to present with fever.

The baby was transferred to our hospital where serological tests performed included syphilis, investigated by Venereal Disease Research Laboratory test (VDRL) analysis of Cerebro-Spinal Fluid (CSF) and blood, found a titer of 1 : 64 in blood with a positive Fluorescent Treponemal Antibody Absorbed assay (FTA-ABS). Maternal and paternal VDRL titers at that moment were 1 : 16 in both cases. During hospitalization, the degree of hepatosplenomegaly increased, and the ascites, edema and anemia persisted, now presenting with high blood pressure (170/110 mmHg), controlled with enalapril. A Doppler echocardiography performed 92 days after birth revealed a ventricular septal defect (VSD), of the perimembranous type, with a size of 3.2 mm (small, <1/3 of the aortic root diameter which was 10.0 mm). No other relevant findings were found in that study. Both the infant and the mother were treated with aqueous and benzathine penicillin G, respectively. After that, his clinical conditions significantly improved in the following 2 weeks. As the VSD was small and there were no further cardiac symptoms, neither medical therapy nor surgery was indicated at the moment. Patient was discharged and follow-up was indicated.

In much of the world, congenital syphilis continues to present a significant public health problem. Untreated syphilis among pregnant women can profoundly affect pregnancy outcome [1], as we see in this case. On the other hand, although VSD is the most common form of congenital heart disease and one of the most frequently seen congenital abnormalities [2], its association with congenital syphilis has not been previously reported. The only similar case was found to be a report in which syphilis was the cause of an abnormal fetal echocardiogram [3]. These findings are supported by studies demonstrating cardiac tropism by *Treponema pallidum* [4]. Recently, some authors have drawn the attention on environmental factors, including infectious diseases, as potential etiological agents of VSD [2, 5].

As most research concludes that the etiology of congenital VSD is most likely to be multifactorial, [2, 5] infectious diseases (including syphilis), where these are at higher prevalences, should be considered in the potential etiology of VSD and other congenital heart diseases.

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Association of Mean Platelet Volume between Glucose Regulation in Children with Type 1 Diabetes

Mean platelet volume (MPV) is a marker for platelet activation. Elevated MPV levels have been reported as an independent risk factor for myocardial infarction in patients with coronary heart disease. Larger platelets are more reactive and aggregable [1, 2]. Type 1 diabetes mellitus is a chronic metabolic disorder caused by an absolute or relative deficiency of insulin. Altered platelet morphology and function

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have been reported in patients with type 1 diabetes. They are likely to be associated with the pathological processes and increased risk of vascular disease seen in these patients. The aim of our study was to evaluate MPV and its relation with glucose regulation parameters in children with type 1 diabetes.

Twenty-four children with diabetes mellitus and 32 healthy children were included into the study and Mann–Whitney U-test was applied for the comparisons of the quantitative data. The mean age of diabetic children was 7.9 ± 4.2 years and control group was 7.4 ± 3.16 years. Age, gender, body mass index (16.4 ± 3.2 vs. $15.7 \pm 3.7\,kg/m^2$) did not differ among groups. There was no significant difference between the groups for platelet count (333.7 ± 71.3 10^9/l vs. 339.6 ± 101.9 10^9/l). We detected significantly higher MPV values in children with type 1 diabetes when compared with control group (8.7 ± 0.49 fl vs. 7.7 ± 0.9 fl, $P = 0.005$). However, there was no significant correlation between MPV and fasting mean glucose, HbA1c, BMI, age.

We detected higher MPV values in diabetic children when compared with control group. In a study performed in 22 diabetic cases by Saigo *et al.* [3], MPV values were detected to be higher, but after the decrease in blood glucose there was a significant decrease in MPV values. Also in the studies of Tschope *et al.* and Hekimsoy *et al.* MPV values were higher in diabetic patients when compared with normal controls [4, 5]. Increased platelet activity could contribute to increased risk of both micro- and macro-vascular complications in type 1 diabetes mellitus. In present study, we have shown that MPV was significantly higher in diabetic children when compared with non-diabetic healthy children. However; we did not find a correlation between HbA1c and the other parameters. It might be related to younger age and short duration time in our diabetic group. We speculated that increased MPV might be associated with diabetic complications and future studies should investigate the correlations increased MPV with vascular complications in diabetic patients.

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