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Case Report (Pages: 7719-7722)

Intracardiac Thrombus in Renovascular Hypertension: A Case Report

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Abstract

Thromboembolism is a rare complication of primary nephortic syndrome. Both venous and arterial thrombosis might occur in steroid responsive and steroid resistant nephrotic syndrome. This is the report of an infant with nephrotic syndrome and renovascular hypertension, complicated with asymptomatic intracardiac thrombus and managed appropriately with medical treatment.

Key Words: Infants, Hypertension, Nephrotic syndrome, Thrombosis, Treatment.

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1- INTRODUCTION

Thromboembolism is a rare and serious complication of primary nephrotic syndrome (NS) with an incidence of 10-30% (1-3). Increased coagulability in NS occurs secondary to urinary excretion of natural anticoagulant proteins (plasminogen, protein C, protein S, and antithrombin III), increased synthesis of procoagulant proteins (fibrinogen and factor VIII), thrombocytosis with enhanced platelet aggregation, decreased thrombolytic activity, volume depletion, hyperlipidemia, hypoalbuminemia in addition to steroids and cyclosporine administration (4-6). Intracardiac thrombosis is the least common variant of thrombosis in NS, which could be seriously complicated with cerebral, brachial or popliteal emboli (3, 4, 7). This is the report of an infant with NS secondary to renin mediated hypertension, and complicated with an asymptomatic intracardiac thrombosis.

2- CASE REPORT

A 4- month- old boy was admitted for irritability, vomiting, seizure and mild leg edema in Ali Asghar Children's Hospital, in Tehran, Iran. He had increased blood pressure (200/120 mm/hg) in 4 limbs with normal peripheral pulse. Growth indices, physical and neurologic examination were normal. Serum level of sodium, potassium, and albumin decreased, contrary to increased renin and aldosterone level. All serologic tests had normal values. Urinalysis showed microscopic hematuria with nephrotic range proteinuria. Small left kidney with increased resistive index, and decreased renal perfusion was reported in sonographic evaluation. Accordingly, selective renal angiography showed stenosis and hypoplastic left renal artery. Brain CT-scan revealed parietal, occipital and temporal lobe ischemia, in favour of hypertensive encephalopathy. Meanwhile, blood vessels seemed normal in brain magnetic resonance angiography. Two dimensional echocardiography revealed a smooth, echogenic non-mobile mass with irregular surface attached to the apical wall of left ventricle, measuring 7×6 mm, which was in favour of Intracardiac thrombosis. Other echocardiographic parameters were within normal limit with preserved left ventricular systolic function, no aneurysm, wall hypokinesia (**Figure.1**), and evidence of bacterial or viral endocarditis. Cardiac magnetic resonance imaging was performed for differentiation of tumoral lesions and intracardiac thrombosis, which showed a 6×6 mm mass, attached to the apical-inferior left ventricular wall and interventricular septum, with no perfusion and delayed post-gadolinium enhancement, diagnostic of intraventricular thrombosis.

Serum level of coagulation factors V and VIII, protein S and C and antithrombin III were normal. Screening for lupus anticoagulant and Anti-cardiolipin antibodies were negative. Cardiothoracic consultation suggested nonsurgical treatment for intracardiac thrombosis. Therefore, anticoagulation therapy with subcutaneous low- molecular weight heparin (LMWH) was started and continued by oral warfarin. Follow-up echocardiography showed minimization of left ventricular thrombosis in 2 weeks, and complete resolution during 4 months with no further evidence of thromboembolism.

Increased blood pressure was controlled with antihypertensive drugs, followed by improvement of proteinuria. Blood pressure and urine protein excretion sustained normally with no further medical treatment after 1 year of follow up. Informed consent was obtained from the legal guardians.

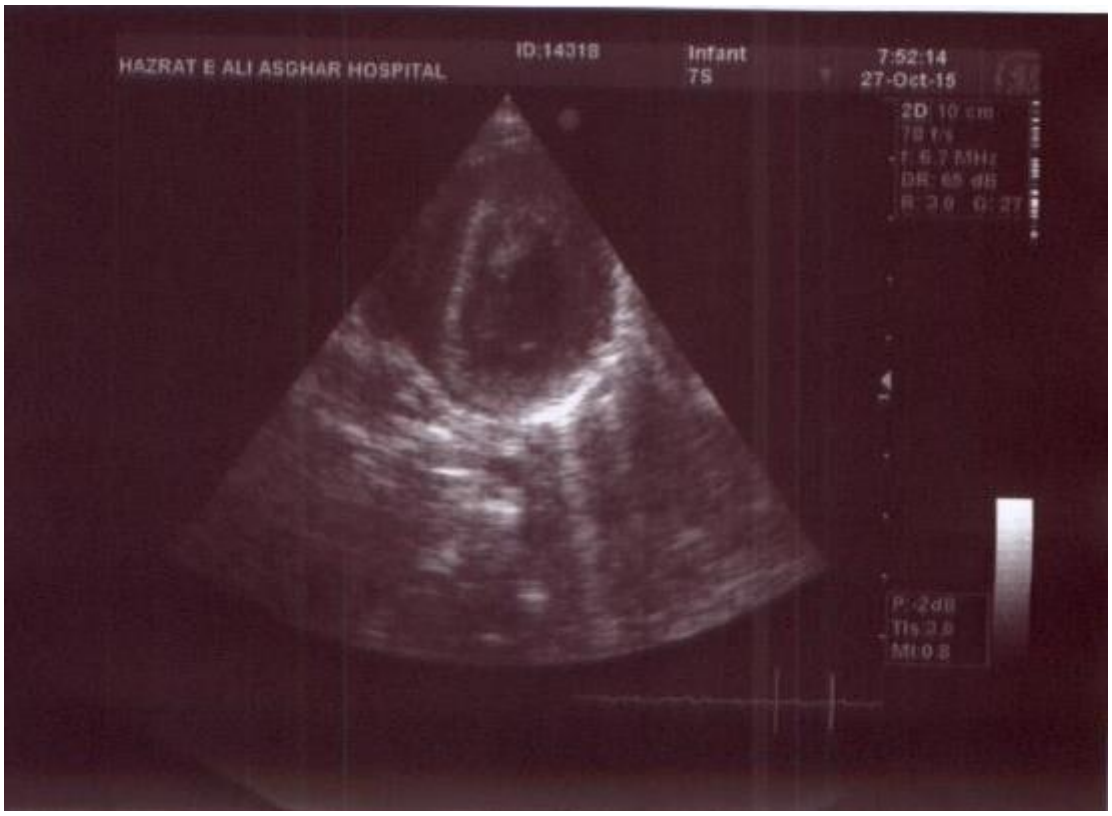

Fig.1: 2- Dimensional echocardiography showing one thrombosis attached to the apical- inferior wall of the left ventricle.

3- DISCUSSION

Thromboembolic events occurs in 1.8-5% of children with NS, with a higher incidence in steroid-resistant (3.8%) than steroid-sensitive patients (1.5%). Vascular thrombosis occurs most commonly in lower leg and renal vein, followed by arterial involvement (1, 5). Intracardiac thrombosis is a rare arterial thrombosis, which have been almost exclusively reported in patients with dilated cardiomyopathy, valvular heart disease, prosthetic heart valves, and myocardial infarction (1, 2). To the best of our knowledge, there are a few reports of intracardiac thrombosis in children with steroid sensitive or resistant NS (7-11). It is usually an asymptomatic complication (6, 10), and routine echocardiography have been suggested as screening test for characterization and therapeutic interventions of intracardiac thrombosis (4-6). In addition, platelet count, and measurement of serum fibrinogen, antithrombin III, and D- dimer might help to identify patients at risk of thrombosis (12). This article represents a rare type of NS secondary to renin mediated hypertension, which was complicated with an asymptomatic large intraventricular thrombosis. Therefore, screening of intracardiac thrombus is suggested in both primary and secondary NS. According to the low incidence and absence of consensus regarding management of this complication, optimal treatment is still unclear. However, anticoagulation with heparin, warfarin, and thrombolysis with fibrinolytic agents have been suggested to decrease life threatening embolic complications in the acute setting of thrombosis. Surgical management has been rarely recommended for the prevention of potential embolization

events in large mobile intracardiac thrombosis (4, 12-13). In this case, minimization of thrombosis started after 2 weeks of anticoagulation therapy and complete resolution occurred after 4 months of warfarin administration. Another interesting point of this patient was complete resolution of hypertension and proteinuria after discontinuation of antihypertensive treatments, which may be related to increased renin secretion of the involved kidney and suppressed renin secretion from the opposite kidney, resultant to normal serum renin level and normal blood pressure with improvement of proteinuria. In addition, antihypertensive treatment is recommended for hypertensive induced NS, to prevent thrombotic complications.

4- CONCLUSION

In conclusion, screening of asymptomatic intracardiac thrombosis is suggested in both primary and secondary NS. In addition, antihypertensive treatment is recommended in hypertensive induced NS, to prevent its related thrombotic complications

5- CONFLICT OF INTEREST: None.

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