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CASE REPORT

Nephrotic Syndrome with Intracardiac Thrombosis: A Case Report

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Abstract

Although thromboembolic events are well known complications of nephrotic syndrome, there are several unanswered questions relating to the nephrotic syndrome and thromboembolism such as the mechanism of thromboembolism and optimal diagnostic and therapeutic management strategies. We report on a case of a 26 yearold female with nephrotic syndrome, intracardiac thrombi, and evidence of embolization to brain and spleen. Diagnostic evaluation revealed a patent foramen oval with intracardiac thrombi. Despite adequate anticoagulation therapy, the left ventricular thrombus embolized into the spleen. However, with continued anticoagulation the patient had a satisfactory recovery and was released from the hospital with minimal neurologic deficit.

Key words: Intracardiac thrombi, Nephrotic syndrome, Thromboembolic Events.

Introduction

Nephrotic syndrome is a clinical condition characterized by proteinuria, hypoalbuminemia, edema, and hyperlipidemia. It was in 1949, when Adis reported for the first time the high incidence of thromboembolic complications of nephritis (1). Since then, a number of reports have described thromboembolic complications involving veins and arteries in nephrotic syndrome (2– 6). Intracardiac thrombosis is a very rare complication of nephrotic syndrome with only 8 such cases reported since 1949 (7–14).

Case report

A 26 year-old Sudanese female with membranous type of nephrotic syndrome since 2006 presented with left side weakness for 5 days duration. No previous cardiac history or other medical conditions. Physical examination revealed lower extremity edema, ascites, and left side hemiparesis, otherwise no other abnormalities were detected. Her kidney

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function test showed creatinine = 1 mg/dl (0.7-1.3 mg/dl), urea = 22 mg/dl (8-20 mg/dl), Na = 135 mmol/l (135-145 mmol/L), K = 3.7 mmol/l (3.5-5.5 mmol/L), and urinalysis showed protein 3+. Other laboratory tests showed Hgb = 10.8g/dl (12-16 g/dl), Platelets = 334,000/uL (150.000-350,000/ uL), PT = 13 s (11-14 s), INR = 1.2 (1.1-1.3), APTT = 33 s (25-35 s), Total Cholesterol = 533 mg/dl (150-199 mg/ dl), Triglycerides = 484 mg/dl (<150 mg/dl), serum albumin = 1.57 g/dl (3.5-5.5 g/dl), total protein = 3.6 g/dl (6-8 g/ dl). On admission, a CT scan of the brain showed right side cerebral infarction, which suggested paradoxical embolization as she was at risk for deep vein thrombosis (DVT). Doppler ultrasound of both legs done and showed no evidence of deep vein thrombosis (DVT). Transthoracic echocardiography was then performed and revealed an entangled thrombus at the site of foramen oval (figure1).



Figure 1: Four chamber view by TTE shows thrombus(16x6mm) in the right atrium attached to inter atrial septum at the foramen ovale

In addition, we observed a second, large (16 X 16 cm) mobile thrombus attached to the apical septal wall of left ventricle (figure 2). Other echocardiography parameters were within normal range showing preserved left ventricular systolic function, no aneurysm, or wall hypokinesia. Thus, we increased the heparin dose and started warfarin. These findings confronted us with a dilemma regarding the appropriate management of the large unstable left ventricular thrombus. Is it prudent to continue anticoagulation alone, or we had to intervene

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Figure 2: Four chamber view by TTE shows thrombus(16x16mm) in the left ventricle attached to the septal wall



Figure 3: Abdominal CT scan shows splenic infarction

surgically to prevent another embolization? A cardiac surgeon recommended not to intervene surgically based largely on the lack of evidence in the literature to support surgical removal of an intracardiac thrombus. Ironically, the next day the patient developed acute onset abdominal pain with no associated symptoms. Basic laboratory tests and abdominal ultrasound were normal. Coagulation profile revealed PT = 21 s (11-14 s), INR = 2.1 (1.1-1.3), APTT 34 s (25-35 s). We ordered abdominal CT scan which showed splenic infarction (figure 3). A repeated

transthoracic echocardiography did not find any intracardiac thrombi. We believe that the large mobile left ventricular thrombus embolized into the spleen, while the right atrial thrombus may regressed with treatment or embolized into the lung. However, prophylactic oral anticoagulation continued and after 19 days of admission transesophageal echocardiography performed and showed no evidence of any intracardiac thrombi. The patient discharged with minimal residual neurological deficit and given appointment for follow-up.

Discussion

An association between the nephrotic syndrome and thromoembolic events has been known for years (15), although the actual mechanism is not yet known. An imbalance between thrombotic and antithrombotic factors as well as other contributing factors such as intravascular volume depletion, thrombocytosis, platelet hyperactivity, hypercholesterolemia, steroid use, and infections have been implicated in the pathogenesis of various thromboembolic phenomena (16). The cumulative incidence of thromboembolic complications in patients with nephrotic syndrome is nearly 50% (17). Among the reported cases, intraventricular thrombosis is considered to be one of the rarest sites of thrombosis. To the best of our knowledge there are few case reports of intraventricular thrombosis with nephrotic syndrome (7, 8, 10, and 12). Because of the rarity of this complication and the absence of consensus regarding the management of intraventricular thrombus in general, the optimal management is still unclear. In all reported cases of intracardiac thrombosis in nephrotic syndrome, treatment with anticoagulation with or without surgical removal has been effective in the resolution of the thrombus, even though there are no controlled studies supporting one approach or another (7-14). As a general rule, surgical removal of intracardiac thrombus should not be considered unless the risk of embolization is felt to be extremely high or the patient is not a candidate for anticoagulation therapy, or both. Multiple echocardiographic characteristics of intracardiac thrombus need to be analyzed to estimate the risk of embolization, including; mobility, shape, heterogeneity, echo density, layering, central echo lucency, presence within an aneurysm, and association with low-density swirling echoes. Incidence of embolization is significantly higher in patients with thrombi that were mobile or protruded into the ventricular cavity (18).

Conclusion

Intraventricular thrombosis is a rare and serious complication of nephrotic syndrome. A high index of suspicion is needed by the physician as many of these thrombi are asymptomatic at presentation but can lead to potentially life-threatening complications. In absence of controlled studies, anticoagulation is the standard treatment in many cases, but surgery should be considered in cases in which the risk of embolization is high.

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