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Case Report

Antiphospholipid Syndrome Presented with Renovascular Hypertension

Abstract

Renovascular pathologies are one of the treatable causes of hypertension. Antiphospholipid syndrome develops owing to a heterogeneous group of antiphospholipid antibodies which causes various thrombotic problems. This entity may effects very small vessels and sometimes leads to hypertension.

We present a 55-year-old female with unilateral renal artery stent implantation because of renovascular hypertension. After application, a re-stenosis developed and in-stent angioplasty was performed, but it was required a nephrectomy because of haemorrhage. Severe ischemic nephropathy was detected in the nephrectomy material. She was diagnosed with antiphospholipid syndrome. Prolonged prothrombin time with hemorrhagic diathesis which coexisting thrombosis responded steroid therapy. But in follow-up, the thrombocytopenia developed, the patient could not receive anticoagulant therapy and died due to a pulmonary embolism-like syndrome.

This case reminds, renovascular hypertension is one of the major reasons of secondary hypertension and antiphospholipid syndrome always should be considered in thrombotic processes.

left side of umbilicus. The patients' laboratory results are presented in **Table 1**. Magnetic resonance (MR) angiography revealed significant (95%) stenosis in left renal artery stent and over 50% stenosis in right renal artery. In-stent angioplasty for left renal artery was performed but an abdominal pain developed 10 days after angioplasty and a perinephric hematoma was detected. Hematoma was removed with surgery but developed again and left nephrectomy was required. Because of postoperative hypervolemia and uraemia, hemodialysis was performed also.

In patients' follow-up, prolonged prothrombin time (PT), activated partial thromboplastin time (PTT) and thrombocytopenia were developed. Mixture test with equal quantity of patient's

Introduction

Antiphospholipid syndrome (APS) is a disorder by reason of antiphospholipid antibodies such as anticardiolipin antibody (ACA) and lupus anticoagulant (LA), and it generally present with venous thromboses and recurrent miscarriages [1]. The kidneys are involved in 25% of the cases [2]. Renal artery stenosis was reported both in lupus-associated and primary APS [1,3].

We presented a fatal case of APS with perirenal hematoma.

Case Report

A 55-year-old female patient presented with headache, flank pain and dyspnea. She had hypertension for 6 years and left renal artery stenosis had been detected 2 years ago. Conventional angiography had revealed 90% and 45% narrowing in left and right renal artery respectively. A stent implantation was applied for left renal artery. She had been administered amlodipine, doxazosin and clopidogrel. In admission, she was incompatible with treatment and used only oral amlodipine and captopril but no clopidogrel.

In physical examination pretibial edema was observed, blood pressure was 210/120 mmHg. A systolic murmur was present on the

Table 1: Patients' initial laboratory results.

	Patient's results	Normal values
Erythrocyte sedimentation rate	30	0-20 mm/h
Hemoglobin	9.8	11.5-16 g/dl
Hematocrit	31	35-45%
MCV	92	80-100 fL
White blood cell	2750	4000-10000/mm ³
Platelet	128000	150-400000/mm ³
BUN	39	5-23 mg/dl
Creatinine	1.5	0.6-1.2 mg/dl
Proteinuria	1-2 g/day	150 mg/day
Creatinine clearance	36	70-145 ml/day
Urinalysis	10-15 erythrocytes	
	7-8 leucocytes and rare hyaline cylinders.	

plasma and normal plasma were performed at 0, 30, 60 and 120 minutes. No improvement was observed at any incubation period. Platelet-derived phospholipids were incubated with patient's plasma and an improvement obtained with PT and aPTT. With positive anticardiolipin IgM and IgG, the findings considered compatible with antiphospholipid syndrome. Prednisolone with dosing 1 mg/kg/day was applied but because of progressive thrombocytopenia, anticoagulation was not fully applied.

In follow-up, a severe chest pain and dyspnea developed because of a possible pulmonary embolism. The cardiopulmonary arrest developed and the patient died when dealing with definite diagnosis.

Discussion

APS is a disorder associated with many heterogeneous autoantibodies such as ACA and LA. In primary form, there is no other accompanying disorder and in this form responsible factors for thrombosis, dysregulation of coagulation system, platelet activation, endothelial cell damage and microvasculopathy [4]. Almost in any organ, the risk of venous thrombosis generally increases [6]. Renal involvement in primary APS is reported in 25% of the cases [2,5]. The major glomerular lesions are capillary thrombosis, duplication of the glomerular basement membrane and glomerular microangiopathy. The clinical manifestations are proteinuria, active urinary sediment, hypertension and progressive renal insufficiency. Renal ischemia and infarcts exist in many of the patients [6].

In our case, based on uncontrolled hypertension, a renovascular problem was detected with finally diagnosed APS. In literature review, renal artery stenosis was observed in 20 (26%) out of 77 APS patients [7]. Also APS nephropathy was associated with extrarenal arterial thrombosis [7]. In patients with APS, because of severe thrombocytopenia and/or antiprothrombin antibodies severe haemorrhages can be observed. In our case, because of existing antibodies that effect both the PT and the aPTT, an improvement of these parameters obtained with addition of phospholipids suggests that antibodies dependent on phospholipids (antiphospholipid antibodies).

In these cases, anti-prothrombin antibodies cause hypoprothrombinemia by binding prothrombin and consequently increase haemorrhage risk [8]. There is no consensus for therapy

options in APS especially before and after vascular stent implantation. In literature, renal angioplasty technic was generally reported as case reports but the clinic results show variation [4,9,10].

In our case, because of re-stenosis of primary stent and following bleeding diathesis, optimal medical follow-up and treatment was not likely.

Conclusion

Renovascular problems may be the first sign in patients with APS. In these cases, because of thrombotic and haemorrhagic processes may follow each other, unfortunately to apply a routine follow-up program and to decide proper applications are quite difficult.

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