

## Blood Pressure Asymmetry Revealing an Antiphospholipid Antibody Syndrome

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### Abstract

### Case Report

Anti phospholipid antibody syndrome (SAPL) is an individualized chronic autoimmune disease such as the combination of thromboses and / or obstetric morbidity and the lasting presence of antibodies to phospholipids and / or their protein cofactors. SAPL is a rare disease. The progress made has led to a better stratification of the individual thrombotic risk. The treatment of SAPL is based on anti thrombotic drugs. We report the case of a patient admitted for accutization of chronic renal failure on bilateral thrombosis of the renal arteries in whom the immunological balance revealed the presence of circulating lupus type anticoagulants making it possible to retain the diagnosis of anti phospholipid antibodies.

**Keywords:** chronic autoimmune, Anti phospholipid antibody syndrome (SAPL), antibodies, immunological balance.

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## INTRODUCTION

In 1963, a cases of thrombosis associated with an prolongation of coagulation tests linked to the presence of a circulating anticoagulant of the lupic type or lupus anticoagulant [1] were described.

Anti-phospholipid syndrome (APS) is defined by thrombophilia which includes an arterial and / or venous or obstetrician thrombotic clinical manifestation in patients with anti-phospholipid antibodies [2], the precocious management of which limits the consequences and to initiate anti thrombotic treatment.

## PATIENT AND OBSERVATION

It was a 51-year-old patient with chronic unexplored renal failure with a plasma creatinine level of 18.3 mg / l, eDFG at 31ml / min / 1.73m<sup>2</sup> of body surface area, followed for HTA for 1 year without a history of diabetes or taking toxic substances. She was admitted for severe hypertension in a context of respiratory distress with dyspnea without lowering visual acuity or palpitations or sweating.

The clinical examination found a blood pressure at 210/130 mmhg with blood asymmetry

between the two arms, all the pulse were present, with the urine strip two blood crosses without proteins.

Biologically, worsening chronic renal failure with a creatinine level of 73.2 mg/l and a urea of 1.9 g/l without associated hydro electrolytic disorders; hemoglobin a 10.6 g/dl with a normal platelet counts without hyperleukocytosis. The inflammatory balance found a CRP at 50mg/l and a PCT level at 1.5 ng/ml.

Thoraco-abdomino-pelvic scanner revealed an obstruction 2 cm from the origin of the left subclavian artery extended over approximately 17mm, absence of opacification of the left renal artery with a dumb left kidney with partial hilar opacification of the right renal artery.

Renal angio-IRM objected to a complete occlusion of the left renal artery with very tight stenosis of the right renal artery.

In terms of infection: the patient was put on antibiotic treatment based on amoxicillin clavulanic acid in the face of suspected infection on renal necrosis with apyrexia after 48 hours.

In front of the atypical renal necrosis, an immunological assessment was carried out objectifying to negative anti-nuclear antibodies (1/80), AC anti negative DNA, soluble nuclear anti-SSA, Anti-SSB, anti-SM, Anti-Sc170, Anti-Jo1, Anti CENP-B, and Ribosome P Proteins had all returned negative.

The anticardiolipin antibodies IGG and IGM (on Alegria by Elisa SMC) and the anti-beta 2 glycoprotein I, IgG autoantibodies were negative. The search for circulating lupus-type anticoagulants had returned positive. The exploration of the endogenous life of hemostasis revealed a very positive factor 8, moreover the balance of thrombophilia had found a normal AT with low S and C proteins.

In consultation with the vascular surgeons, there are no indications of an emergency revascularization given the subacute character; on the

other hand anticoagulation was introduced on the basis of AVK under the supervision of the INR: International Normalized Ratio. The evolution was marked by the worsening of renal function and the conventional hemodialysis.

## DISCUSSION

The incidence of SAPL is estimated around five new cases per 100,000 subjects per year and its prevalence, approximately 20-50 cases per 100,000 people, varying according to ethnic groups. The SAPL mainly affects young or middle-aged subjects, in 85% of cases between 15 and 50 years of age. The male / female ratio is 1 / 3.5 in the case of primary SAPL and 1/7 if the SAPL is associated with lupus [3]. Conventionally, SAPL can be primitive or secondary to systemic diseases [4] (Table 1).

**Table-1: Clinical situations with antiphospholipid antibodies often non-pathogenic**

<p>Healthy subjects especially if old or parents of an individual with SAPL          All autoimmune diseases          Inductive treatment: procainamide, phenothiazine, hydantoin, quinidine, hydralazine, beta-blocker, interferon alpha, etc.          Infections: acute viral diseases, HIV, hepatitis C, Lyme, tuberculosis, malaria, etc.          Solid cancers, malignanthemopathies, monoclonal immunoglobulins          Miscellaneous: sarcoidosis, Crohn's disease, spondylarthropathies, insulin-dependent diabetes, end-stage renal disease, acute hepatocellular insufficiency, chronic ethylism, periodic disease, sterility, CIVD          Early and accelerated atherosclerosis</p>
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SAPL: antiphospholipid syndrome; HIV: human immunodeficiency virus; CIVD: disseminated intravascular coagulation.

During SAPL, the precise multiple mechanisms leading to thromboses or obstetric morbidity are still very incompletely elucidated. Many studies argue for a prominent role in the activation of monocytes, endothelial cells, platelets, complement as

well as interference with proteins that have an inhibitory role in coagulation. The result is vasculopathy, initiated mainly by intimal hyperplasia which can lead to vascular occlusions and obstetric disorders [3].

The positive diagnosis of SAPL is based on clinical-biological criteria; these diagnostic criteria were clarified during the Sapporo conference (Table 2).

**Table-2: International consensus on the criteria for classification of defined antiphospholipid syndrome [5]**

<p>Clinical criteria          Vascular thrombosis (arterial, venous, or microcirculatory)  <math>\geq 1</math> clinical episode confirmed by imagery or histology at the exception of superficial phlebitis Obstetric morbidity  <math>\geq 1</math> unexplained death of a fetus morphologically normal to from the 10th week of pregnancy          Or <math>\geq 1</math> premature birth of a morphologically normal newborn baby to or before the 34th week of pregnancy due to eclampsia severe or placental insufficiency          Or <math>\geq 3</math> unexplained consecutive miscarriages before 10th week of pregnancy, after exclusion of anatomical causes, hormonal and chromosomal</p>
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<p>Biological criteria          IgG and / or IgM anticorps, medium or high          (<math>&gt; 40</math> LPG or MPL or <math>&gt; 99</math>th percentile by a standardized Elisa method)          Lupus anticoagulant present in plasma according to international recommendations (ISTH)          Anti-beta antibodies 2 glycoprotein 1 IgG and / or IgM in one way  <math>&gt; 99</math>th percentile, by a standardized Elisa method</p>
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IgG: immunoglobulins G; IgM: immunoglobulins M; LPG: IgG antiphospholipid units; MPL: IgM antiphospholipid units; Elisa: enzyme linked immunosorbent assay; ISTH: International Society on Thrombosis and Haemostasis.

A clinical criterion and a biological criterion are sufficient with the following restrictions: the biological criterion must be present on two examinations at least 12 weeks apart, between 12 weeks and 5 years after the clinical event.

Renal damage during SAPL classically corresponds to a vascular nephropathy which can affect all renal vascular structures of which two types are described: an arterial form (proximal and / or distal, acute and / or chronic) and a venous form [6].

Proximal arterial nephropathy is defined by the presence of stenosis or thrombosis (in situ or from emboles) in large renal arteries. The clinic is that of renal infarction.

Distal arterial nephropathy is the most frequent renal impairment, the diagnosis is made on the renal biopsy objectifying congestion in the glomerular capillaries with the presence of clear subendothelial spaces and double contours of the walls of the glomerular capillaries. The venous form is rarer than the arterial form; it is mainly characterized by thrombosis of the renal vein or upstream veins.

Since renal vascularization is terminal, thrombi are responsible for infarction or area of ischemia / renal necrosis with intense stimulation of the renin-angiotensin-aldosterone system causing severe secondary HTA [7, 8].

Detection of kidney damage should be early. In the absence of treatment, the evolution is unpredictable with attacks that can be acute and / or chronic and the possibility of rapid installation of terminal renal failure. Therapeutic management could be based on effective anticoagulation without forgetting the use of classical nephroprotection (strict control of blood pressure with in particular therapies blocking the renin-angiotensin-aldosterone system).

## CONCLUSION

Renal damage is frequent and severe with lesions of nephropathy associated with SAPL which are today well described. This damage leads in a non-exceptional way to chronic terminal renal failure in the absence of codified management.

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