

# Clinical Case of a Patient with Antiphospholipid Syndrome

# Diana Smilkova\* and Georgi Marinov

Clinic of Cardiology, University Hospital, "Prof. Dr. St. Kirkovich", Stara Zagora, Bulgaria

\*Corresponding Author: Diana Smilkova, Associate Professor, Clinic of Cardiology, University Hospital, "Prof. Dr. St. Kirkovich", Stara Zagora, Bulgaria.

Received: January 28, 2020; Published: February 10, 2020

#### **Abstract**

Antiphospholipid syndrome is a systemic autoimmune disease with thromboembolic complications and/or pregnancy disturbances, characterized by a polyorganogenic effect on the background of an increased titer of antiphospholipid antibodies. It was first described in 1983 by Hizzis. Originally known as anticardiolipin syndrome, and a few years later as an antiphospholipid syndrome.

The patient is a woman, aged 37 years, hospitalized at the Clinic of Cardiology at the University Hospital, Prof. Dr. St. Kirkovich "in Stara Zagora, Bulgaria, with manifestations of cardiac failure and polyorganic deficiency.

Keywords: Antiphospholipid Syndrome; Autoimmune Disease; Thromboembolic Complications

## **Introduction and Case Study**

#### Anamnesis

The complaints of the patient began two months ago, resulting in progressive fatigue after antibiotic treatment of staphylococcus infection of the upper respiratory tract and in the days before hospitalization with nausea, vomiting and short-term syncope in the bathroom during toilet.

The performed echocardiography registered a symmetric left ventricular hypertrophy with suppressed systolic function of the LV with ejection fraction 30% and restrictive type diastolic dysfunction.

Valve lesions were detected - high-grade mitral and tricuspid regurgitation, moderate aortic regurgitation and PAH with 60 mmHg systolic pulmonary artery pressure.

ECG registered a sinus rhythm, voltage criteria for LVH with secondary repolarization changes.

During the stay in the clinic, the patient had clinical manifestation of polyorganic deficiency:

- 1) Renal dysfunction High levels of urea, creatinine and microalbuminuria.
- 2) Hepatic dysfunction High liver enzyme levels.
- 3) Symptoms by CNS Confusion, bradypsychia and left-sided hemiparesis.

Abdominal ultrasound was performed and registered diffuse renal parenchyma changes suspected to chronic glomerulonephritis or interstitial nephritis, cholelithiasis of a multiple microlithiasis type and bilateral exudative pleuritis.

In connection with CNS symptoms, opinion of neurologist was acquired and appointed CPT and MRI of the cerebrum, with data of a small ischemic cortex stroke and supratentorial focuses of ischemic induced gliosis.

Taking into account the age of the patient and the symptoms, a systemic illness was discussed and consulting with a rheumatologist was performed.

A Fabry, Goshe and Pompe disease testing was performed. An immunological testing was appointed with evidence of high level of anti-cardiolipin and anti-beta2-glycoprotein antibodies titers.

#### Laboratory results

- Hemoglobin g/l (135 180) 97; 96; 100; 107.
- Erythrocytes 10<sup>12</sup>/l (4.5 5.8)- 3.72; 3.61; 3.72; 4.06; morphology presence of poikylocytes, ellipticocytes, target cells, schizocytes, acanthocytes, hypochromia.
- Hematocrit (0.400 0.540) 0.29; 0.284; 0.293/0.329.
- Leukocytes 10<sup>9</sup>/l (3.5 10.5) 14.0; 13.16; 15.74; 12.57.
- Platelets 10<sup>9</sup>/l (140 440) 196/164/159/106.
- Glucose-serum-mmol/l (2.8 5.6) 11.4; 6.0; cholesterol-mmol/l (3.4 5.2) 4.5; LDL-mmol/l (0 2.09)- 2.81; HDL- mmol/l (0.0 2.09)- 0.96.
- Urea- mmol/l (1.7 8.3)- 26.9; 35.3; 39.2; 38.5; 34.9; 37.3; 35.5; 34.7.
- Creatinine- umol/l (80 127) 302; 410; 398; 319; 271; 283; 285; 249.
- Sodium- mmol/l (135 155)- 140; 139; 137; 136; 139; 139; 134.
- Potassium- mmol/l (3.5 5.6)- 4.5; 5.0; 5.2; 4.9; 4.5; 4.9; 5.0; 5.2.
- ASAT-U/l (0 37)- 54.0; 58.2; 79.1; 72.0; 43.7; 26.2; 21.1; 29.5.
- ALAT-U/l (0 41)- 106.0; 124.6; 171.1; 196.1; 153.9; 104.5; 79.8; 63.7.
- Creatine phosphokinase- U/l (24 195) 150; 127; 113; 130; 71; 34; 35; 39.
- Creatine phosphokinase MB- U/l (0 25) 24.0; 22.0; 20.4; 24.0; 17.7; 11.6; 11.6; 27.0.
- Troponin- ng/ml (0.0 0.04) 4.175; 7.135; 8.037; 8.876; 8.429; 9.363; 8.942; 7.970.
- LDH- U/l (225 450)- 990; 938; 1135; 992; 956.
- Bilirubin- umol/l total (0.0 17.0)- 15.7; 12.1; direct (0.0 4.7) 5.3; 3.6.

# Immunological results

- Anti-Nuclear Antibodies (ANA) Sketch ILF- Titre 1: 640 Norm <1:80.
- Anti-Nuclear Antibodies (ANA) Immunoblot Negative.
- Anti-ds DNA (double-stranded) negative.
- ANCA immunoblot negative.
- Antiglaucoma antibodies negative.
- Anti-Mitochondrial Antibodies negative.
- Anticardiolipin antibody (ACL) ELISA
  - IgG 35.6 IU/ml- positive (n < 10 IU/ml)
  - IgM 18.8 IU/ml- positive (n < 7 IU/ml)
- Anti-β2-glycoprotein 1 antibodies (Anti-β2-GP1) ELISA
  - IgG 22.1 IU/ml- positive (n < 5 IU/ml)
  - IgM 14.9 IU/ml- positive (n < 5 IU/ml)</li>

According to the symptoms, data of polyorganic deficiency as well as the performed imaging, laboratory and immunological tests, a primary antiphospholipid syndrome was adopted as the leading diagnosis.

Following the diagnosis, to the treatment, including a standard medication regimen for the treatment of heart failure and hypertension disease (venous diuretic, angiotensin converting enzyme inhibitor, beta blocker, dihydropyridine calcium antagonist), hepatoprotector, nootropic drug and water-salt solutions, were added corticosteroid- Methylprednisolone 40 mg and oral anticoagulant - Acenocoumarol with dose titration according to INR.

During the hospital stay, a total of 9 days, as result of the drug therapy, an improvement in the clinical condition of the patient was achieved. An improvement in LK systolic function was observed, with the ejection fraction reaching 50% before discharge.

The patient was discharged in good general condition and directed for outpatient monitoring by a cardiologist and rheumatologist.

## Home therapy

Acenocoumarol 4 mg- with dose titration according to INR, Lercanidipine 10 mg daily, Metoprolol Succinate 50 mg daily, Rosuvastatin 10 mg daily, Methylprednisolone 20 mg daily, Moxonidine  $2 \times 0.3$  mg, Vinpocetine  $2 \times 10$  mg, Perindopril 10 mg daily, Indapamide 2.5 mg daily, Chloroquine Phosphate 250 mg daily.

#### Conclusion

The diagnosis of Antiphospholipid syndrome may be challenging. The main pathogenic mechanism is thrombosis. The desiese must be suspected in patients with clinical symptoms of dysfunction of different organs and systems, especially atypical of their age, especially young women, with data of thrombosis and thrombocytopenia. In such cases a study of antiphospholipid antibodies should be performed. The early diagnosis and beginning of treatment are of importance [1].

# **Bibliography**

1. N Stoilov., et al. "Antiphospholipid syndrome - historical data, etiology and pathogenesis". Rheumatology 119 (2011).

Volume 7 Issue 3 March 2020

© All rights reserved by Diana Smilkova and Georgi Marinov.