AB0103 ACUTE PANCREATITIS AS INITIAL MANIFESTATION OF ANTI-PHOSPHOLIPID SYNDROME

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Background A 17 years old Belgian man was admitted with acute non-necrotic pancreatitis.

Laboratory data showed high levels of amylase and lipase, leucocytosis, erythrocyte sedimentation rate (ESR) 24 mm/h and CRP 8.7 mg/100 ml, IgM against Epstein-Barr Virus (EBV) and lupus anticoagulant (detected according to the guidelines of International Society on Thrombosis and Haemostasis) were present. The evolution was spontaneously favourable and the diagnosis of acute pancreatitis on EBV infection was retained.

Objectives Four months later, recurrent abdominal pain appeared and the only positive clinical finding was a livedo reticularis.

Laboratory test showed a high ESR (63 mm/h) but a normal CRP (0.2 mg/100 ml), haemolytic anaemia, leucopenia, hypo-complementemia, presence of lupus anticoagulant, antinuclear antibody at 1/640 with anti dsDNA 517, EBV IgM antibody title has not changed.

Neither cutaneous nor renal involvement nor serositis was observed.

Mesenteric angioCT was normal.

The patient recovered under a treatment of steroids, anticoagulation and hydroxychloroquine.

Methods

Results Pancreatitis is a rare manifestation in the anti-phospholipid syndrome (APS) or systemic lupus erythematosus (SLE) and is often associated with severe multi-systemic complication and of poor prognosis.

Although the aetiology is not known, vasculitis lesion and thrombosis, adverse effect of steroid and azathioprine were thought to be the cause.

Conclusion We describe a case of pancreatitis as initial manifestation of APS, since ACR criteria for SLE are not fulfilled.

No further complication was observed at 2 months? follow-up.

The presence of EBV IgM antibody is probably due to polyclonal activation.

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AB0104 ADRENAL INSUFFICIENCY PRIMARY ANTIPHOSPHOLIPID ANTIBODY SYNDROME AND AUTOIMMUNE HYPOTHYROIDISM

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Background Adrenal Insufficiency (A. I.) is an uncommon complication of the antiphospholipid antibody syndrome (APS). Several cases of A. I. are described in association with APS in patients with Systemic lupus erythematosus (SLE) or other connective tissue disease; in contrast A. I. in association with Primary Antiphospholipid Syndrome (PAPS) is seldom described.

Objectives We report a case of adrenal failure as first manifestation of APS and autoimmune hypothyroidism. Our patient is a 65 years old female who a month before was admitted to her local hospital for fever, cough and dyspnea. She had no previous history of thromboembolic or connective tissue disease. Chest radiograph showed an infiltrate and pleural effusion, which cleared over the next two weeks and she was discharged. Within few days, she developed nausea, vomiting, mild abdominal pain and fever. On admission to our hospital, she was dehydrated with pulse of 110/min, blood pressure 100/50 mmHg.

Methods Laboratory findings were: Hb 9.9 g/dl, WBC $16 \times 10^9/\mu\text{l}$, PLT $167 \times 10^3/\mu\text{l}$, ESR 90 mm 1st h. Natremia was 128 mEq/l, kaliemia was 4.3 mmol/l. Gastroscopy was normal, a CT scan revealed a bilateral enlarged adrenal gland with a normal cava and iliac and femoral vein. Anorexia, vomiting, weakness, weight loss, orthostatic hypotension and hyponatremia led us to suspect an adrenal insufficiency diagnosis, that was confirmed by a low plasma cortisol concentration 3.5 mg/dl (n. 6-30) and by an ACTH test. The serum ACTH concentration was 900 pg/l (n.6-46), aldosterone < 5 pg/ml (n. 7.5-150). APTT was 83.8 sec.(n. 20-40sec) with normal PT. Lupus anticoagulant was positive, Antiphospholipid antibodies IgG were positive:21.4 U/ml (n. <5). ANA, dsDNA and ENA were negative. TPHA, VDRL test, cryoglobulin and rheumatoid factor were negative. C3, C4 were normal. Antibodies against the adrenal cortex, tuberculin skin test and antibodies to HIV were negative. Thyroid hormones were: T3 0.56(n.0.45-1.37 ng/dl);T4 5.0 (n.4.50-12.00 ng/dl); FT3 1.90 (n.1.45-3.48 pg/ml); FT4 0.81 (n.0.71-1.85 ng/dl); TSH 7.44 (n.0.465-4.680 mU/ml); TgAb 1:160; TMAb 1:160.

Results The diagnosis of AI secondary to PAPS associated with bilateral adrenal infarction, which was probably secondary to thrombosis of the adrenal glands veins, was made. In the course of hospitalisation the patient developed an extensive deep venous thrombosis of right iliac and femoral vein with extending up to the inferior vena cava. She was treated with hydrocortisone, fluorydrocortisone, heparin and L-thyroxine with rapid improvement in clinical and biochemical status.

Conclusion This case suggest that thrombosis of adrenal vein causing A. I. may be the first manifestation of PAPS and that the diagnosis of PAPS should be considered in patient with A. I; particular intriguing is the finding of autoimmune thyroid disease in a patient with A. I and PAPS.

FRI0170 ANTICARDIOLIPIN ANTIBODIES IN SYSTEMIC RHEUMATIC DISEASES PATIENTS

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Background Anticardiolipin antibodies (aCL) are quite common findings in patients with systemic rheumatic diseases. However, clinical significance of aCL presence still remains not completely clear despite some very well known clinical associations.

Objectives The purpose of our study was to evaluate clinical significance of aCL in our patients with systemic rheumatic diseases.

Methods We examined 242 pts who suffered from rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), and systemic sclerosis (SS) and underwent treatment in Kaunas University of Medicine Rheumatology department. The control pts group