defects on ventilation-perfusion scan, typical of acute pulmonary embolism (Figure 1). She was initially diagnosed with acute pulmonary embolism and treated empirically with heparin. Subsequently a negative venous Doppler, absence of changes of acute or chronic thromboembolic disease on pulmonary angiogram, elevated ESR of 70 mm/hr, and the typical angiographic changes (Figure 2), led us to make the correct diagnosis of Takayasu's arteritis. Her initial arch angiogram was normal (Figure 3A). She responded to treatment with prednisone, with both clinical improvement and a 50% increase in the calibre of both right and left pulmonary arteries on a MR angiogram done six months later. A repeat pulmonary angiogram 11 months later showed a decrease in pulmonary artery pressures from 65/12 mm Hg to 49/10 mm Hg. She was lost to follow-up and returned six years later with symptoms of dizziness and angiographic evidence of carotid artery involvement (Figure 3B), which required arterial bypass grafting. Histopathological examination of the artery specimen showed patchy acute and chronic inflammation of adventitia, media and intima, focal granulomatous foreign body reaction, and intimal proliferation, changes consistent with Takayasu's arteritis.

Conclusion In young women presenting with a clinical picture of acute pulmonary embolism without any risk factors or a previous history of thromboembolism, pulmonary TA must be considered in the differential diagnosis. A careful evaluation of the clinical features, laboratory findings and angiographic appearance, will often lead to the correct recognition of pulmonary Takayasu's arteritis and the initiation of appropriate therapy.

REFERENCES

- 1 Lie JT. Isolated pulmonary arteritis: clinicopathologic characteristics. *Mod Pathol* 1996;**9**(5):469-74
- 2 Haque U, Hellmann D, Traill T, Venbrux A, Stone J. Takayasu's Arteritis involving proximal pulmonary arteries and mimicking thromboembolic disease. *J Rheumatol* 1999;**26**:450-3
- 3 Nakabayashi K, Kurata N, Nangi N, Miyake H, Nagasawa T. Pulmonary artery involvement as first manifestation in three cases of Takayasu's arteritis. *Int J Cardiol* 1996;**54**(Suppl):S147-53
- 4 Kerr KM, Auger WR, Fedullo PF, Channick RH, Yi ES, Moser KM. Large vessel pulmonary arteritis mimicking chronic thromboembolic disease. *Am J Respir Crit Care Med* 1995;**152**:367-73

AB0092 POLYARTERITIS NODOSA PRESENTING WITH BILATERAL PERIRENAL HEMATOMA IN PATIENT WITH FMF

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Background

Objectives

Methods Case Report: A 17-year old Turkish boy was admitted to our clinic with complaints of weight loss, fatigue and abdominal pain. He had been on colchicine therapy for 6 years with the diagnosis of FMF. On January 2000, he was diagnosed as having acute hepatitis A infection in another centre. Two weeks later he was hospitalised due to severe abdominal pain and abrupt decrease in his haemoglobin values. A right renal subcapsular hematoma was demonstrated by computerised tomography (CT). During follow-up haemoglobin values progressively declined and the hematoma showed enlargement in control CT. Therefore right nephrectomy was performed. After the patient was stabilised he was discharged. However he was again hospitalised with similar complaints 4 days later. This time subcapsular hematoma

was demonstrated in the left kidney and he was referred to our hospital in March 2000. In physical examination, blood pressure was 160/125 mmHg. Laboratory evaluation demonstrated anaemia and elevated erythrocyte sedimentation rate and C-reactive protein. Serological tests for anti-hepatitis B surface antigen, anti-hepatitis A IgG and IgM were positive.

Nephrectomy slides were re-evaluated in our pathology department. Patchy and segmental areas of renal infarct were demonstrated. No amyloid deposits could be noted in the histopathology specimens. Renal angiography demonstrated 2 microaneurysms on intertubular arteries. Together with these findings the diagnosis of PAN was made and the patients was treated with intravenous corticosteroids, intravenous cyclophosphamide and oral corticosteroids together with antihypertensive therapy. He is still being followed-up by our clinic in good health.

Results

Conclusion Perirenal hematoma, which is a rare complication of isolated PAN, have been reported in approximately half of the patients with FMF who developed PAN. The exact pathogenetic relationship of FMF and PAN also is not clear. Immune-related mechanisms have been demonstrated in FMF. It has been proposed previously that infection-related antigenic stimuli could trigger the development of vasculitides. In the present case an usual response to hepatitis A infection and/or previous hepatitis B infection might have been among the triggers of vasculitic process. Additional reports demonstrating direct causal relationship between infectious agents and vasculitic syndromes in patients with FMF will help us to clarify the relationship between the two disorders.

AB0093 CALCIPHYLAXIS DUE TO LONG-TERM STEROID USING IN A PATIENT WITH RHEUMATOID ARTHRITIS

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Background Calciphylaxis is a rare and life-threatening condition of progressive cutaneous necrosis secondary to small and medium-sized vessel calcification seen almost exclusively in patients with end-stage renal disease and hyperparathyroidism.

Objectives We report a case with calciphylaxis due to long-term steroid using in a patient with rheumatoid arthritis (RA).

Methods

Case report.

Results A 43-year-old man admitted with a history of progressive left leg pain and ulcerations. Several hemoragic bullae were present in these areas as well, and the patient offered that the ulcers began as blisters that then broke down. His past medical history included RA and he had been using steroid in doses of 5-40 mg/day according to his complaints for 20 years when he was admitted to our hospital. His examination revealed destructive and erosive arthritis affecting his bilateral wrists, MCP, and PIP joints, and ischaemic necrosis in the distal parts of the 2nd and 3rd digits of the left hand. Laboratory results were as follows: erythrocyte sedimentation rate 77 mm/h, urea 9 mg/dl, creatinine 0.8 mg/dl, calcium 7 mg/dl, phosphate 3.7 mg/dl, albumin 2 gr/dl, parathyroid hormone 43 pg/ml (n = 12-72), protein S 38% (n = 7-115%). Roentgenogram of the lower aspect of the leg showed calcified larger vessels and subcutaneous vessels. His lesions progressed and required above the left knee and left 2nd, 3rd digits amputations. Pathologic examination showed extensive arterial wall calcification. Now he has