months); and (5) antecedent term pregnancy.[6] In addition, Lurain and associates[15] noted that there are three factors primarily responsible for treatment failure: extensive disease, inadequate initial treatment, and failure of the chemotherapy protocol used.

Our case demonstrated several poor prognostic factors. The patient's clinical disease, dated from her molar pregnancy, was more than 15 months in duration. The patient had extensive disease at presentation with a large primary uterine lesion, bilateral diffuse lung metastases and bilateral multiple choroidal metastases. Despite prompt diagnosis and institution of combined chemotherapy and radiotherapy, her serum βhCG levels were massively increasing and the disease progressed rapidly. Following evacuation of the molar tissue it is recommended that patients require weekly βhCG determinations until the βhCG titre is within normal limits for 3 weeks. The titres are observed at monthly intervals for 6 months and then every 2–3 months for a further 6 months. Those patients who continue to show a plateau or rise in titres require chemotherapy.[6] In our case the disease was not recognised early because the patient had not been followed-up as recommended.

In conclusion, we report a case of metastatic uterine choriocarcinoma in which bilateral choroidal metastases were the initial manifestation of choriocarcinoma. Although choriocarcinoma is a rare cause of choroidal metastasis, it should be considered in the differential diagnosis in a young woman with choroidal metastases.

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Sir,

Central retinal artery occlusion associated with primary antiphospholipid syndrome

Antiphospholipid syndrome is a newly recognised entity. The clinical manifestations of this syndrome include hypertension, chronic renal failure, recurrent spontaneous abortions and deep vein thrombosis.[1] These manifestations are caused by arterial and venous thrombosis due to anticardiolipin antibodies, which may be the primary abnormality (primary antiphospholipid syndrome) or part of other systemic disease such as lupus erythematosus (secondary antiphospholipid syndrome).

As many as half of the patients with primary antiphospholipid syndrome may experience a decrease in visual acuity.[2] The funduscopic findings in primary antiphospholipid syndrome have recently been reviewed[2] and include retinal venous hypertortuosity, choriocapillary and retinal capillary dropout, vitreous haemorrhage or bands, serous macular detachment and optic disc oedema. Ischaemic optic neuropathy has also been described.[3] These findings were attributed to capillary vaso-occlusive disease and not to arterial occlusion and differ somewhat from the findings in secondary antiphospholipid syndrome, in which retinal arterial and vein occlusions may be quite frequent. We describe central retinal artery occlusion (CRAO) as a previously unreported complication of primary antiphospholipid syndrome.

Case report

A 50-year-old Caucasian woman presented with an acute decrease in vision in her right eye that deteriorated over approximately 48 h. Visual acuity was counting fingers from 1 foot on the right and 20/80 on the left. There were nuclear cataracts in both eyes. The right fundus demonstrated retinal oedema, cherry-red spot and 'kickboxing' of the retinal arterial flow. The left fundus appeared normal. Since the symptoms had been present for a little less than 48 h the patient was treated with ocular massage, sub-lingual isosorbide dinitrate, intravenous acetazolamide 500 mg, parenteral glycerol 50% 1 mg/kg, anterior chamber paracentesis, retrobulbar tolazoline and intravenous methylprednisolone 500 mg followed by streptokinase 750 000 units, but vision remained unchanged. Concomitantly, she was treated with coumadin 2.5 mg/day.

Two years earlier the patient had been diagnosed as having primary antiphospholipid syndrome. She suffered from systemic hypertension, chronic renal failure, deep vein thrombosis, ulcus cruris, thrombocytopenia, seizure disorder and had a history of 11 consecutive spontaneous abortions followed by 2 normal deliveries. On admission, the coagulation profile showed a prolonged partial thromboplastin time (52 sec), IgG anticardiolipin antibodies and positive lupus anticoagulant. Serum creatinine was 4.1 mg% and urea 122 mg%, indicating impaired renal function. Plasma cholesterol was 230 mg% and triglycerides were 186 mg%. Other serial laboratory tests were negative for antinuclear and anti-DNA antibodies, VDRL and HbS antigen. Serum protein electrophoresis was normal. Echo Doppler sonography of the carotids and echocardiography were normal. However, magnetic resonance imaging of the brain demonstrated multiple cortical infarcts around the tentorium and in the left thalamus, and cortical atrophy. When CRAO occurred the patient was under treatment with nifedipine 20 mg b.i.d., benazepril HCl 2.5 mg 1.q.d., pentoxifylline 400 mg t.i.d., allopurinol 100 mg 1.q.d., frusemide 40 mg 1.q.d. and slow-Fe 160 mg 1.q.d. Currently the patient also takes dipyridamole 75 mg t.i.d. and lovastatin 20 mg 1.q.d., and her blood pressure is well controlled.

Comment

The ophthalmic findings described in primary antiphospholipid syndrome have been attributed to microemboli, initiated by circulatory immune complexes. In our patient they may have occurred in a larger vessel, the central retinal artery, without any other funduscopic findings. Demonstration of multiple brain infarcts by magnetic resonance imaging suggests that these are also a result of multiple emboli or thrombi; and these findings in undiagnosed patients should raise the suspicion of other possible systemic disorders such as systemic lupus erythematosus (as part of secondary antiphospholipid syndrome), Sneddon's syndrome and Susac's syndrome.[4] All these clinically different entities may eventually be reclassified due to different underlying defects.

Although streptokinase may disintegrate the thromboemboli, the time interval between the occlusion and treatment of the widespread coagulation disorder was probably the cause for the irreversible visual loss.

Several factors, including systemic hypertension and hyperlipidaemia, may have caused CRAO in this patient; however, the underlying disorder for these findings is primary antiphospholipid syndrome. We suggest that primary antiphospholipid syndrome should be considered as a rare cause for retinal artery and vein occlusions, especially in young patients and patients with history of thrombotic events. A complete coagulation profile should be obtained for these patients to rule out other coagulation disorders, including secondary antiphospholipid syndrome, protein C and protein S deficiency,[5] since these entities share a common final pathway causing thromboembolic events.

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Sir,

Posterior dislocation of Staar plate haptic silicone lenses following Nd:YAG capsulotomy

We present three cases of delayed spontaneous posterior dislocation of Staar plate haptic silicone lenses following Nd:YAG capsulotomy. All patients underwent elective phacoemulsification with continuous circular