The basal ganglia sends fibres to the ipsilateral cortex to modulate voluntary motor activity of the contralateral limbs. The fibres to the cortex and the returning corticospinal tracts both pass through the internal capsule which, in turn, is close to the caudate nucleus. We cannot exclude the possibility that involvement of either of these fibre pathways is responsible for the prevention of the tardive dyskinesia. However, we consider these possibilities to be far less likely since there was no sensory deficit and the left hemiparesis was minimal. We consider the prevention of tardive dyskinesia to be more likely due to the caudate infarction visible on CT scan (fig). Tardive dyskinesia is thought to be caused by hypersensitive postsynaptic dopaminergic receptors on the neostriatum (caudate and putamen) portion of the basal ganglia. Eliminating the receptors before they have a chance to become hypersensitive would be expected to prevent tardive dyskinesia.

The upper motor neurons comprise the final common pathway for all motor movement. The second cerebrovascular accident shows that their destruction can suppress an already existing tardive dyskinesia. While all the right hemi-humeral dyskinetic movements were orginally suppressed by the CVA, they returned with greater severity in the leg as it was less affected by the stroke.

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References

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Chorea and the lupus anticoagulant
Sir: Chorea is a well recognised but rare accompaniment of systemic lupus erythematosus (SLE) and may occasionally be the only neurological feature. The pathogenesis is obscure but recently an association with the lupus anticoagulant has been suggested. I report the case of a young woman with acute chorea in whom the presence of the lupus anticoagulant was the only serological abnormality. She had the other complications commonly associated with the anticoagulant: recurrent spontaneous abortion, thromboses and mild thrombocytopenia, but none of the classic features of SLE. Her chorea stopped after the introduction of low dose aspirin.

A 22 year old white Caucasian was admitted to the Radcliffe Infirmary, Oxford, on 2 September 1985 for investigation of chorea. One month prior to admission she had developed right-sided involuntary movements which started in the hand but rapidly progressed to involve the whole of the right side. Her speech had become slurred. One week prior to admission, her mother had noted left-sided involuntary movements of which the patient was unaware. There were no other neurological or psychiatric symptoms.

In November 1983, she had an incomplete abortion at 12 weeks gestation and in December 1984 a missed abortion at 22 weeks. In January 1985, she had been admitted to another hospital with an acute anterior myocardial infarction. This was complicated by a life threatening pulmonary embolus. A coronary angiogram 6 months later showed stenosis of the left anterior descending artery, thought to be the result of a recanalised thrombus.

There was no family history of chorea, dystonia, psychiatic, rheumatological or vascular disorders. She smoked 20 cigarettes per day. She was on no medication and had never received an oral contraceptive pill.

On examination, she was generally fit and well. There were no rashes, arthropathy or lymphadenopathy. She was in sinus rhythm, blood pressure was 110/55 mm Hg. Her heart was not enlarged, but there was a soft systolic murmur compatible with mild mitral regurgitation secondary to papillary muscle dysfunction. Bed-side testing for higher mental function was normal. She was mildly dysarthric. Her gait was steady, but on walking she had wild gyrations of the arms. Choreiform movements of all the limbs, head and face were present at rest more marked on the right side. She was unable to maintain tongue protrusion. Cerebellar movements and the remainder of the cranial nerves were unremarkable. Power, tendon sensation in the limbs were normal, all deep tendon reflexes were present and the plantar responses were flexor.

The following investigations were all normal: urea and electrolytes, creatinine, liver function tests, calcium and phosphorous, glucose, blood urea, thyroid function tests, cardiolipin, serum and urinary copeptidase and fasting cholesterol and triglycerides. Her haemoglobin 11.7 g/dl. White cell count 6.3 x 10^9/l with a normal differential. Platelets 150 x 10^4/l. ESR 30 mm/hour. Prothrombin time ratio (PT) 1.2, kaolin clotting time (KCT) 83 seconds (normal 64 seconds). Kaoion cephalin clotting time 52 seconds, (normal less than 40 seconds); attempted correction with 1:1 patient and normal serum only reduced the kaolin cephalin clotting time to 50 seconds, indicating the presence of an inhibitor rather than clotting factor deficiency. The thrombin time, aPTT, thrombin III and factor VIII levels, and fibrinogen titre were normal. Anti-nuclear and double stranded DNA antibodies negative. Cardiolipin antibody (VDR1) positive but specific syphilis tests negative. Coagulation levels C3 92 mg/dl (69-130), C4 47 (12-27). Creative protein <0.6 mg/dl (<0-8). Chest radiographs and CT scan of the brain were normal. Electrococardiograms showed evidence of an old full thickness

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anterosetal infarction. Urine microscopy was normal. The CSF contained no cells, protein 0.35 g/l, Ig/albumin ratio 21%, oligoclonal bands negative. Following the discovery of the lupus anticoagulant she was commenced on aspirin 300 mg a day and within a few days the chorea stopped completely. She was discharged from hospital and at follow-up 6 months later she remained free of involuntary movements and had developed no other symptoms or signs.

Chorea has long been recognised as a complication of SLE.1-5 Lasins et al in 1975, reported three cases and were able to review 31 cases culled from the world literature.9 Necropsy studies have failed to reveal any constant pathological change in the basal ganglia and the most consistent finding has been of a diffuse cortical encephalomalacia with microinfarction secondary to thrombosis of small arterioles, often with little or no accompanying inflammatory change.1,4 These observations led to the belief that chorea in SLE is not related to lesions in specific sites, but rather that "multiple strategically placed lesions" throughout the brain can result in dyskinesia by releasing motor activity from higher control.3 However, the cause of the underlying infarction remained obscure. Recent work has suggested that an antibody, erroneously called the lupus anticoagulant, may be responsible for many of the vascular and other complications of SLE, including chorea. Furthermore, it now appears that three seemingly unrelated clinical features of SLE (recurrent venous thrombosis, recurrent abortion and central nervous system disease) share this common pathogenic mechanism.6,7 The finding in our patient of these complications together with lupus anticoagulant, but without other serological or clinical evidence of SLE adds further weight to the proposed association.

The lupus anticoagulant is an immunoglobulin, either IgG or IgM, that reacts with platelet wall phospholipids (platelet factor III) and inhibits the generation of prothrombin activator complex. The hallmark of its presence is prolongation of phospholipid-dependent coagulation tests which fail to correct after mixture with normal plasma. The best test for its detection is the kaolin clotting time;7 the prothrombin time is usually normal. A biological false positive test for syphilis may be a pointer to the presence of the antibody.6,5,9 Although the anticoagulant is identified by its in vitro heparin like affect its most important clinical implications are concerned with thrombosis, either venous or arterial.8-10 There is highly significant association between the presence of thrombotic complications in SLE and related connective tissue disease and the presence of the lupus anticoagulant.10 The mechanism by which it induces thrombosis is still uncertain.

Bouchez et al have suggested a specific association between chorea and the anticoagulant in patients with systemic lupus.5 They reported three young women with SLE all of whom developed chorea and had a circulating anticoagulant. In their comprehensive review of the literature they found 41 cases of SLE and chorea; nine (22%) had evidence of a circulating lupus anticoagulant and 17 (42%) had a biological false positive syphilis serology. All together 25 (60%) had evidence of a antiphospholipid antibody (positive syphilis serology or circulating anticoagulant). They point out that many of the cases were reported prior to the discovery of the anticoagulant and in the more modern reports the finding of positive antibodies was much more consistent. They propose a direct link between the lupus anticoagulant and the development of chorea. In view of the known thrombotic tendency of such patients and the reported histopathology in cases of chorea and SLE it seems probable that this plays a major part in the pathogenesis of chorea.

The case reported seems to be unique. She had all the complications associated with the anticoagulant: recurrent abortions, arterial and venous thrombosis, and thrombocytopenia, together with chorea, but had no other serological or clinical features of SLE. I suggest that the lupus anticoagulant should be looked for in all unexplained cases of chorea, especially when any of its clinical associations are present.

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