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Arg296 to Cys296 polymorphism in exon 6 of cytochrome P-450-D26 (CYP2D6) is not associated with multiple system atrophy

An allelic association between mutant alleles of the cytochrome P-450-D26 (CYP2D6) and Parkinson's disease has been shown by several groups. The analysis of these mutant alleles among patients with multiple system atrophy showed no difference in the frequency of these alleles from that in control subjects.

Ataxic hemiparesis with bilateral leg ataxia from pontine infarct

Ataxic hemiparesis is a syndrome characterised by weakness and cerebellar-like ataxia on the same side of the body. A lesion resulting in ataxic hemiparesis must involve both the corticospinal fibres and the afferent or efferent cerebellar fibres in locations where the tracts are in close proximity. The afferent and efferent cerebellar fibres form a loop extending from the cerebral cortex to the medullary regions of the pons, through the corticopontine peduncle to the cerebellar cortex and then extending from the dentate nucleus through the superior cerebellar peduncle, red nucleus, and thalamus back to the cerebral cortex. Ataxic hemiparesis has been associated with lesions in the corona radiata, thalamus, midbrain, and pons. Fisher and Cole first reported that a paramedian infarct of the basis pontis located at the junction of the upper one third of the pons with the lower two thirds could produce a contralateral ataxic hemiparesis. One of the major questions concerning pontine ataxic hemiparesis is why the limb ataxia is more often contralateral to the lesion and not bilateral. The corticopontine fibres terminate by synapsing with the pontine nuclei and motor fibres then cross the midline to enter the contralateral midbrain cerebelar peduncle. A basis pontis infarct might thus be expected to produce bilateral limb ataxia because it would involve ipsilateral pontine nuclei and corticospinal fibres as well as pontocerebellar fibres that have crossed from the contralateral side. We report a case of a mid-pontine paramedian infarct with caudalateral extension resulting in ataxic hemiparesis with bilateral leg ataxia.

An 80 year old white man with a history of coronary artery disease suddenly noticed left sided weakness. On examination, he had
A blood pressure of 120/72. He had a left central facial weakness and a mild left hemiparesis affecting the arm and leg equally. Incordination of the left upper extremity was present on finger to nose testing and bilateral heel-knee-shin ataxia was present, worse on the left. The muscle stretch reflexes were normal and the plantar reflexes were flexor bilaterally. Brain MRI disclosed an area of increased signal intensity involving both the paramedian mid-pons and extending laterally in the caudal third of the pons (fig 1).

It is unclear why patients with ataxic hemiparesis usually have ataxia contralateral to the lesion rather than bilateral limb ataxia. It has been suggested that the pontine nuclei are more vulnerable to ischaemia than the transverse pontocerebellar fibres crossing from the contralateral side but there is no histological evidence for this.2 Huang and Chang2 suggested that the crossing transverse pontocerebellar fibres take an oblique downward course to the contralateral middle cerebellar peduncle and in this way a more rostrally placed pontine lesion may involve only the corticopontine fibres and pontine nuclei ipsilaterally, but miss the more caudally placed crossing pontocerebellar fibres. A more caudalateral lesion would be expected to give rise to ipsilateral ataxia (fig 2). Our case supports this suggestion, as the lesion extended more laterally and caudally than previously described pontine infarcts associated with ataxic hemiparesis. The more caudalateral part of the infarct in our patient may have damaged the crossed pontine fibres and have resulted in the leg ataxia ipsilateral to the infarct, whereas the more medial and rostral part may have given a contralateral atactic hemiparesis (fig 2). This suggestion is supported by Fisher’s report of a lower lateral pontine infarct1 and Fisher and Tapia’s report of a lateral medullary infarct extending into the lower lateral pons.6 Both these infarcts gave rise to ataxia only ipsilateral to the lesion.

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3 Nakatane H, Fukuyama H, Abiguchi I, Kameyama M, Nishimura K, Torizuka K. Pontine atactic hemiparesis studied by a high resolution magnetic resonance imaging sys-

Autoimmune chronic active hepatitis and polymyositis in a patient with myasthenia gravis and thymoma

Myasthenia gravis is known to occur with other autoimmune diseases. It is rarely associated with polymyositis,1 and very exceptionally with autoimmune chronic active hepatitis.2 We describe a patient with generalised myasthenia gravis, cortical thymoma, polymyositis, and autoimmune chronic active hepatitis.

A 25 year old Chinese woman presented with intermittent weakness of the limbs for one month with no diplopia, speech disturbance, or bulbar symptoms. She had mild ptosis of her left eye which increased progressively with maintained upward gaze, and proximal limb muscle weakness. The rest of the examination was normal. She had a positive edrophonium test, and serum anti- acetylcholine receptor and antistriated muscle antibody titres. Chest radiography disclosed a mediastinal mass which was subsequently confirmed by CT to be a thymoma measuring 4.2 cm (anteroposterior) × 3.1 cm (width) in the left lobe of the thy-