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Arg296 to Cys296 polymorphism in exon 6 of cytochrome P-450-2D6 (CYP2D6) is not associated with multiple system atrophy

An allelic association between mutant alleles of the cytochrome P-450-2D6 (CYP2D6) and Parkinson’s disease has been shown by several groups 5,6. The analyses used in exon 4 and 5 of the CYP2D6 gene, homoygous CYP2D6 genotype, and the allele frequency in the European population. The autoantibodies were a key in the diagnosis of this disease.

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 pons and then to the cerebellum, where the fibres cross to the opposite side of the brain. The afferent fibres then return to the cerebral cortex via the thalamus and hypothalamus. The efferent cerebellar fibres extend from the tectum of the midbrain to the cerebellar cortex and then extend from the dentate nucleus through the superior cerebellar peduncle to the cerebellum cortex and then extend 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 contralateral to the lesion and not bilateral. The corticofugal fibres terminate by synapsing with the pontine nuclei and most fibres then cross the midline to enter the contralateral middle cerebellar peduncle. A basis pontis infarct might thus be expected to produce bilateral limb ataxia because it would involve ipsilateral pontine nuclei and corticopontine fibres as well as ponsocerebellar fibres that have crossed from the contralateral side. We report a case of a mid-pontine paramedian infarct with caudalateral extension resulting in atactic 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
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