The neurobiology of TS involves frontal subcortical circuits and neurotransmitter systems. The usefulness of antipsychotics in the treatment of TS has been documented, but is often associated with incapacitating side-effects. There are a few case reports of Tardive dyskinesia (TD) in TS after treatment with typical antipsychotics unlike with atypical antipsychotics. We discuss the case of a 24-year-old male who first exhibited repetitive head and neck twisting movements that started at 15 years of age. Nine years after the onset of symptoms when a definitive diagnosis of TS was made, his symptoms included motor and phonic tics [Yale Global Tic Severity Scale (YGTSS) = severe] as well as obsessional thoughts [Yale-Brown Obsessive Compulsive Scale (Y-BOCS) = mild]. There was no movement other than tics. His biochemical and neuromaging tests were normal. Initially he was treated with oral sertraline 50 mg and haloperidol 0.5 mg daily, but was lost to follow-up and reported for treatment after a year with identical symptoms. Oral risperidone was started as well as maintained at 1 mg with dinner and fluoxetine was increased to 40 mg with breakfast. Although his tics were minimal at this dose (YGTSS = mild), he developed severe TD of the lower jaw [oro-mandibular dyskinetic movements) after four months of treatment [Abnormal Involuntary Movement Scale (AIMS) rating = severe]. This movement was entirely different from the initial symptoms both anatomically and phenomenologically. Following this, risperidone was stopped and patient was started on vitamin E and clonazepam. In about 45 days there was significant improvement in the dyskinetic movements (AIMS score = mild) but the TS worsened significantly causing severe distress (YGTSS = severe). Risperidone has a higher affinity for 5-hydroxytryptamine 2A receptors and lower dopamine D2 receptor binding than haloperidol. As the 5-HT2A receptor has been implicated in the pathophysiology of TS, risperidone may be of theoretical benefit in TS, especially if the patient has OCD/OCS where 5-HT has been implicated. Several reports illustrate efficacy and lack of extrapyramidal side-effects in TS patients treated with risperidone. To our knowledge, this is the first report of TD complicating the use of risperidone in the treatment of TS. We wish to highlight the need for awareness among clinicians and patients about the potential risk of getting new movement disorders during the course of treatment with risperidone for tic disorders.
Primary stenting in acute carotid dissection

Sir,

A 49-year-old man presented to us with aphasia and right hemiplegia of 6 h duration. On examination he had a gaze preference to the left side, global aphasia and dense right hemiplegia with Grade 0/5 power. He had no history of trauma or risk factors for stroke. Computerized tomography (CT) scan of brain showed a hyperdensity in the left sylvian fissure suggestive of thrombosis in middle cerebral artery (MCA) branches [Figure 1]. He was taken up for digital substraction angiography (DSA) which showed a tapering occlusion of the left internal carotid artery (ICA) 3 cm after the origin with absent intracranial flow and very poor collateral flow via the contralateral ICA at 6 h. In view of the mechanical occlusion and the elapsing of the time window for intra-arterial thrombolysis, primary stenting was offered as an option. A microcatheter and microwire were passed through the true lumen of the ICA and a single tapering self-expanding nitinol 8>6 × 30 mm Protégé stent was deployed across the lesion by 7 h after the onset of stroke. Post-deployment angiogram showed excellent recanalisation of the ICA with filling of the left anterior cerebral artery (ACA) and superior division of the MCA [Figure 2]. Further thrombolysis of the inferior division was not attempted. Diffusion weighted magnetic resonance imaging (DWMRI) on Day 3 showed only a periventricular infarction in the left MCA territory [Figure 3]. CT angiography on Day 3 demonstrated the stent in situ with recanalisation of

References


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