was achieved. Dynamic CT scan revealed adequate reduction of the atlantoaxial rotatory subluxation. However, at the end of two weeks, she returned with torticollis despite wearing the cervical collar. She was again treated with Gardner-Well’s traction for two weeks followed by cervical collar for three months. At 42 months follow-up, she was asymptomatic.

Rotatory atlantoaxial subluxation is an uncommon and poorly understood clinical entity. Various conditions which can predispose to rotatory subluxation include inflammation, and surgical procedures in and around the throat, neck and cervical spine. The physiological laxity of the ligaments around the CV junction may be the underlying predisposing factor for rotatory subluxation. The presence of a torticollis in a child should arouse suspicion of rotatory atlantoaxial subluxation, particularly when there is a recent history of recent throat infection or trauma.

Fielding and Hawkins classified atlantoaxial rotatory fixation into 4 types depending on the degree of atlantodental distance. In Type I, the rotatory subluxation is associated with normal atlantodental interval. In Type II and Type III, the rotatory subluxations are associated with 3 to 5 mm and more than 5 mm of anterior displacement of the atlas respectively. Type IV rotatory subluxation is associated with posterior shift of the atlas due to failure of the dens. The Type I variety is the commonest and the most benign form of rotatory subluxation, probably because the transverse ligament is intact. The diagnosis of atlantoaxial rotatory subluxation requires a high degree of suspicion based on the signs and symptoms. A CT scan of the atlantoaxial complex is most valuable for demonstrating the anatomy and a 3D-CT scan provides more direct visualization of the abnormal anatomy. When diagnosed early, most patients respond well to a conservative treatment with cervical collar and bed-rest. Surgical intervention is advised when conservative treatment fails to achieve reduction or is followed by a recurrence of the deformity.

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References

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Nadroparin in acute ischemic stroke

Sir,

I read with interest the recent article by Sarma GRK et al. Based on a randomized controlled trial, they conclude that the outcome of stroke is significantly better in the group receiving nadroparin and aspirin versus the group receiving aspirin alone. However, I would like to make certain comments.

Firstly, data regarding the incidence of recurrent or progressive infarction during the follow-up period has not been provided. It is well known that both aspirin and heparin are mainly useful in preventing either re-infarction or progressive infarction. Moreover, in previous studies, a similar reduction in the rate of re-infarction or progressive infarction was seen with low molecular weight heparin (LMWH) and aspirin. Therefore, anticoagulation with LMWH, in order to prevent new infarction or worsening of neurological status, is not recommended in the early management of stroke.

The second issue is regarding the risk of hemorrhagic complications. Sarma GRK et al noted upper gastrointestinal bleeding in one patient (5%) in the LMWH group (requiring withdrawal of nadroparin) as compared to none in the aspirin group. Other investigators too have observed a significantly higher risk of bleeding with LMWH as compared to aspirin.

Thirdly, Sarma GRK et al draw an analogy between nadroparin and recombinant tissue plasminogen activator (rtPA) and advocate an early initiation of the former. However, it should be noted that rtPA is the only proven therapy for restoring or improving cerebral perfusion till date and nadroparin does not have this ability. Therefore, it may be inappropriate to draw any comparisons between the two, as their roles in treatment of acute stroke are entirely different.

Finally, there are a few limitations as mentioned by Sarma GRK et al, such as small sample size (only 20 in each arm) and lack of follow-up beyond three months, which further limit the usefulness of this trial. Moreover, studies with a larger sample size (> 1000 patients) and longer follow-up (six months) have failed to demonstrate any significant benefit with LMWH over aspirin.

In conclusion, data provided in this study is inconclusive and does not instill enough confidence to make a change in the current practice of avoiding combination of LMWH and aspirin in the treatment of acute ischemic stroke.

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Olanzapine for delirium in parkinsonism: Therapeutic benefits in lieu of adverse consequences

Sir,

Drug-induced (dopaminomimetic) psychosis and confusional states (i.e. delirium) are found to occur frequently in parkinsonism.1 Delirium is generally managed by the withdrawal of anticholinergic and dopamin agonist drugs, individually or concomitantly.2 If necessary, antipsychotics are instituted in low doses.2 Atypical antipsychotics (clozapine, olanzapine) though reportedly useful, are associated with delirium or confusion as an adverse effect due to intrinsic anticholinergic properties,1 mediated through muscarinic M1 receptors in the brain.1

A 31-year-old woman presented with history of obstructive hydrocephalus (due to aqueductal stenosis) 2-1/2 years back, which was successfully managed with a ventriculo-peritoneal shunt. There was a recurrence of symptoms, associated with delirium, about 1 year back. This was followed by the appearance of extrapyramidal symptoms in the form of bradykinesia, rigidity, and tremors. A diagnosis of post-anoxic parkinsonism was made. She was treated with a combination of 25/100 mg levodopa/carbidopa half tablet three times daily, and trihexyphenidyl 2 mg once daily. She improved but developed withdrawn and retarded behavior one month prior to our evaluation. Imipramine (a tricyclic antidepressant) 25 mg three times daily was added by an external agency, but this led to worsening of symptoms. On psychiatric evaluation she was found to be in delirium, diagnosed as per DSM-IV criteria, for a period of 2 weeks. She was initially managed by stoppage of all drugs except levodopa/carbidopa (25/100 mg), which was reduced from four tablets to half tablet three times daily. No clinical change in delirium with worsening of parkinsonism features (gross tremors leading to inability to swallow and sit or walk unsupported) necessitated hike in dose of levodopa/carbidopa (25/100 mg) to three times daily. Haloperidol, a preferred drug for treating delirium,4 and other typical antipsychotics were not initiated as they could have worsened the parkinsonism features.5 Clozapine was not started due to lowered level of consciousness and drooling of saliva secondary to the clinical diagnosis; these could have worsened due to the side-effect profile of clozapine. Risperidone (an atypical antipsychotic) could not be tolerated as, on initiation, at 1 mg/day there was marked drowsiness and increase in rigidity. Low-dose olanzapine (initiation dose = 1.25 mg once daily, maximum dose = 3.75 mg once daily), gradually titrated, led to resolution of delirium with no further worsening of motor symptoms over a three-week period. The patient thereafter maintained this delirium-free state over a follow-up period of eight weeks. During this time, no increased motor symptomatology was reported and none was detected on examination. Dopaminergic medications were not decreased. Thereafter, olanzapine was discontinued and she has maintained well for a period of twelve months.

Literature till date has mentioned the efficacy of olanzapine in treating dopaminomimetic psychosis, simultaneously cautioning about delirium5,6 and worsening of parkinsonism5,7 as its side-effects. Our patient did not develop worsening of motor symptomatology during treatment with olanzapine, a response that was maintained on follow-up too. The delirium also responded favorably.

Olanzapine demonstrates significant antagonism of serotonergic 5HT3 receptors leading to potentially cognitive-enhancing effects.6 It is also associated with differential antagonism of dopaminergic, histaminergic, cholinergic and serotonergic receptors in the brain. This differential antagonism of M1 and 5HT3 receptors could possibly explain the observed beneficial effect rather than a deleterious effect on the delirium in our case. To the best of our knowledge, this is the first case report in which olanzapine has been successfully used to treat delirium, an otherwise documented side-effect of the same.6 This patient’s experience also illustrates that failure of one atypical neuroleptic does not preclude success with a second atypical neuroleptic. Further studies are warranted to understand the biological basis of this therapeutic response so as to establish the potential of olanzapine as a possible useful agent for managing psychotic and confusional states in patients with parkinsonism.

References


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