Letter to Editor - Spectrum of Cerebral Venous Thrombosis

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Sir,

I read the original article “Spectrum of cerebral venous thrombosis in Uttarakhand” by Sunil Jee Bhat and Priyanka Vikas Kashyap in July 2018 issue of JAPI, with interest.† It was a good article bringing out their observations in septic and aseptic types of cerebral venous thrombosis (CVT). Though I agree with some conclusions there are many with which I disagree and suggest a change in the approach to this not uncommon problem.

The same condition is referred to as CVT and CVS in different parts of the same article. I think it was probably an editorial error and was not intentional. If not so, each term needs to be elaborated as to what it means.

All the references in the index article are before 2006 except one. The scenario of CVT is changing. Now-a-days we are seeing more number of male patients affected by the condition unlike what the statistics in the article show.2,3 And some such patients are alcoholics. There was no mention of alcoholic CVT in the article. In a prospective study done from our department including 50 patients of CVT (unpublished data), there were more number of male patients (62%) with CVT and nearly 10% of them were alcohol related.

Many articles in the references are about septic cavernous sinus thrombosis, and the discussion in the article also is mostly about treatment of that condition. In clinical practice and also in their own article (22:8) the authors noted that nonseptic cases are more common. Septic cases are less common and are often treated by critical care specialists, while nonseptic cases are treated by neurologists and physicians.

According to literature, among the female patients, oral contraceptive pill consumption is a commoner cause of CVT when compared to postpartum CVT,4 unlike in the index article where the postpartum CVT cases outnumber contraceptive pill related cases. It was not mentioned in the article how and when the prothrombogenic disorder profile was obtained. While evaluating such patients one must take care not to order such tests while the patient is on anticoagulants. Otherwise there can false results defeating purpose. I would order such tests when the patient had taken 3-6 months of oral anticoagulants, and stopped them for at least 10-15 days.

Corticosteroids cannot be recommended routinely in CVT patients to reduce vasogenic edema, and they may even promote thrombosis.4 The authors suggested that ‘anticoagulants have to be continued till clinical or radiological evidence of complete resolution’. They themselves mentioned that the role of anticoagulants is controversial in septic CVT cases. In nonseptic cases, it is common knowledge that clinical resolution will be complete in 10-15 days. Radiologically, the MR venogram will not be completely normal even after one year. Hence the authors’ suggestion about the duration of treatment is ambiguous.

In our department we had performed mechanical thrombectomy for refractory CVT in 4% cases and intrasinus thrombolysis in another 4% cases with good outcomes. The proper cases selection is important. Whenever the patient is not responding to adequate anticoagulation and the sensorium or the lesion size is/were worsening within the first 10 days after onset, intervention needs to be considered. There were no cases of deep venous system involvement in their series. Such cases are difficult to diagnosis, are associated with more mortality and morbidity and may present in a comatose state.

References