Strokes in the young account for nearly 30% of all cases of stroke in India and cerebral venous thrombosis (CVT) accounts for 10-20% of these cases. Two-thirds of them develop the same in the post-partum period. In the recent years, a number of inherited and acquired pro-thrombotic states have been identified. However, the exact etiology of CVT remains unidentified in a significant proportion of cases. The treatment approach of CVT has undergone significant re-appraisal, especially in the last decade, with the advent of novel strategies like selective sinus catheterization and thrombolysis. Current therapeutic options for CVT treatment include anti-thrombotic therapy with un-fractionated heparin, low-molecular-weight heparins (LMWH), oral anticoagulants, intravenous thrombolysis, local thrombolysis by selective sinus catheterization and a combination of thrombolysis and anticoagulation in addition to symptomatic therapy. Anticoagulation can prevent further thrombus formation and thereby prevent the progression of infarction and pulmonary embolism. However, in patients with hemorrhagic infarctions, worsening of bleeding was feared. Some authors recommended the use of anticoagulation only in patients with CVT without radiological evidence of hemorrhage, while most favor a more aggressive approach and recommend anticoagulation in all. The risk of hemorrhagic complications due to heparin has been over-estimated. A number of individual case reports, uncontrolled series, retrospective studies and randomized trials have demonstrated the efficacy and safety of heparin in CVT, even in the presence of hemorrhagic infarctions. A number of well-documented case reports described worsening of the clinical condition when heparin was replaced by oral anticoagulants and rapid improvement after restarting heparin. Diaz et al reviewed 203 CVT cases between 1942 and 1990 and compared the outcome of patients treated with and without heparin. Ninety-one percent survived in the first group compared with 36% in the second. Randomized trials demonstrated the safety of intravenous/sub-cutaneous heparin in patients with non-hemorrhagic and hemorrhagic venous infarctions. The results of the two prospective controlled trials at National Institute of Mental Health and Neurosciences support the use of even lower doses (15,000 units per day in three divided doses) of heparin in puerperal CVT. A more recent multicentric, double blind placebo controlled trial of nadroparin, a low molecular weight heparin, by de Bruijn and Stam randomized 59 patients to receive nadroparin and placebo for 3 weeks, followed by oral anticoagulation for 3 months (nadroparin group only). After 3 weeks, there was no difference in the outcome measured by death or Barthel index score. After 12 weeks, 13% in the nadroparin group and 21% in the placebo group had poor outcome. However, this difference was not significant, probably because of the small number of patients. The bulk of evidence favors the use of heparin in CVT, but the best indications for its use are not universally agreed upon. All would agree that heparin is indicated in patients with co-existent pelvic or deep leg vein thrombosis and pulmonary embolism or in conditions associated with prothrombotic states. Neonatal CVT is the only situation in which heparin has not been shown to improve the outcome and most authors do not recommend its use in this situation. Most of the studies have used higher doses of heparin to maintain activated partial thromboplastin time (APTT) 2.5 times the control. But, in puerperal CVT smaller dose (5000 unit 8th hourly- subcutaneous) is safe and effective and obviates APTT monitoring which is not available at peripheral centers.
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After the acute period, oral anticoagulation with warfarin is typically used for 1 to 3 months. The desired international normalized ratio is 1.4 to 2.8. Patients are then re-evaluated with MR imaging and MR venography to determine if flow is re-established. Prolonged anticoagulation therapy may be required for refractory cases or patients with underlying prothrombotic state. In patients with previous postpartum CVT, the chance of recurrence is 5% and the use of prophylactic doses of heparin in the post-partum period in the subsequent pregnancies may not be warranted.

Thrombolysis is an attractive option in the management of CVT but the indications for and the most efficacious technique for achieving thrombolysis remain uncertain. Various techniques of achieving thrombolysis include pharmacotherapy with urokinase or rtPA, mechanical thrombolysis and rheolytic thrombectomy.

Intravenous thrombolysis for CVT has been of value in a few case series, but data are limited. Local urokinase or rtPA infusion into the dural sinus thrombus has been reported to be successful in several patients and may be associated with a relatively lower risk of systemic hemorrhage. Frey et al administered combined intra-thrombus rtPA and intravenous heparin in 12 patients with CVT in an uncontrolled study. Pretreatment MRI showed subtle hemorrhage in four patients and obvious hemorrhagic infarction in three cases. A loading dose of rtPA was administered through the clot at 1 mg/cm followed by continuous intra-thrombus infusion at 1-2 mg/hour. Intravenous heparin was infused concomitantly. Flow was restored completely in six and partially in three with a mean rtPA dose of 46 mg (range, 23 to 128 mg) at a mean time of 29 hours (range, 13 to 77 hours). All the nine patients had symptomatic improvement. In one patient, treatment was stopped because no progress was made. In two patients, hemorrhagic worsening occurred. No patient died. Some workers successfully used mechanical wire micro snare maceration of the thrombus when pharmacological thrombolysis had failed to achieve patency of sinus. Another novel method is Angio-jet rheolytic thrombectomy, which involves the use of Bernoulli effect to create a vacuum that fragments and aspirates the thrombus. This has the added advantage of lack of hemorrhagic complications and a potential for use even in hemorrhagic CVT. Chow et al first used this technique in 4 patients with excellent results and this technique warrants further investigations. Transcranial doppler studies have been used to monitor the venous hemodynamics and collateral pathways in patients with CVT, but this method cannot be recommended for routine clinical use, due to its lack of sensitivity and specificity for diagnosis of CVT. Thrombolytic therapy appears attractive. However, in view of the proven efficacy of heparin, thrombolytic therapy should be reserved for only those patients who deteriorate despite adequate anticoagulation. Mechanical and rheolytic methods are, at present, in their infancy. Anti-platelet drugs have been advocated in CVT but have never been systematically studied. Furthermore, their use seems illogical since they are of no proven benefit in the acute treatment of venous thrombosis.

Symptomatic management of CVT includes attention to treatment of seizures, metabolic derangements, cerebral edema, and elevated intra-cranial pressure as dictated by the clinical situation. As far as anticonvulsants are concerned, some favor their routine administration, while others restrict them to patients with seizures. There is little evidence to suggest that one anti-epileptic drug is superior to the other. The optimum duration of the anti-epileptic therapy is not established, but one year of seizure free period seems reasonable. A number of approaches have been used in the treatment of raised intracranial pressure and include steroids, mannitol, glycerol, acetazolamide, lumbar punctures, shunting, surgical decompression, and even barbiturate induced coma. The choice among these methods depends on the clinical situation. If the level of sensorium deteriorates or if the headache is severe, mannitol is advisable. Surgical decompression will be required in the case of continuing deterioration, inspite of the maximum medical management. The treatment of CVT has to be aggressive as morbidity is minimal.

References