

Clustering of Cortical Venous Thrombosis in the Thar Desert of Western Rajasthan during Peak Summer Season

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Abstract

Cerebral sinous venous thrombosis(CSVT) or Cerebral venous thrombosis (CVT) is rare in comparison to arterial stroke and have acute, subacute to chronic presentation with varied clinical manifestations like headache, vomiting, seizure focal neurological deficit etc. Like wise it has diverse etiologies. Dehydration is also an important precipitating factor in ischaemic stroke. We describe a small study but clustering of CVT cases in last summer season of Thar Desert of western Rajasthan, India. In our experience of about more than two and half decade we never encountered such clustering of CVT cases in summer so reporting this study may serve guide in future plannings.

Introduction

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 [1]. Cerebral venous thrombosis (CVT) accounts for approximately

0.5-1% of all strokes [2]. Cerebral venous thrombosis (CVT) is presumed to be a rare venous stroke, which accounts for 0.5-3% of all stroke types. Dehydration infection, pregnancy are common risk factors in developing countries. Dehydration is known to cause hypercoagulability which precipitates various venous thromboembolic complications in acute stroke like venous thromboembolism, deep vein thrombosis (DVT). The changing climate is likely to have great impact on it as it has been studied in a recent multinational trials [3]. However there is paucity of studies focusing on direct effect of dehydration on precipitated by dehydration. Dehydration is a known and preventable risk factor thus clinicians should consider it as an important precipitating factor for ishemic stroke/CVT specially in hot areas like Thar desert of western Rajasthan during summer season.

Materials and Methods

This study was conducted over a period of three months may 2022 to july 2022. This was a retrospective and observational study. This study was conducted in the Department of Neurology at a tertiary care teaching center. All the patients with suspected symptoms had documented CVT on MR Venogram. All the patients were managed as per the standard protocols with IV fluids to correct dehydration, decongestive agents, low molecular weight heparin (LMWH) and anticoagulation etc. All patients underwent basic investigations, such as hemogram, electrolytes, blood sugar levels, renal function tests, and chest radiographs. Liver function tests, coagulation studies, inflammatory markers, and homocysteine levels were done in all patients however thrombophilia work could be done only in two patients because of financial constraints.

Results

The details of the patients is being given in Tabulated form. Of the 12 patients in our study, 66 % were males and 34% females. The most common presenting symptom was headache, diplopia and seizures. the risk factors were present in 33% patients mostly oral contraceptive pills (OCP) intake and alcoholism. Dehydration was a common factor in all the patients and in few precipitated by vomiting and alcoholism which in itself predisposes individuals to a prothrombotic state by inducing a state of dehydration and hyper viscosity and increasing platelet reactivity. The most common sinuses involved were transverse (left>right) 90%, sigmoid sinus 83% and superior saggital sinus in 41%.

S.NO	AGE	RISK FACTORS	SYMPTOMS	IMAGING	BASELINE
1	30/M		HEADACHE	Left transverse and sigmoid sinus thrombosis	NORMAL
2	20/M		HEADACHE AND VOM- ITING	LEFT TRANSVERSE , LEFT SIGMOID SINUS , SUPERI- OR SAGGITAL AND LEFT IJV THROMBOSIS	NORMAL

Table: The patient details in tabulated form

3	30/F	OCP	HEADACHE	Right transverse and Right sigmoid sinus and Right internal jugular vein thrombosis	NORMAL
4	19/F		HEADACHE DIPLOPIA	s/o left transverse and sigmoid sinus thrombosis.	NORMAL
5	26/M		HEADACHE VOMITING DIPLOPIA	s/o left transverse sinus throm- bosis	NORMAL
6	24/M	ALCHOLO- IC	HEADACHE LEFT HEMI- PARISIS	superior saggital and b/l trans- verse and sigmoid sinus s/o thrombosis	NORMAL
7	26/M		HEADACHE VOMITING SEIZURES	the left transverse and sigmoid sinus s/o thrombosis	NORMAL
8	47/F	ОСР	HEADACHE GIDDINESS	left transverse and sigmoid sinus thrombosis	NORMAL
9	48/M	ALCOHOL- IC HTN		SUPERIOR SAGGI- TAL,TORCULA,RIGHT TRANSVERSE,SIG- MOID,B/L SUPERIOR TRO- LARD VEIN	NORMAL
10.	20/M		HEADACHE SEIZURES	superior saggital and b/l trans- verse and sigmoid sinus s/o thrombosis	Thrombo- phillia panel normal
11	32/m		headache	s/o left transverse, sigmoid and straight sinus thrombosis.	Thrombo- phillia panel normal
12	50/F	ANEMIA	HEADACHE	POSTERIOR 1/3 OF SUPE- RIOR SAGITAL SINUS	IRON DEFI- CEN ANE- MIA

Discussion

Cerebral venous sinus thrombosis (CVT) predominantly affects young patients <50 years and women of reproductive age as compared to DVT/PE, which becomes more common with age and affects roughly equal numbers of men and women [1,2]. The Virchow triad which consists of stasis of the blood, changes in the vessel wall, and changes in the composition of the blood is a risk factor for venous thrombosis. Predisposing causes of CVT are multiple, are usually divided into acquired risks (eg, surgery, trauma, pregnancy, puerperium, antiphospholipid syndrome, cancer, exogenous hormones) and genetic risks (inherited thrombophilia) [4,5].

The Thar Desert of India receives ~80% of its annual precipitation from the southwest summer monsoon and the eastward migration of mid-latitude winter cyclones (originating over the west Asia and Mediterranean) contributes < 20% of the annual rainfall. In this region, the maximum temperature reaches up to 45-50 $^{\circ}$ C during summer months [6].

Dehydration has a high prevalence in ischemic stroke patients on admission, ranging from 29 to 70%. Dehydration leads to hematological disturbances like increased blood viscosity, high hematocrit and red cell aggregation [6]. Intracranial sinuses do not have valves. This allows the blood to circulate in both directions but may fail to ensure blood flow velocity in some cases and favors thrombosis. An increase in pressure in the venous system coupled with dehydration caused by sustained effort could reduce intracranial venous return, inducing a decrease in blood flow in the intracranial vein and triggering thrombosis. In CVT, thrombus development may be subacute and symptoms are delayed. Appropriate dehydration therapy might be beneficial to reduce the ischemic penumbra and improve cerebral perfusion [7-9].

A cohort with 182 stroke patients indicated that dehydration on admission might be associated with a higher risk of venous thromboembolism [8]. Although the underlying mechanism is not clear, dehydration status is known to reduce cerebral perfusion, decrease collateral circulation, increase blood viscosity, and cause hypercoagulability which require further large scale prospective study [10-12].

Drawbacks

There are two drawbacks of our study- there is no definite parameter to access dehydration and relatively small sample size so possibility of selection bias and amount of fluid received before baseline investigations as most patients receive fluids from periphery. Due to financial constraints we couldn't do thrombophilia work up however all the patients had a negative history of any previous vascular event or family history for any vascular event and baseline coagulation studies were normal.

Conclusion

It is important to establish the cause of CVT to determine optimal long term preventive treatment and help the patient avoid future thrombogenic conditions. However, in around 12% of cases the cause remains unknown. Dehydration is a known and preventable risk factor thus clinicians should consider it as an important precipitating factor for ishemic stroke/CVT in hot climate areas to avoid morbidity.

As because of climatic changes like global warming the seasons are taking extremes and unexpected turns so such reporting of an of course small experience will help plan future strategies so as to triage the prompt tackling of such situations, as it has been warned by the world bank [12].

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