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CEREBRAL VENOUS SINUS THROMBOSIS AMONG SUDANESE PATIENTS IN THE TERM OF CLINICAL PRESENTATION, RISK FACTOR & OUTCOME

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ABSTRACT
Back ground: The cerebral venous sinus thrombosis (CVST) is a rare origin of stroke worldwide. However, the mode of presentation and aetiology are various. The prognosis is better than arterial thrombosis. The age of the onset is early than arterial. Magnetic resonance imaging (MRI), MR venography (MRV) CT venography is currently the key diagnostic. Objectives: To estimate the incidence, risk factor, clinical presentation, and outcome, of patients with CVT during a period of the study. Also to study other parameters related to disease. Methods:We studied 70 cases collected in the department of neurology of the university hospital of Khartoum (Sudan). This study lasted 42 months. The cerebral-CT scan was performed in 32 cases (45.7%) while MRI examination was done in 68 cases (97.1%) and most patients (75.7%) received anticoagulation therapy. Results:The mean age of our patients was of26 years old, ranging between 13 years old and60 years old. Predominance of female was noticed (80%). The clinical presentations of the patients were dominated by the following profile: Headache in 60 cases 85.7%, Seizure in 37 cases 52.9%, Paresis in 31cases 44.3%, and Papilledema in 27 cases 38.6%. Diplopia in 26 cases 37.1%, disturbance of the level of consciousness in 22 cases 31.4%, Nausea/vomiting in 15 cases 21.4%, neck pain in 15 cases 21.4%, fever in 11 cases 15.4%, aphasia in 11 case 15.4%, sensory symptoms in 5 cases 7.1%, visual loss in 2 cases 2.9%, and mental disorder in one case 1.4% CVT associated to gynecological causes in 35 cases 50%, drugs used in 13 cases 18.6%, infection origin in 10 cases 14.3%, prothrombotic causes in 10 cases 14.3%, 3 cases 2.9% with vasculitis, 2 cases 2.9% with thyroid disease, and one case 1.4% for the following nephrotic syndrome, malignancy-solid tumor,and polycythemia. Optic nerve is the most cranial nerve involved in 25 cases 33.3% Followed by Abducent nerve in 19 cases 27.4%. The superior sagital sinus affected in 47 patients 67.1%, Lt Transverse sinus affected in 40 patients 57.1%, Rt transverse sinus affected in 29 patients 41.4%. The evolution was good in 43 cases 61.4% minor squeals were observed in 14 patients 20% , while major squeals were observed in one cases1.4% and death of 12 cases 17.1% was also registered. Conclusion: **CVT is** pathology of good prognosis once the diagnosis is promptly performed early and anticoagulation treatments initiated with close monitoring and follow up and early surgical intervention if needed.

KEYWORDS: Cerebral Venous Sinus Thrombosis, Clinical Presentation, Risk Factor, Outcome and Sudan.

INTRODUCTION
Cerebral venous sinus thrombosis (CVST) is specific type of cerebrovascular disorder whose epidemiology has changed over the past few decades. Patients present signs of intracranial hypertension such as headache, vomiting, and papilloedema. Now today it is no longer regarded as a uniformly fatal disease, with early use of anticoagulation, and others surgical interventions. The international incidence of CVST is about 5 million/year and account about 0, 5%to1% of all strokes. CVST is not a rare disorder; the incidence was most likely underestimated before the advent of accurate noninvasive imaging methods, but it may have a differential geographic distribution worldwide with a higher incidence in the Asian world. It is more common among neonates, younger women and men, where hypercoagulable state, either acquired (eg cancer, antiphospholipid syndrome, and nephritic syndrome) or a genetic prothrombotic condition like protein S, and protein C deficiency may be present, and so that should be considered in all cases of young stroke and neurological syndromes in appropriate setting. Unfortunately, the clinical presentation of CVT is not specific, and patients may present with wide spectrum of signs and symptoms mimicking conditions as diverse as arterial stroke, brain tumours, encephalitis, meninogoencepgalitis and benign intracranial hypertension. The possible risk factors and clinical manifestations of this disorder are many and varied from person to anther and depend on many factors for example

the effect and side, size of the lesion on the cerebral hemisphere and the part of venous or sinuses which occluded so imaging plays a primary and major role in the diagnosis, for example Magnetic resonance (MR) imaging, unenhanced computed tomography (CT), unenhanced time-of-flight MR venography, and contrast material–enhanced MR venography and CT venography are particularly useful techniques for detecting cerebral venous and brain parenchymal changes that may be related to thrombosis, so to achieve an accurate diagnosis, it is important to have a detailed knowledge of the normal venous anatomy and variants, the spectrum of findings (CVST and recanalization, parenchymal diffusion or perfusion changes or hemorrhage), other potentially relevant conditions (deep venous occlusion, isolated cortical venous thrombosis, idiopathic intracranial hypertension), and potential pitfalls in image interpretation.

METHODOLOGY

Study area: This is prospective descriptive cross sectional, hospital based study, conducted at Soba University Hospital, which is a tertiary hospital, was established in 1975, for teaching the students of medicine and training medical cadres; in addition to providing treatment services for the communities.

Study Design: The consent was taken from the patient or the family when the patient is confused. The study was approved by the local ethics committee. The data were collected using carefully designed questionnaire. The informers were either the patients or close relatives. A full detailed history and proper systemic and neurological examination was performed to each patient by the authors. The following investigations were done for each patient: Urine analysis, complete blood count, erythrocyte sedimentation rate, renal function test, liver function test, CT brain, brain MRI and magnetic resonant venogravy. The rest of the investigations depend on the underlying causes of dural venous thrombosis e.g. sickling test, protein C, protein S, antithrombin 111 assessment, antinuclear antibodies, anti DNA antibodies and antiphospholipid antibody. All patients were closely followed up for early detection of complications and outcome using Rankin scale.

Study population: All adult Sudanese patients with dural sinus thrombosis who were admitted to the hospital and accepted to participate in the study period were enrolled.

RESULTS

This is a descriptive prospective hospital based study enrolled 70 Sudanese patients with CVST conducted at Soba University Hospital during the period of the study.

Regarding incidence

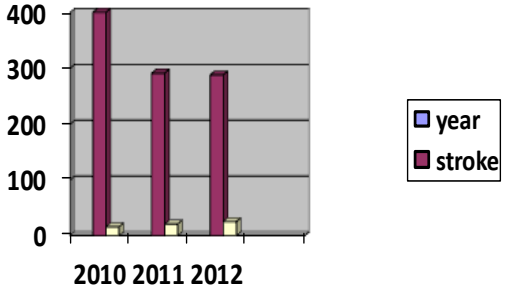

Figure. 1: Compare the incidence of CVT to stroke patients per year during the period of the study.

Regarding age & gender distribution

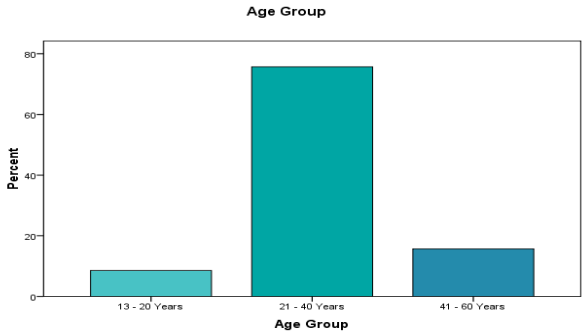

Figure. 2: Incidence of CVT according to the age of the patients.

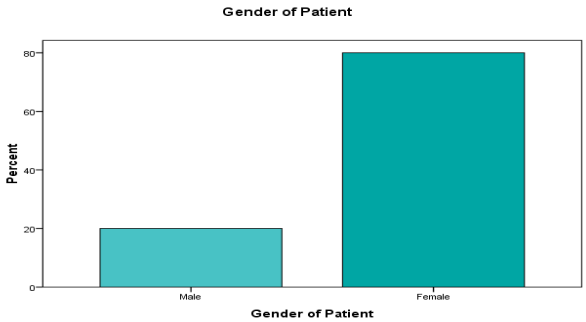

Figure. 3: incidence of CVT according to the gender of the patient.

Regarding occupation& address of patients

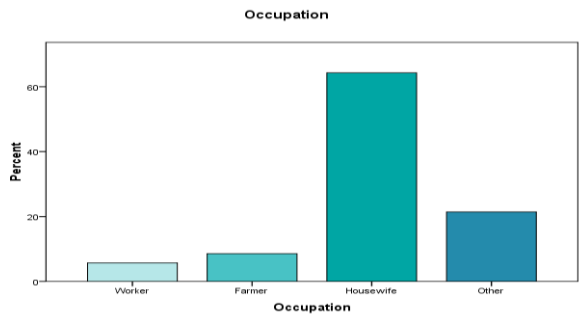

Figure. 4: incidence of CVT according to the occupation of the patient.

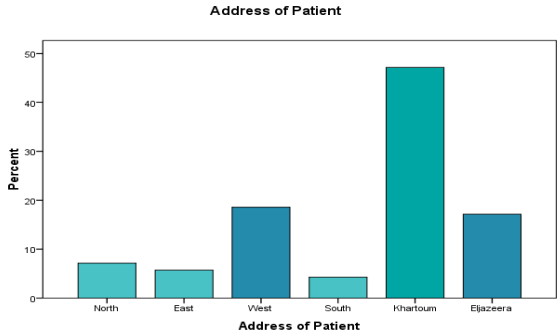

Figure. 5: Incidence of CVT according to the address of the patient.

Regarding symptoms and signs

Table. 1: Symptoms and signs of included patients.

Symptoms and signs	Frequency	Percent
Headache	60	85.7
Visual loss	2	2.9
Papilledema	27	38.6
Diplopia	26	37.1
Nausea/vomiting	15	21.4
Neck pain	15	21.4
Fever	11	15.4
Drowsy	8	11.4
Coma	14	20
Motor aphasia	7	10
Global aphasia	4	5.7
Mental disorder	1	1.4
Lt paresis	17	51.5
Rt paresis	14	42.4
Seizure	37	52.9
Sensory symptoms	5	7.1

Table. 2: Type of Headache of included patients.

Type	Frequency	Percent
Acute headache	21	35
Sub acute headache	39	65
Bilateral headache	53	88.3
Unilateral headache	2	3.3
Localized headache	4	6.7

Table. 3: Type of seizure of included patients.

Type	Frequency	Percent
Generalized	29	87.9
Seizure	4	21.1
Focal seizure	1	3
Other seizure	5	7.1

The main patterns of cranial nerve involvement are listed in Table [4].

Table. 4: Cranial nerve involvement in included patients.

Cranial nerve	Frequency	Percent
CN II R	25	65.8
CN II L	25	65.8
CN III L	1	2.6
CN VI R	9	23.7
CN VI L	10	26.3
CN VII R	4	10.5
CN VII L	7	18.4
CN IX L	1	2.6
CN X L	1	2.6
CN XI L	1	2.6
CN XII L	1	2.6

Regarding imaging features of included patients:The main patterns of imaging are listed in Table [5].

Table. 5: Imaging features on CT/MRI of included patients.

Imaging features	`Frequency	Percent
Left hemisphere infarct	8	11.4
Right hemisphere infarct	5	7.1
Left hemisphere hemorrhage	19	27.1
Right hemisphere hemorrhage	15	21.4
Posterior fossa hemorrhage	2	2.9
Bilateral parenchymal lesions	5	7.1
Unilateral parenchymal lesions	2	2.9
Subarachnoid hemorrhage	3	4.3

Regarding occluded sinus / vein:The main patterns of occluded sinus and vein are listed in Table [6].

Table. 6: Occluded sinus/vein of included patients.

Occluded sinus/vein	Frequency	Percent
Superior sagital sinus	47	67.1
Lt transverse sinus	40	57.1
Rt transverse sinus	29	41.4
bilateral transverse sinus	21	30
Straight sinus	6	8.6
Deep venous system	4	5.8
Cortical veins	1	1.4
Jugular veins	7	10
Cavernous sinus	3	4.3
Sigmoid sinus	18	25.7
Trolard vein	1	1.4
Confluence sinus	1	1.4

Regarding risk factors identified in included patients The main risk factors are listed in Table [7].

Table. 7: Risk factors of included patients.

Risk factor	Frequency	Percent
None identified	5	7.1
Thrombophilia	4	5.7
Acquired-antiphospholipid	6	8.6
Nephrotic syndrome	1	1.4
Solid tumor outside CNS	1	1.4
Polycythemia	1	1.4
Vasculitis SLE	2	2.9
Rheumatoid arthritis	1	1.4
Thyroid disease	2	2.9
Pregnancy	8	14.3
Puerperium	1	1.4
Postpartum	23	32.9
Abortion	3	5.4
CNC infection	7	10
Parameningeal infection	5	7.1
Oral contraceptives pills	7	10
Hormonal replacement therapy	2	2.9
Cytotoxic drugs	2	2.9
Steroid	2	2.9
Dehydrated	5	7.1

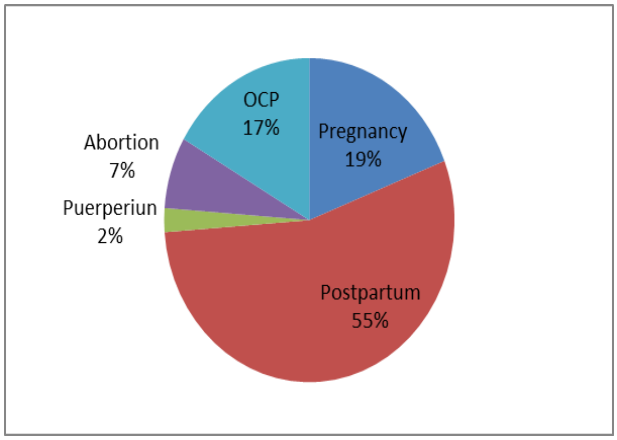

Figure. 6: The percentage of the Gynecological risk factors.

Regarding treatment of included patients: Table [8]

Table. 8: Treatment of included patients.

Type of treatment	Frequency	Percent
HMWH	5	7.1
LMWH	54	77.1
Oral anticoagulant	53	75.7
Anti -convulsion	34	48.6
Antibiotics	10	14.3
Steroid	14	20.0
Other treatment	11	15.7

Regarding outcome (modified RANKIN scale at discharge) of included patients: Table [9] Table [10] Table [11] and figure [7].

Table. 9: Disability (modified RANKIN scale at discharge).

Scale	Frequency	Percent
No Symptom	43	61.4
Minor Symptom not Interfering with Lifestyle	9	12.9
Minor Handicap	4	5.7
Moderate Handicap	1	1.4
Moderately Sever Handicap	1	1.4
Death	12	17.1

Table. 10: Outcome According to the Gender.

Rankin Scale	Gender		Total
	Male	Female	
No Symptom	6	37	43
Minor Symptom not Interfering with Lifestyle	2	7	9
Minor Handicap	0	4	4
Moderate Handicap	1	0	1
Moderately Sever Handicap	0	1	1
Death	5	7	12
Total	14	56	70

Table. 11: compare between Occluded sinus/vein& death.

Occluded sinus/vein	Frequency	Percent
Superior sagital sinus	8	66.7
Lt transverse sinus	7	58.3
Rt transverse sinus	6	50
bilateral transverse sinus	6	50
Straight sinus	1	8.3
Deep venous system	0	0
Cortical veins	0	0
Jugular veins	0	0
Cavernous sinus	1	8.3
Sigmoid sinus	2	16.7
Trolard vein	0	0
Confluence sinus	0	0

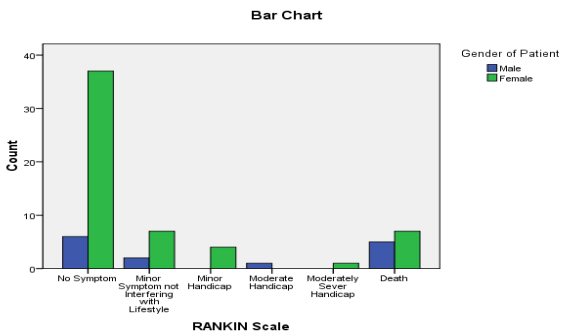

Figure. 7.

Patients' characteristics, signs and symptoms, risk factors, confirmation methods, imaging finding, treatment and outcome are summarized in Table [12].

Table. 12: Demographic, clinical and imaging features, risk factors, treatment and outcome in 70 patients with CVT.

Pt	Age	Sex	Cause	signs and symptoms	Confirmation Method	Imaging finding	Site of Thrombosis	Treatment	Out Come mRs
	35Y	F	Antiphosolipid SYNDROM	Headache papilledema	MRI MRV	========	SSS/ lt LTs/ Rt LTs / SS	LMWH Warfirin	0
	25Y	F	Antiphosolipid SYNDROM + pregnancy	Headache Papilledema Lt paresis Seizure	MRI MRV	Rt cerebral haemorrhage	SSS	LMWH Warfirin Lamotergn	1
	39Y	F	OCP	Lt paresis Seizure Aphasia	MRI MRV	Unilateral parenchymal lesion	Lt LTs	LMWH Warfirin Lamotergn	1
	24Y	F	OCP	Headache Neck pain Lt paresis Seizure	CT MRI MRV	Lt cerebral haemorrhage	SSS/ lt LTs / SS	LMWH Warfirin Lamotergn Steroid	0
	21Y	F	Voluntary abortion	Headache Seizure	CT MRI MRV	Rt cerebral infarct	SSS	LMWH Warfirin Lamotergn	0
	16Y	F	CNS and parameningeal infection	Headache Neck pain Diplopia Fever Papilledema	MRI MRV	Bilateral parenchymal lesion	SSS	LMWH Warfirin Lamotergn Steroid Antibiotic Antiviral	0
	35Y	F	Postpartum	Headache Aphasia Rt paresis Coma	CT MRI MRV	Lt cerebral haemorrhage and posterior fossa	SSS	LMWH Warfirin Lamotergn	4
	33Y	F	Protein S deficiency + pregnency	Headache Diplopia	MRI MRV	=====	SSS / Lt LTs	LMWH	0
	60Y	M	Nephritic syndrome + CNS infection	Coma Aphasia Fever	MRI MRV	Bilateral parenchymal lesion	Cavernous sinus	LMWH Steroid Antibiotic	6
	23y	F	OCP	Headache Papilledema Visual loss	MRI MRV	Bilateral parenchymal lesion	SSS	LMWH Warfirin	6
	35Y	F	CNS infection	Headache Neck pain Coma	CT MRI MRV	Rt cerebral haemorrhage	SSS / lt TLs/ Rt LTs	LMWH antibiotic Lamotergn	6

				Fever seizure					
12	29Y	F	Pregnancy	Headache Papilledema Lt paresis Diplopia	MRI MRV	Bilateral parenchymal lesion	SSS / lt LTs/ Rt LTs	LMWH Warfirin Lamotergn	0
	21Y	F	Postpartum	Coma seizure	MRI MRV	Rt cerebral haemorrhage	lt LTs/ Rt LTs	phenytoin	6
	50Y	F	Rheumatoid Arthritis +steroid+ cytotoxic drug	Rt paresis Seizure Aphasia	MRI MRV	Lt cerebral infarct	SSS	Warfirin Lamotergn	1
	25Y	F	Postpartum	Headache Papilledema Coma Seizure	CT MRI MRV	Bilateral parenchymal lesion	SSS / lt LTs/ Rt LTs / SS/Js/ Straight sinus	LMWH Warfirin Lamotergn	0
	21Y	F	Postpartum	Headache Neck pain Diplopia Papilledema	CT MRI MRV	====	SSS ltLTs/ Rt TLs/SS/ Js/ DvS	LMWH Warfirin	0
	26Y	F	Postpartum	Headache Diplopia	MRI MRV	Lt cerebral haemorrhage	SSS / lt LTs / SS	LMWH Warfirin	0
	38Y	F	OCP/HRT	Headache Seizure Aphasia Drowsiness	MRI MRV	Lt cerebral haemorrhage	SSS / SS	LMWH Warfirin Lamotergn	1
	13Y	F	SLE	Headache N/V Diplopia Papilledema	MRI MRV	Rt cerebral infarct	SSS	Steroid	6
	25Y	F	OCP	Headache Diplopia Papilledema	MRI MRV	======	SSS / lt LTs/ Rt LTs	LMWH Warfirin	0
	12y	F	Postpartum	Headache Papilledema Seizure	MRI MRV	Lt cerebral haemorrhage	SSS / lt LTs/ SS	LMWH Warfirin Lamotergn	0
	28y	F	Postpartum	Headache Papilledema Seizure	MRI MRV	Lt cerebral haemorrhage	SSS / lt LTs	HMWH Warfirin	0
	20y	F	CNS and parameningeal infection	Headache Fever Seizure	MRI MRV	Unilateral parenchymal lesion	lt LTs/ Rt LTs	LMWH Warfirin Antibiotic	2

				Lt paresis				Antiviral	
	42y	F	Antiphosolipid SYNDROM	Headache Coma	MRI MRV	Lt cerebral + Posterior foss haemorrhage	SSS / Rt LTs SS/Js/ straight	LMWH Warfirin	0
	30y	F	OCP	Headache Diplopia	CT MRV		SSS / lt LTs/ Rt LTs / SS/Js	HMWH Warfirin steroid	O
	47y	F	Voluntary abortion +HRT	Rt paresis Seizure Aphasia	CT MRI MRV	Lt cerebral haemorrhage	lt Ls	LMWH Warfirin Lamotergn	0
	26y	F	Pregnancy	Headache Diplopia Papilledema	CT MRI MRV	=====	SSS/ lt LTs/ Rt LTs /DVS	LMWH	0
	50y	M	Dehydration	Coma Lt paresis	CT	Rt cerebral haemorrhage	SSS	Antibiotic Antiviral	6
	20y	F	Postpartum	Headache Seizure Rt paresis	CT	Lt cerebral haemorrhage	SSS	Warfirin Lamotergn	0
	32y	M	Dehydration	Headache Seizure Lt paresis Drowsiness	MRI MRV	Rt cerebral haemorrhage	SSS / lt TLs/ RtLTs	HMWH Warfirin Lamotergn	0
	49y	F	CNS infection	Headache Coma Papilledema	MRI MRV	=====	SSS/SS	LMWH Antibiotic Antiviral	6
	32y	F	Antiphosolipid SYNDROM + Postpartum	Headache Seizure	MRI MRV	Lt cerebral haemorrhage	SSS	LMWH Warfirin Lamotergn	0
	32y	F	Non identified	Headache Coma Seizure	CT MRI MRV	Lt cerebral haemorrhage	lt LTs	LMWH Warfirin Lamotergn	0
	32y	M	Dehydration	Headache Neck pain Diplopia Papilledema Drowsiness	CT MRI MRV	====	SSS	LMWH Warfirin Lamotergn	0
	15y	M	Steroid	Headache Neck pain Diplopia N/V Visual loss	MRI MRV	====	SSS /Rt LTs	LMWH Warfirin Surgery	1
	24y	F	Pregnancy	Headache Neck pain Diplopia	CT MRI MRV	Rt cerebral haemorrhage	SSS /DVS	LMWH Warfirin Lamotergn	0

				N/V Seizure Lt paresis					
	36y	F	Hypothyroidism + Postpartum	Headache Rt paresis	CT MRI MRV	Lt cerebral haemorrhage	lt LTs/ troland Straight sinus	LMWH Warfirin	2
	29y	F	Non identified	Headache Papilledema	MRI MRV	Lt cerebral haemorrhage	SSS / lt LTs/ SS	LMWH Warfirin	0
	18y	M	Dehydration	Headache Diplopia aphasia	CT MRI MRV	=====	Straight sinus	LMWH Warfirin Steroid	3
	55y	M	hypothyroidism	Headache Neck pain Diplopia N/V fever	CT MRI	SAH	Lt LTs / SS/Js	LMWH	0
	45y	F	parameningeal infection	Headache Neck pain Diplopia fever	MRI MRV	=====	Cavernous sinus	LMWH Warfirin Antibiotic Steroid	0
	40y	F	Postpartum	Headache Seizure	CT MRI MRV	Rt cerebral infarct	SSS	LMWH Warfirin Lamotergn	0
	30y	F	Postpartum	Headache Seizure Lt paresis	MRI MRV	Rt cerebral haemorrhage	SSS / ltLTs	LMWH Warfirin Lamotergn	0
	30y	F	CNS infection	Headache Neck pain Aphasia fever Papilledema Drowsiness	MRI MRV	Bilateral basal ganglia haemorrhage	SSS / lt LTs/ Rt LTs	LMWH Warfirin Antibiotic Antiviral Steroid	6
	30y	F	Postpartum	Headache Seizure Lt paresis Drowsiness Papilledema	CT MRI MRV	Rt cerebral haemorrhage	SSS / lt LTs/ Rt LTs/ SS/Js	LMWH Warfirin Lamotergn	0
	25y	F	Postpartum	Headache Rt paresis Drowsiness	CT MRI MRV	Lt cerebral haemorrhage	lt LTs/ Rt LTs	LMWH	6
	38y	M	Dehydration	Headache Seizure Lt paresis coma	CT MRI MRV	Bilateral cerebral haemorrhage	SSS / lt LTs/ Rt LTs Straight sinus	LMWH Lamotergn	6

	35y	F	Postpartum	Headache Seizure Lt paresis	CT MRI MRV	Rt cerebral haemorrhage	SSS/ Rt LTs/SS	LMWH Warfirin Lamotergn	0
	22y	M	parameningeal infection	Headache N/V Diplopia fever	MRI MRV	=====	Cavernous sinus	Antibiotic Steroid	0
	40y	F	Postpartum	Headache Seizure Rt paresis	CT MRI MRV	Lt cerebral haemorrhage	SSS/ lt LTs/ Straight sinus	LMWH Warfirin Lamotergn	1
	34y	F	Postpartum	Headache Seizure Lt paresis Coma	CT MRI MRV	Rt cerebral haemorrhage	SSS/ lt LTs/ RtLTs	HMWH Warfirin Lamotergn	1
	17y	F	Non identified	Headache Papilledema	CT MRI MRV	=====	lt LTs/ Rt LTs	LMWH Warfirin	0
	34y		polycythemia	N/V Seizure	MRI MRV	====	SSS/ lt LTs/ Cortical sinus	LMWH Warfirin	0
	23y	F	OCP	Headache Papilledema Diplopia	CT MRI MRV	====	lt LTs	LMWH Warfirin	0
	38y	F	parameningeal infection	Seizure Rt paresis Neck pain	CT MRI MRV	Lt cerebral haemorrhage SAH	SSS/ Rt LTs	LMWH Warfirin Lamotergn	2
	35y	F	Postpartum	Headache Seizure Lt paresis Papilledema	MRI MRV	Bilateral cerebral Infarct	SSS/ lt LTs/ Rt LTs Straight sinus	LMWH Warfirin Lamotergn	0
	37y	F	SLE Pregnancy	Headache Lt paresis	MRI MRV	Bilateral parenchymal lesion	lt LTs	LMWH	0
	30y	F	Protein S deficiency	Headache Papilledema Rt paresis Aphasia Coma	CT MRI MRV	Lt cerebral haemorrhae	SSS	Warfirin	0
	36y	F	Postpartum	Headache Papilledema Diplopia N/V	MRI MRV	Lt cerebral haemorrhage	lt LTs/ Rt LTs	LMWH Warfirin Lamotergn	0
	23y	F	Postpartum	Headache Papilledema Diplopia Rt paresis	MRI MRV	Lt cerebral haemorrhage	SSS	LMWH Warfirin Lamotergn	0
	26y	F	Postpartum	Headache Neck pain Diplopia Rt paresis	CT MRI MRV	Rt cerebral haemorrhage	SSS/SS	LMWH Warfirin Lamotergn	0

	28y	F	Non identified	Headache Papilledema Diplopia	MRI MRV	=====	SSS / lt LTs	LMWH Warfirin	0
	35y	F	CNS infection +Abortion	Fever Rt paresis Aphasia Coma	MRI MRV	------===	SSS / lt LTs/ Rt LTs	LMWH Antibiotic Steroid	6
	26y	F	Non identified	Headache Papilledema Diplopia Rt paresis Seizure	CT MRI MRV	Lt cerebral infarct	Lt LTs	LMWH Warfirin Lamotergn	0
	42y	M	CNS infection	Headache Fever Seizure Drowsiness	MRI MRV	Lt cerebral infarct	SSS	Antibiotic Steroid Antiviral	6
	42y	F	Pregnancy	Headache Rt paresis	CT MRI MRV	Lt cerebral infarct	Lt LTs	LMWH	2
	42y	F	Solid tumour out CNS +cytotoxic drugs	Headache Fever N/V Seizure	CT MRI MRV	Rt cerebral haemorrhage SAH	SSS	===	1
	28y	F	Postpartum	Headache Seizure Lt paresis Neck pain Coma	MRI MRV	Lt cerebral infarct	LtLTs/JV/SS	LMWH Warfirin Lamotergn	0
	31y	M	Protein C deficiency	Drowsiness	MRI MRV	=====	lt LTs/ Rt LTs	HMWH	0
	25y	M	Protein S &C deficiency	Headache Diplopia	MRI MRV	=====	Lt LTs	LMWH Warfirin	0

F-Female, M-male, CT-Computed tomography, MRI-Magnetic resonance imaging, MRV - Magnetic resonance venography, OCP-Oral contraceptive pill, CNS- Central nervous system, N&V –Nausea &Vomiting, SSS-Superior sagittal sinus, SS - Straight sinus, Lt LTs-Left Lateral transverse sinus, Rt LTs-right Lateral transverse sinus, JV-Jugular veins, DVS -Deep venous system, SLE- Systemic lupus erythromatosus ,LMWH- Low-molecular-weight heparin, HMWH-High-molecular-weight heparin, mRs – Modified RANKIN Scale.

DISCUSSION

In this hospital-based prospective study, we described the clinical features, risk factors, CT/MRI/MRV findings, treatment, and outcome in 70 patients with CVST during the period of the study at Soba University Hospital. Now the availability of CT, MRI, MRV, and CT venography facilities in our country vicinity helped us to establish the diagnosis of CVST with adequate certainty, increase the awareness of clinician doctors about CVST and early diagnosis with increase the number of neurological consultant and well train and qualified radiological consultant became easy to diagnose, how to deal and follow up patient with CVST. In our study the incidence of CVST is increase and account about 6.6% to 7.3% of all stroke, which is high when compare with the international incidence of CVST which account about 0, 5%to1% of all strokes, so CVST is not a rare disorder Consistent with several large studies CVT is believed to be more common in women than men, the female gender predominated in our study.[1,2]

This is usually related to gender associated risk factors such as the use of oral contraceptives, pregnancy, puerperium, postpartum, and abortion. But the incidence increase in male, male to female ratio is 1:4 in our study compared with local studies, a prospective study described the clinical features, risk factors, and outcome of CVT in patients admitted to the National Center for Neurological Diseases, Khartoum, Sudan, during the period from February 2001-October 2006 included 15 patients,[3] and a prospective descriptive, cross sectional, hospital based study conducted in Elshaab Teaching Hospital- Khartoum Sudan, in the period from November 2008 to July 2010 included 50 patients,[4] and most probably related to new discovered of gold miner and exposed male to dehydration, but less than incidence worldwide, In a series of 110 cases, Ameri and Bousser found a female-to-male ratio of 1.29:1.7 Ferro ET al,[5] made the same observations in a prospective study from 1995 to 1998. Similar to what was reported by the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT), to study done in 1998 by American stroke association (ASA) of 67 cases showed the average age was 26 years, published in April 2011, and to local studies above, the ages of the majority of our patients were between 21-40years. This most properly is due to the fact that this age group is the reproductive age and so patients are more prone to use pills or become pregnant[1] the mean age in men appeared to be the same as in women this may related to their heavy work at this age.

Depending on the degree and rate of the involvement of the cerebral veins, degree of recanalization, and collateral venous formation, the presentation can vary from a slow process to an acute episode. In our patients, manifestation of intracranial hypertension, namely, headache mainly sub acute and generalized (was present in nearly 90% of patients in the ISCVT), papilledema, diplopia (caused by sixth nerve palsy), nausea, vomiting, neck pain was the most common clinical presentation followed by related to focal brain injury from venous ischemia/infarction or hemorrhage seizure, limb paresis, aphasia, disturbance of the level of consciousness, patients may present with symptoms simulating transient ischemic attacks or subarachnoid hemorrhage. The similar mode of presentation was seen in many other studies.[1,2,6,7] The comparision between Symptoms and signs in our study, Ferro et al. and Wasay et al are listed in table.[13]

Fever saw in 15.4% related to the underling infections. The comparision between Symptoms and signs in our study and tow local studies are listed in table.[14]

Regarding cranial nerve the second cranial nerve is most affected 65.8%, followed by right abducent nerve palsy 23.7%,then left abducent nerve palsy 26.3%, left facial nerve palsy 18.4%, with less frequency involvement other cranial nerves, which it`s similar to local study.

Regarding risk factors of Dural Venous Thrombosis, our findings did not differ from what was mentioned in the literature.[5] Our study showed that gynecological risk factors including pregnancy, puerperium 45.7% were the most common ones and most of them during the postpartum period mainly during the first and second week post delivery. Recently, it was reported that in pregnant women, hyperhomocysteinemia was associated with increased risk of puerperal CVT (OR 10.8, 95% CI 4.0 to 29.4) in a study of 60 case subjects and 64 control subjects.[8] In a study from Mexico, _50% of CVT occurred during pregnancy or puerperium.[9] In our study cesarean section with spinal anaesthia found in 43.5% of postpartum, one study reported that the risk of peripartum CVT increased with increasing maternal age, increasing hospital size, and cesarean delivery, as well as in the presence of hypertension, infections, and excessive vomiting in pregnancy.[10]

We have three patients 5.4% in our study presented with voluntary abortion.

In our study found that the prothrombotic risk factor just only 7.1% which it`s very low related to wide world studies and this most probably related to that not all our patients our screening because most of them were taken oral anticoagulation, The largest study, the ISCVT, prospective observational study with 624 patients. Thirty-four percent of these patients had an inherited or acquired prothrombotic condition[1] Recently, another group in the United States reported that 21% of 182 CVT case subjects in 10 hospitals had a prothrombotic condition.[11] and local study was done in Elshaab Teaching Hospital- Khartoum Sudan during period from February 2001-October 2006 included 15 patients; a prothrombotic risk factor was identified in approximately 75% of patients with CVT.[3]

In our study Antiphospholipid and Anticardiolipin Antibodies found in 8.6% of the patients, in another study from India with 31 CVT patients, anticardiolipin antibodies were detected in 22.6% of CVT patients compared with 3.2% of normal control subjects.[12]

Similar findings (5.9%) were observed in the ISCVT study.[1]

In our study seven patients 10% were take oral contraceptives pills compared to 54.3% were observed in the ISCVT, and this related to decrease use of oral contraceptives pills in our country compared to western countries. In our study five patients 10% presented with CNC infection, 5 patients 7.1% with parameningeal infection (ear, sinus, mouth, face, and neck). These causes only explained 8.2% of all cases in the ISCVT series.[12]

In our study 4.3% of cases of CVT were associated with Cancer or treatment with cytotxic therapy. In the ISCVT[1] 7.4% of cases of CVT were associated with cancer. Other risk factors reported among our studied group included, dehydrated, thyroid disease, steroid medications, polycythemia, and rheumatoid arthritis this is similar to was reported by other researchers worldwide[13,14,15] The comparison between Risk factors in our study and ISCVST are listed in table.[15]

Regarding imaging features of included patients in our study CT brain was done to 32 patients 45.7% and was informative in 30% of them, in ISCVT study a plain CT being abnormal only in _30% of CVT cases.[16]

Intra cerebral haemorrhage detected in 48.5% of the included patients in our study, compared to approximately 30% to 40% of patients with CVT present with intracranial haemorrhage in ISCVT.[17]

Isolated subarachnoid hemorrhage occurs due to CVT found in 4.3%, although this is rare (0.8% of patients in ISCVT). And cerebral edema found in 18.6% compared with 20% of patients in ISCVT.[1] Regarding occluded sinus and vein the superior sagital sinus was most commonly affected 67.1%, followed by Lt transverse sinus 57.1%, Rt transverse sinus 41.4%, bilateral transverse sinus 30%, sigmoid sinus 25.7%, jugular veins 10%, straight sinus 8.6%, deep venous system 5.7%, cavernous sinus 4.3%, cortical veins 1.4%, Trolard vein 1.4%, and confluence sinus in 1.4%, there is little variation in compare with the results of the largest cohort ever published (624 patients), the superior sagittal sinus (62%), left and right transverse sinus (respectively 44.7% and 41.2%), straight sinus (18%), cortical veins (17.1%), deep venous system (10.9%), cavernous sinus (1.3%), and cerebellar veins (0.3%).[1] The compare between Occluded sinus and vein in our study and ISCVT study are listed in table.[16]

Regarding treatment and outcome nearly all patients treated with anticoagulant, and mortality was 17.1%, 61.4% had complete recovery (modified Rankin scale [mRs] score of 0 to 1), 20% had mild to moderate disability (mRS score 2 to 3), and 1.4% remained severely disabled (mRs score 4 to 5), in the largest study by ISCVT, which included 624 patients at 89 centers in 21 countries. Nearly all patients were treated with anticoagulation initially, and mortality was 8.3% over 16 months; 79% had complete recovery (modified Rankin scale [mRS] score of 0 to 1), 10.4% had mild to moderate disability (mRS score 2 to 3), and 2.2% remained severely disabled (mRS score 4 to 5). Data from observational studies suggest a range of risks for ICH after anticoagulation for CVT from zero to 5.4%.[18, 19, 20, 21] Compared with local study a prospective study described the clinical features, risk factors, and outcome of CVT in patients admitted to the National Center for Neurological Diseases, Khartoum, Sudan, during the period from February 2001-October 2006 included 15 patients, 46.7% attained complete neurological recovery, 26.7% developed optic atrophy, and 13.3% died,[3] and other cross sectional, hospital based study conducted in Elshaab Teaching Hospital-Khartoum Sudan, in the period from November 2008 to July 2010 included 50 patients, improvement in 72%, 14% left with residual weakness, Mortality was 6%.[4]

Table. 13: compare between Symptoms and signs in our study, Ferro et al. and Wasay et al.

Symptoms and signs	Ferro et al.: 624 patients	Our Study: 70 Patients	Wasay et al.: 182 Patients
Headache	88.8	85.7 71	
Visual loss	13.5	2.9 ---	
Papilledema	28.3	38.6 ---	
Diplopia	13.5	37.1 ---	
Nausea/vomiting	---	21.4 35	
Neck pain	---	21.4 ---	
Fever	---	15.4 14	
Drowsy	13.9	11.4 28	
Coma	13.9	20 15	
aphasia	19.1	15.7 16	
Mental disorder	22	1.4 18	
Any paresis	37.2	44.3 ---	
Seizure	39.3	52.9 32	
Sensory symptoms	5.4	7.1 ---	

Table. 14: Compare between Symptoms and signs in our study and tow local studies.

Symptoms and signs	Idris et al 15 patients	our study : 70 patients	Local study: 50 patients
Headache	100	85.7 96	
Visual loss	--	2.9 0.08	
Papilledema	86.7	38.6 ---	
Diplopia	--	37.1 ---	
Nausea/vomiting	---	21.4 --	
Neck pain	---	21.4 90	
Fever	---	15.4 ---	
Drowsy	--	11.4 ---	
Coma	--	20 46	
aphasia	--	15.7 --	
Mental disorder	--	1.4 --	
Any paresis	20	44.3 28	
Seizure	20	52.9 46	
Sensory symptoms	--	7.1 ---	

Table. 15: compare between Risk factors in our study and ISCVST.

Risk factor	ISCVST	Our study: 70 patients
None identified	12.5	7.1
Thrombophilia	34.3	5.7
Acquired-antiphospholipid	5.9	8.6
Nephrotic syndrome	0.6	1.4
Solid tumor outside CNS Cytotoxic drugs	7.4	4.3
Polycythemia	2.8	1.4
Vasculitis SLE	1	2.9
Rheumatoid arthritis	--	1.4
Thyroid disease	1.7	2.9
Pregnancy /Puerperium	21	45.7
Abortion	--	5.4
CNC infection	--	10
Parameningeal infection	12.3	7.1
Oral contraceptives pills	54.3	10
Hormonal replacement therapy	2	2.9
Cytotoxic drugs	2	2.9
Steroid	--	2.9
Dehydrated	--	7.1

Table. 16: Compare between Occluded sinus/vein in our study and ISCVT study.

Occluded sinus/vein	ISCVT study	Our study
Superior sagital sinus	70	67.1
Lt transverse sinus	26	57.1
Rt transverse sinus	26	41.4
bilateral transverse sinus	18	30
Straight sinus	14	8.6
Deep venous system	8	5.8
Cortical veins	17	1.4
Jugular veins	12	10
Cavernous sinus	3	4.3
Sigmoid sinus	15	25.7
Trolard vein	--	1.4
Confluence sinus	--	1.4

CONCLUSION

❖CVTS is not uncommon in Sudan. Commonly affect female during child burring period.

❖Gynecological included postpartum and infectious causes are more frequent in this study

❖Thrombophilia and acquired antiphospholipid syndrome as aetiology seems to be underestimated.

❖Most common symptom was headache visual disturbance, and seizure.

❖ Papiledema, paresis and VI cranial nerve palsy were the commonest examination finding.

❖The investigation of homeostasis and thrombophilia included protein C, protein S, antithrombin deficiency, antiphospholipid syndrome, prothrombin G20210A mutation, and factor V Leiden should be a routine after cerebral venous thrombosis.
❖The uncontrasted CT, MRI, MRV, and CT venography are the key diagnostic tools.
❖Great majority of the patients showed remarkable improvement
❖The outcome is favorable using heparin treatment

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