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ORIGINAL ARTICLE

Neuro-ophthalmological Features of Cerebral Venous Sinus Thrombosis

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ABSTRACT

Neuro-ophtalmological symptoms of 49 patients with cerebral venous sinus thrombosis and different onset of the disease were evaluated. Papilloedema was revealed in 84.6% of patients with acute and subacute onset of the disease and in all patients with chronic onset. Visual disturbances due to papilloedema and post-papilloedema optic atrophy were found in 65.2% of patients with chronic onset. Patients with acute onset of cerebral venous sinus thrombosis were successfully treated with local endovascular thrombolysis. Patients with chronic onset of cerebral venous sinus thrombosis in cases with visual disturbances needed lumboperitoneal shunting to prevent further visual loss.

Keywords: Cerebral venous sinus thrombosis, papilloedema

INTRODUCTION

Cerebral venous sinus thrombosis (CVST) is a rare disease. Among adult population it is more common in women.[1–4] CVST clinical manifestations is varied.[1,4] Neuro-ophthalmological symptoms are related to intracranial hypertension and presented with papilloedema.[1,4] The goal of our investigation was to describe neuro-ophthalmological features and results of different methods of treatment in patients with CVST.

MATERIALS AND METHODS

We studied 49 patients with CVST admitted to the Burdenko Neurosurgery Institute. Patients aged from 5 to 54 (median 33) years. There were 35 (71.4%) women (13–52 years old, median 33 years), 12 (24.5%) men (21–54 years old, median 36 years), and 2 boys (5 and 10 years old).

The diagnosis of CVST was confirmed by computed tomography (CT) or magnetic resonance (MR) venography in all patients. Digital subtraction angiography (DSA) was performed in addition in 14, who were treated with local endovascular thrombolysis.

Thirty patients (61.2%) had multiple sinuses and/or cerebral veins involvement; single cerebral sinus was affected in 19 patients (38.8%) (Table 1). Superior sagittal sinus and lateral sinus thrombosis were diagnosed more frequently. Eleven patients (22.4%) had single superior sagittal sinus thrombosis, 7 (14.3%) had lateral sinus/sinuses thrombosis, 11 (22.4%) were presented with thrombosis of superior sagittal sinus in combination with lateral sinus, and 10 (20.4%) patients had superior sagittal sinus and lateral sinus/sinuses thrombosis with involvement of other sinuses or cerebral veins.

All patients were examined at the first investigation and at least once for a follow-up by neuro-ophthalmologist.

All patients were divided into three groups according to the onset of the disease. The first group (15) consisted of patients with acute onset (Table 2). They were admitted to the Burdenko Neurosurgery Institute within 3–14 days from the onset of symptoms. The second group (11) consisted of patients with

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TABLE 1 Site of venous occlusion in patients with cerebral venous sinus thrombosis.

Venous sinus thrombosis	*n*
Superior sagittal sinus	11
Superior sagittal sinus + lateral sinus	7
Superior sagittal sinus + two lateral sinuses	4
Superior sagittal sinus + lateral sinus + sigmoid sinus	4
Superior sagittal sinus + two lateral sinuses + jugular vein	1
Superior sagittal sinus + lateral sinus + straight sinus	1
Superior sagittal sinus + two lateral sinuses + straight sinus	2
Superior sagittal sinus + two lateral sinuses + sigmoid sinuses	1
Superior sagittal sinus + lateral sinus + sigmoid sinus + jugular vein	1
Lateral sinus	5
Two lateral sinuses	2
Lateral sinus + straight sinus	1
Lateral sinus + sigmoid sinuses	4
Lateral sinus + sigmoid sinus + jugular vein	1
Sigmoid sinus + jugular vein	1
Straight sinus	1
Sigmoid sinus	1
Confluence of the sinuses	1
Total	49

subacute onset of the disease and progression of clinical features from 15 to 30 days (Table 3). In the same period these patients were admitted to the Burdenko Neurosurgery Institute. The third group consisted of patients with chronic onset (23) symptoms developed more than 1 month (Table 4).

Patients with multiple sinuses and/or cerebral veins involvement showed acute onset in 10 patients, subacute onset in 7, and chronic onset in 13. Patients with one cerebral sinus thrombosis showed acute onset in 5 patients, subacute onset in 4, and chronic onset in 10. Fourteen patients were treated with local endovascular thrombolysis with Actilyse (rtPA), prourokinase, urokinase, in one case with mechanical thrombus extraction. Fourteen patients required lumboperitoneal shunting.

RESULTS

Predisposing factors for CVSF were identified in 19 patients (38.8%), including pregnancy and puerperium (3), oral contraceptives (4), congenial coagulopathies (4), blood donation (2), acute lymphoblastic leukaemia (1), T-cell lymphoblastic lymphoma (1), head trauma (1), sinusitis maxillaris (2), and dehydration (1). The cause of CVST was not revealed in 30 patients (61.2%).

In the group with acute onset, 10 patients had multiple sinuses involvement, 5 had single sinus thrombosis. The first group (15) was presented by focal deficit such as hemiparesis and seizures in 3 patients. All patients had symptoms of raised intracranial pressure such as headache and nausea. Unilateral or bilateral sixth nerve palsy was revealed in 4 patients. Papilloedema was found in 12 patients. Two patients had low degree of papilloedema, 5 moderate, and 5 marked. Haemorrhages over and adjacent to the optic disc were observed in 7 patients with papilloedema. Macular oedema and haemorrhages were revealed in 1 patient. This patient had visual disturbances of the right eye, with visual acuity 0.01 and central scotoma due to haemorrhages in macular. Other patients of this group had normal visual functions. Anticoagulant therapy was used in all patients. It was successful in 4 patients who showed recanalisation of cerebral venous sinuses and regress of signs of intracranial hypertension. Eleven patients were treated with local endovascular thrombolysis, one of them in combination with mechanical endovascular thrombectomy. According to the control angiography immediately after local thrombolysis, complete or partial recanalisation of the sinuses was achieved in 10 patients. These patients showed regression of headache and nausea immediately after endovascular surgery. Endovascular thrombolysis was not successful only in 1 patient, which required lumboperitoneal shunting. We observed regression of papilloedema in patients with acute onset of the disease during 7–45 days. Two patients had increased haemorrhages near the optic disc after local endovascular thrombolysis, followed by resorption of haemorrhages.

In the group with subacute onset, 7 patients had multiple sinuses thrombosis, 4 had single sinus involvement. The evaluation of patients with subacute onset (11) revealed hemiparesis and seizures in 4 patients. All patients had headache and nausea. Sixth nerve palsy was revealed in 4 patients. Ten patients had papilloedema: 2 low degree, 3 moderate, 5 marked. Haemorrhages over and adjacent to the optic disc were found in 6 patients with papilloedema. Visual function was normal in all patients. All patients received anticoagulant therapy. Successful local endovascular thrombolysis was performed in 3 patients. The indication for endovascular treatment was severe intracranial hypertension and relatively short period of the disease (15 days). Two patients with sinusitis maxillaries were treated with antibiotic therapy. Resolution of papilloedema was observed in patients with subacute onset during 14–60 days.

There were 13 patients with multiple sinus thrombosis and 10 with single sinus thrombosis among patients with chronic onset of the disease (23). This group was presented with focal neurological deficit in 2 patients, which was less than in patients with acute and subacute onset of the disease (2 of the 23 and 7 of the 26; $p = 0.01$). Sixth nerve palsy was found in 2 patients.

TABLE 2 Clinical presentation, methods of treatment, and outcome in patients with acute thrombosis of cerebral sinuses and veins.

Case	Age (years)	Sex	Site of venous occlusion	Days from onset to admission	Clinical presentation	Grade of papilloedema OU	Visual acuity OD/OS	Visual field OU	Treatment	Outcome
1	42	M	SSS, 2LS	12	Intracranial hypertension, papilloedema	Moderate + H	1.0/1.0	Normal	A, T (not successful), LPS	Normalisation of intracranial pressure
2	22	F	SSS	10	Intracranial hypertension, papilloedema, hemiparesis	Marked + H	1.0/1.0	Enlarged blind spot	AC, T	Recanalisation of the sinuse, normalisation of the intracranial pressure
3	31	F	SSS, 2LS, 2IJV	14	Intracranial hypertension, papilloedema	Moderate	1.0/1.0	Normal	AC, T	Recanalisation of the sinuses, normalisation of the intracranial pressure
4	30	M	SSS, 2LS, CS	4	Intracranial hypertension	Normal	1.0/1.0	Normal	AC, T	Recanalisation of the sinuses, normalisation of the intracranial pressure
5	37	F	2LS, right S	8	Intracranial hypertension, papilloedema	Marked + H	1.0/1.0	Enlarged blind spot	AC, T	Recanalisation of the sinuses, normalisation of the intracranial pressure
6	26	F	SSS, right LS	10	Intracranial hypertension, papilloedema, sixth nerve palsy	Marked + H	1.0/1.0	Enlarged blind spot	AC, T	Recanalisation of the sinuses, normalisation of the intracranial pressure
7	36	M	SSS, right LS, right S	10	Intracranial hypertension, papilloedema, confusion	Moderate	1.0/1.0	Normal	AC, T	Recanalisation of the sinuses, normalisation of the intracranial pressure
8	33	F	SSS, 2LS	7	Intracranial hypertension, papilloedema	Moderate	1.0/1.0	Normal	AC, T	Recanalisation of the sinuses, normalisation of the intracranial pressure
9	37	M	SSS, right LS	7	Intracranial hypertension, papilloedema, confusion	Low degree	1.0/1.0	Normal	AC, T	Recanalisation of the sinuses, normalisation of the intracranial pressure
10	15	F	SSS	3	Intracranial hypertension, hemiparesis, seizures	Normal	1.0/1.0	Normal	AC, T	Recanalisation of the sinuse, normalisation of the intracranial pressure
11	21	M	SS, right LS	14	Intracranial hypertension	Normal	1.0/1.0	Normal	AC	Recanalisation of the sinuses, normalisation of the intracranial pressure
12	33	F	SSS	7	Intracranial hypertension, papilloedema, sixth nerve palsy	Moderate	1.0/1.0	Normal	AC	Recanalisation of the sinuse, normalisation of the intracranial pressure
13	35	F	SSS, left LS, left S	9	Intracranial hypertension, papilloedema, hemiparesis, sixth nerves palsy	Low degree + H	1.0/1.0	Normal	AC	Recanalisation of the sinuses, normalisation of the intracranial pressure
14	23	F	SSS	14	Intracranial hypertension, papilloedema, visual impairment of the left eye	Marked + H + macular H	1.0/001	OD: normal OS: central scotoma	AC, S, acetazolamide	Recanalisation of the sinuse, normalisation of the intracranial pressure, improvement of visual functions
15	19	F	SSS	10	Intracranial hypertension, papilloedema, sixth nerve palsy	Marked + H	1.0/1.0	Enlarged blind spot	A, T, acetazolamide	Recanalisation of the sinuse, normalisation of the intracranial pressure

SS = superior sagittal sinus; LS = lateral sinus; SS = straight sinus; S = sigmoid sinus; IJV = internal jugular vein; CS = confluence of the sinuses; H = haemorrhages; AC = anticoagulants; T = local endovascular thrombolysis; LPS = lumboperitoneal shunting; S = steroids.

TABLE 3 Clinical presentation, methods of treatment, and outcome in patients with subacute thrombosis of cerebral sinuses and veins.

Case	Age (years)	Sex	Site of venous occlusion	Days from onset to admission	Clinical presentation	Grade of papilloedema OU	Visual acuity OD/OS	Visual field OU	Treatment	Outcome
1	27	F	Left LS, left S	30	Intracranial hypertension, papilloedema	Marked + H	1.0/1.0	Enlarged blind spot	AC	Normalisation of the intracranial pressure
2	27	F	SSS, SS, right LS	15	Intracranial hypertension, papilloedema, seizures	Moderate	1.0/1.0	Enlarged blind spot	AC, T	Recanalisation of the sinuses, normalisation of the intracranial pressure
3	5	M	SSS	15	Intracranial hypertension, papilloedema, sixth nerve palsy	Moderate	1.0/1.0	Normal	AC, T	Recanalisation of the sinuses, normalisation of the intracranial pressure
4	32	F	Left LS, left S, left IJV	20	Intracranial hypertension, papilloedema, seizures, hemiparesis	Low degree + H	1.0/1.0	Normal	AC, S	Normalisation of the intracranial pressure
5	24	F	SSS, left LS, left S	21	Intracranial hypertension, papilloedema, sixth nerve palsy	Marked + H	1.0/1.0	Enlarged blind spot	AC, S	Normalisation of the intracranial pressure
6	31	F	Right LS, right S	20	Intracranial hypertension, seizures, hemiparesis	Normal	1.0/1.0	Normal	AC, S	Normalisation of the intracranial pressure
7	21	M	SSS	20	Intracranial hypertension, papilloedema, seizures	Low degree	1.0/1.0	Normal	AC	Normalisation of the intracranial pressure
8	16	F	Right LS	20	Intracranial hypertension, papilloedema, sixth nerve palsy	Marked + H	1.0/1.0	Enlarged blind spot	AB, AC	Normalisation of the intracranial pressure
9	40	F	SSS	15	Intracranial hypertension, papilloedema, sixth nerve palsy	Marked + H	1.0/1.0	Enlarged blind spot	AC, T, S	Recanalisation of the sinuses, normalisation of the intracranial pressure
10	13	F	2LS	30	Intracranial hypertension, papilloedema	Moderate + H	1.0/1.0	Normal	AC, AB	Normalisation of the intracranial pressure
11	33	F	SSS, right LS, right, IJV, right S	30	Intracranial hypertension, papilloedema	Marked	1.0/1.0	Enlarged blind spot	AC	Normalisation of the intracranial pressure

SS = superior sagittal sinus; LS = lateral sinus; SS = straight sinus; S = sigmoid sinus; IJV = internal jugular vein; CS = confluence of the sinuses; H = haemorrhages; AC = anticoagulants; T = local endovascular thrombolysis; LPS = lumboperitoneal shunting; S = steroids; AB = antibiotics.

TABLE 4 Clinical presentation, methods of treatment, and outcome in patients with chronic thrombosis of cerebral sinuses and veins.

Case	Age (years)	Sex	Site of venous occlusion	Months from first symptoms to admission	Clinical presentation	Grade of papilloedema OD/OS	Visual acuity OD/OS	Visual field OD/OS	Treatment	Outcome
1	27	F	SSS, SS, 2LS	1.5	Intracranial hypertension, papilloedema, visual impairment	Marked + H/Marked + H	0.1/0.6	Concentrical constriction/ Concentrical constriction	LPS	Normalisation of the intracranial pressure, improvement of visual functions
2	36	F	2LS	12	Intracranial hypertension, papilloedema, visual impairment	Marked/Marked	0.6/0.8	Nasal step/Concentrical constriction	LPS	Normalisation of the intracranial pressure, visual diturbanses without dynamic
3	42	F	CS	10	Intracranial hypertension, papilloedema, visual impairment	Marked/Marked	0.3/1.0	Nasal step/Nasal step	LPS	Normalisation of the intracranial pressure, visual diturbanses without dynamic
4	51	F	SSS, left LS	2	Intracranial hypertension, papilloedema, visual impairment	Marked + H/Marked + H	0.2/0.3	Nasal step/Nasal step	AC, S	Normalisation of the intracranial pressure, improvement of visual functions
5	34	F	SSS	1.5	Intracranial hypertension, papilloedema, visual impairment	Marked + H/Marked + H	0.1/0.1	Nasal step/Nasal step	AC, S	Normalisation of the intracranial pressure, improvement of visual functions
6	35	F	Left LS	4	Intracranial hypertension, papilloedema, visual impairment	Marked + H/Marked + H	1.0/1.0	Nasal step/Nasal step	LPH	Normalisation of the intracranial pressure, visual diturbanses without dynamic
7	47	F	SSS, right LS	1.5	Intracranial hypertension, papilloedema	Marked/Marked	1.0/1.0	Enlarged blind spot/ Enlarged blind spot	AC, S	Normalisation of the intracranial pressure
8	41	F	SSS, 2LS	4	Intracranial hypertension, papilloedema, visual impairment	Marked + H/Post-papilloedema secondary optic atrophy	0.4/HM	Nasal step/–	LPH	Normalisation of the intracranial pressure, visual diturbanses without dynamic
9	52	F	SSS, SS, 2LS	24	Intracranial hypertension, post-papilloedema secondary optic atrophy, visual impairment	Post-papilloedema secondary optic atrophy/ Post-papilloedema secondary optic atrophy	0.02/1.0	Temporal island/ Nasal step	AC, S	Normalisation of the intracranial pressure, visual diturbanses without dynamic
10	48	F	SSS, left LS	30	Intracranial hypertension, post-papilloedema secondary optic atrophy, visual impairment	Post-papilloedema secondary optic atrophy/ Post-papilloedema secondary optic atrophy	0.2/0.5	Temporal island/ Temporal island	LPH	Normalisation of the intracranial pressure, visual diturbanses without dynamic
11	38	F	SSS, 2LS	12	Intracranial hypertension, papilloedema, visual impairment	Marked/Marked	0.9/0.6	Nasal step/Nasal step	AC, S	Normalisation of the intracranial pressure, visual diturbanses without dynamic
12	21	F	Left LS	12	Intracranial hypertension, papilloedema	Low degree/Low degree	1.0/1.0	Enlarged blind spot/ Enlarged blind spot	AC, S	Normalisation of the intracranial pressure, visual diturbanses without dynamic
13	31	F	SSS	2	Intracranial hypertension, papilloedema	Marked/Marked	1.0/1.0	Enlarged blind spot/ Enlarged blind spot	AC, S	Normalisation of the intracranial pressure, stable visual functions
14	47	F	Left LS, left S	6	Intracranial hypertension, papilloedema, blindness	Marked + H/Marked + H	0/0	–	LPH	Normalisation of the intracranial pressure, visual diturbanses without dynamic

(*continued*)

TABLE 4 Continued

Case	Age (years)	Sex	Site of venous occlusion	Months from first symptoms to admission	Clinical presentation	Grade of papilloedema OD/OS	Visual acuity OD/OS	Visual field OD/OS	Treatment	Outcome
15	36	M	SSS, right LS, right S	2.5	Intracranial hypertension, papilloedema, visual impairment	Marked + H/Marked + H	0.5/0.8	Enlarged blind spot/ Central scotoma	LPS	Normalisation of the intracranial pressure, improvement of visual functions
16	33	F	Right LS	6	Intracranial hypertension, post-papilloedema secondary optic atrophy, blindness, sixth and seventh nerve palsy	Post-papilloedema secondary optic atrophy/ Post-papilloedema secondary optic atrophy	0/0	–	LPS	Normalisation of the intracranial pressure, visual diturbanses without dynamic
17	10	M	SSS	4	Intracranial hypertension, papilloedema, visual impairment	Marked + H/Marked + H	1.0/1.0	Nasal step/Nasal step	LPS	Normalisation of the intracranial pressure, visual diturbanses without dynamic
18	36	M	SS	5	Intracranial hypertension, papilloedema, monoparesis, fifth, sixth, seventh nerve palsy	Moderate + H/ Moderate + H	1.0/1.0	Enlarged blind spot/ Enlarged blind spot	AC, S	Normalisation of the intracranial pressure,
19	46	M	SSS, left LS	6	Intracranial hypertension, post-papilloedema secondary optic atrophy, visual impairment	Post-papilloedema secondary optic atrophy/ Post-papilloedema secondary optic atrophy	0.01/0.6	Temporal island/ Temporal island	LPH	Normalisation of the intracranial pressure, visual diturbanses without dynamic
20	24	M	SSS, right LS	1.5	Intracranial hypertension, papilloedema	Marked + H/Marked + H	1.0/1.0	Enlarged blind spot/ Enlarged blind spot	AC, S	Normalisation of the intracranial pressure
21	52	F	Right S	3	Intracranial hypertension, papilloedema, post-papilloedema secondary optic atrophy, visual impairment	Marked/Post-papilloedema secondary optic atrophy	0.9/0.9	Nasal step/Nasal step	LPH	Normalisation of the intracranial pressure, visual diturbanses without dynamic
22	52	M	Right IJV, right S	5	Intracranial hypertension, papilloedema, post-papilloedema and secondary optic atrophy, visual impairment	Marked + H/Post-papilloedema secondary optic atrophy	1.0/HM	Enlarged blind spot/–	LPS	Normalisation of the intracranial pressure, visual diturbanses without dynamic
23	54	M	Right LS	3	Intracranial hypertension, papilloedema	Marked + H/Moderate	1.0/1.0	Enlarged blind spot/ Enlarged blind spot	AC, S	Normalisation of the intracranial pressure

SS = superior sagittal sinus; LS = lateral sinus; SS = straight sinus; S = sigmoid sinus; IJV = internal jugular vein; CS = confluence of the sinuses; H = haemorrhages; AC = anticoagulants; LPS = lumboperitoneal shunting; S = steroids.

All patients of the third group had papilloedema. One patients had low degree of papilloedema, 1 moderate, 1 marked on one eye and moderate on the other eye, 13 marked, 3 marked on one eye and post-papilloedema secondary optic atrophy on the other eye, and 4 post-papilloedema secondary optic atrophy. Haemorrhages over and adjacent to the optic disc were observed in 12 patients. Visual acuity loss was revealed in 15 patients. All of them had marked papilloedema or post-papilloedema optic atrophy. Two patients were blind; visual acuity of one eye ranged from hand motion to 0.02 in 4 patients. Nine patients had visual acuity 0.1–0.9 in one or both eyes. Visual field defects in one or both eyes were presented with nasal step, concentrical constriction, and temporal island in 14 patients. Thirteen patients required lumboperitoneal shunting for worsening vision. Resolution of papilloedema in these patients was achieved within a month. Ten patients received anticoagulant therapy, acetazolamide and steroids. Papilloedema regressed in this group in 6–9 months.

DISCUSSION

In adults, CSVT most commonly occurred in young and middle-aged women.[1–4] In our series, 71.4% of patients were women 13–52 years old. Underlying risk factors were presented only in 38.8% of our patients, whereas other authors found them in 26–87.5%.[1,5–7]

The superior sagittal and lateral sinus thrombosis was diagnosed more frequently in our and other series.[1,7] More than one sinus was involved in 61.2% of our patients.

It is seen from review of the literature that clinical presentation of CVST is varied.[1,4,7] The results of international studies revealed that the most frequent symptoms were headache, paresis, papilloedema, visual loss, diplopia, focal seizure, or seizure with generalisation.[1,4] Papilloedema was found in 28–45%.[1,4,7] Visual disturbances of varying severity were revealed in 13.2–52.5%.[1,8]

Bousser et al.[7] in the review of 38 cases with CVST showed that clinical presentation was slightly different in patients with single and multiple sinus occlusion. Patients with single sinus thrombosis showed somewhat slower onset and more frequently isolated intracranial hypertension. Multiple thrombosis of cerebral sinuses and veins were associated with acute onset and focal signs.[7] In our series, there was no significant correlation between the site and extent of thrombosis and clinical parameter. Maybe we need to study more patients. However, our study showed that clinical manifestation of CSVT depended on the mode of onset. Acute and subacute CVST often presented with focal symptoms in combination with signs of intracranial hypertension, whereas for chronic CVST isolated intracranial hypertension was more typical.

The evaluation of our patients with acute and subacute CVST revealed papilloedema in 22 of 26 patients. It should be noted the high frequency of haemorrhages over and adjacent to the optic disc in patients with papilloedema. Visual disturbances were not frequent in these groups. In the present series, only 1 patient had visual loss due to papilloedema associated with macular haemorrhages.

Clinical features of chronic CVST often looked similar to symptoms of idiopathic intracranial hypertension.[6] Percentage of CVST among patients with idiopathic intracranial hypertension is not clear. Indian studies identified CVST in 11.4%[5] and 36.5%[8] of patients with presumed idiopathic intracranial hypertension. Purvin et al.[9] believed that clinical manifestation of CVST may be differentiated from idiopathic intracranial hypertension by the abrupt onset and marked severity of symptoms. In the present series, patients with chronic CVST came to the neuro-ophthalmological department for consultation with the papilloedema and visual disturbances identified in other hospitals. Papilloedema was revealed in all patients with chronic CVST. High incidence of haemorrhage over and adjacent to the optic disc was noticed in these patients, as in the groups with acute and subacute onset. In the group with chronic onset, visual impairment due to marked papilloedema or post-papilloedema secondary optic atrophy occurred in 15 of 23 patients. Visual loss in some cases was dramatic. Visual disturbances were typical for marked papilloedema and post-papilloedema secondary optic atrophy and had been described in details in the literature.[10]

CVST represents secondary form of intracranial hypertension.[11] We agree with Biousse et al.[6] who believed that CVST should not be classified as idiopathic intracranial hypertension. As symptoms of CVST are often identical to idiopathic intracranial hypertension, it is very important to differ these diseases for proper management. Patients with suspected CVST need MR or CT venography. Risk factors, including genetic and acquired prothrombotic disorders, infection, inflammatory diseases, anaemia, thrombocythaemia, drags, trauma, and dehydration should be estimated. Methods of treatment depend on the duration of the disease and presence of visual disorders. Local endovascular thrombolysis sometimes in combination with mechanical thrombectomy could be used in patients with acute onset and duration of symptoms less than 15 days. In our series, endovascular thrombolysis led to quick regression of signs of intracranial hypertension. For the last 20 years, several series on local endovascular thrombolysis in CVST have been published.[12–17] High efficacy of endovascular treatment has been shown. Wasay et al.[18] in a non-randomised study showed that

local thrombolysis may be more effective than systemic anticoagulation alone in treating superior sagittal sinus thrombosis. However, local thrombolysis may carry a higher risk of cerebral haemorrhage.[16,19] We consider that endovascular techniques of mechanical thrombectomy especially in patients with intracranial haemorrhage has significant potential. European Federation of Neurological Societies guideline speaks in favour of further international multicentre study to clarity the role of local thrombolysis in the treatment of CVST.[20]

Lumboperitoneal shunting should be performed in patients with chronic onset of CVST in cases with papilloedema and visual disorders to prevent further deterioration of vision. Anticoagulants should be used in all patients with CVST.

CONCLUSION

Neuro-ophtalmological symptoms in patients with CSVT present with signs of intracranial hypertension. Papilloedema is one of the most frequent sign. Methods of treatment in patients with CSVT depend on the onset of the disease. Patients with acute onset of CSVT could be successfully treated with local endovascular thrombolysis. Patients with chronic onset of CVST in combination with papilloedema and visual disturbances need shunting procedure.

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