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THE PRIMARY THROMBOSIS OF DURAL SINUSES AND CEREBRAL VEINS IN ADULT LIFE

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The thrombosis of the dural sinuses and of the cerebral veins is a nosological entity which is less frequent in our days, but they are difficult to diagnose and have a less severe prognosis. We had analyzed 22 cases diagnosed with this type of ailment. Out of the 22 cases, 17 (77.27%) had been women and only 5 (22.72%) men.The causes of the respective thrombosis had been primary, secondary and idiopathic. The magnetic resonance venography and angiography of the 4 cerebral blood vessels had represented methods for the diagnosis of the above mentioned patients. The heparin treatment had been administered in the first 48 hours for the patients with a dramatic onset, followed by the treatment with low-molecular-weight heparin fractions, and thereafter we had proceeded to the administer oral anticoagulants for several months. Generally, depending on the case, besides the anticoagulant medication, we had also administered antiepileptic and symptomatic treatment, as well as performing surgery. In this way we had achieved healings in 15 (68.18%) of the patients, improvements in 6 (27.27%) of the cases, while 1 (4.54%) had died. The prognosis had been generally favorable for the patients with occlusions of the lateral sinus, and pejorative for those with thrombosis of the superior sagittal sinus.

Key words: thrombosis, dural sinuses, cerebral veins, nosological entity, diagnose.

INTRODUCTION

The thrombosis of the dural sinuses and of the cerebral veins is an infrequent condition which accounts for 0.5 up to 1% of the total number of strokes[1-5] or 3 up to 5 cases per one million of the[6-7].

It can occur at any age, but compared with other types of stroke, it is encountered more frequently in women, with a 3:1 ratio[4,7], and in young patients[2]. These cerebral venous thromboses are more frequent and with a higher mortality rate in poor countries, where it is especially associated with the puerperal pathology[4]. It is possible that inappropriate perinatal care, dehydration, iron deficiency anemia and infections associated to childbirth are factors that account for this greater frequency[8,9].

Accordingly, the thromboses of the cerebral veins and of the dural sinuses are nosological entities that are less frequent in our days, have a less severe prognosis and MRI in combination with magnetic resonance venography has proved to have the highest sensitivity and specificity in establishing a diagnosis.

The thrombosis can afflict the sinuses of the dura mater (superior sagittal, transverse, sigmoid and straight) or the cortical veins, with variable consequences which are dependent on their anastomotic possibilities, which are extremely variable from one individual to another.

According to Stam (2005)[6] and Piazza (2012)[7], there are two mechanisms that can explain the development and the symptomatology of the cerebral venous thromboses: the increase in the intracranial pressure caused by impaired cerebrospinal fluid (CSF) reabsorption at the level of the arachnoid granulations and the obstruction of the venous circulation, with the subsequent increase

in the capillary pressure which causes the cerebral parenchymal dysfunction. These can lead to the occurrence of hemorrhages.

Thus, depending on the venous disposition and on the collateral drainage pathways, we may find a vasogenic edema, which is reversible, or a cytotoxic edema that is responsible for the parenchymal lesions. Depending on the patient and on the condition of his/her veins, the clinical manifestations and the symptomatology differ greatly from one case to another[10]. The imprecise onset and evolution lead often to the lateness of diagnosis.

The identifiable factors which had been found at admission as associated with a poor short-term outcome are coma, intracerebral hemorrhage, the presence of the delta sign on CT, central nervous system infection, focal deficits, symptoms of encephalopathy (generalized seizures, abulia, delirium, stupor or coma), bilateral pyramidal tract signs of deep location of the thrombosis (the straight sinus or the great vein of Galen)[4, 10- 21].

The factors which had been found at admission, as associated with a good short-term outcome are normal consciousness, the absence of intracerebral hemorrhage or infarction, younger age, the absence of sings of encephalopathy, isolated intracerebral hypertension, the absence of the focal deficit and early heparin therapy[10-23].

In the following pages we describe our 22 cases, followed by the diagnosis and comments.

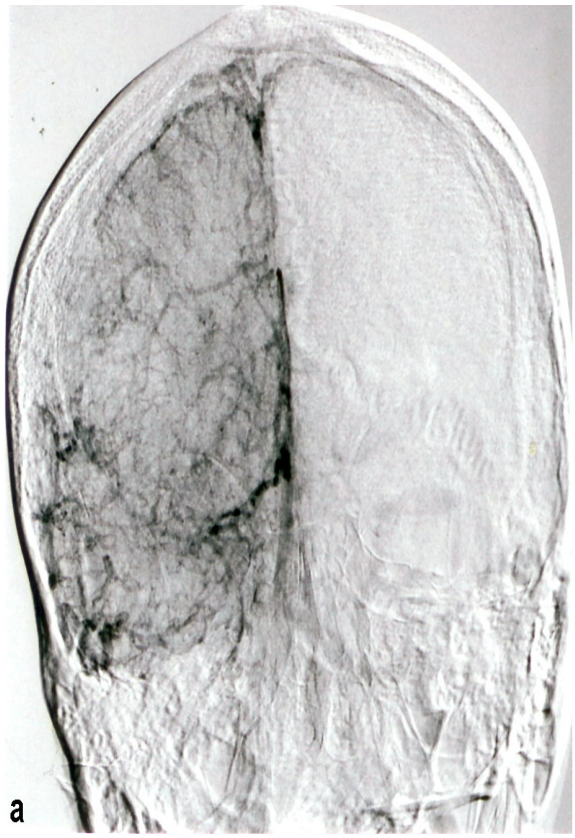
PATIENTS AND METHOD

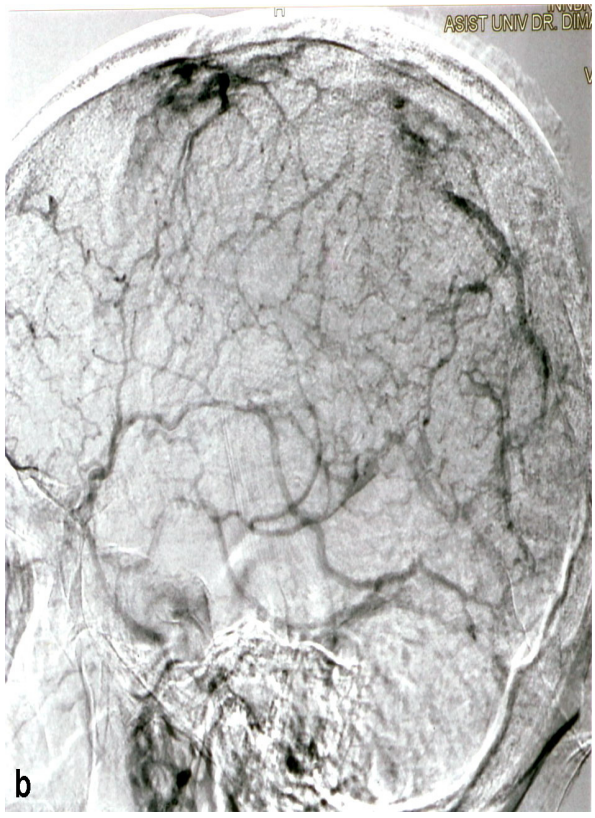
Case No. 1

BC, a male patient aged 56 years old, suffering with chronic alcoholism, had been admitted in our vascular neurosurgery department with first degree of coma and left hemiplegia.

The cerebral angiography of the 4 blood vessels had rendered evident the complete thrombosis of the superior sagittal sinus (Figures 1a, 1b, 1c), while the computed tomography had revealed the presence of a right parietal cerebral haemorrhage (Figure 1d).

It had been performed the surgical intervention during which it had been evacuated a right parietal intracerebral haemorrhagic softening of brain matter. Postoperatively, the neurological and the general health condition of the patient had improved progressively, with the persistence of a pronounced left motor deficit and of a left hemi-hypoesthesia. He had received treatment with Clexane, 2 ampule per day, for 12 days, and then he had been transferred to a motor recovery department. At the control performed after two months it had been found conscious, but the motor deficit had improved to a very small extent.





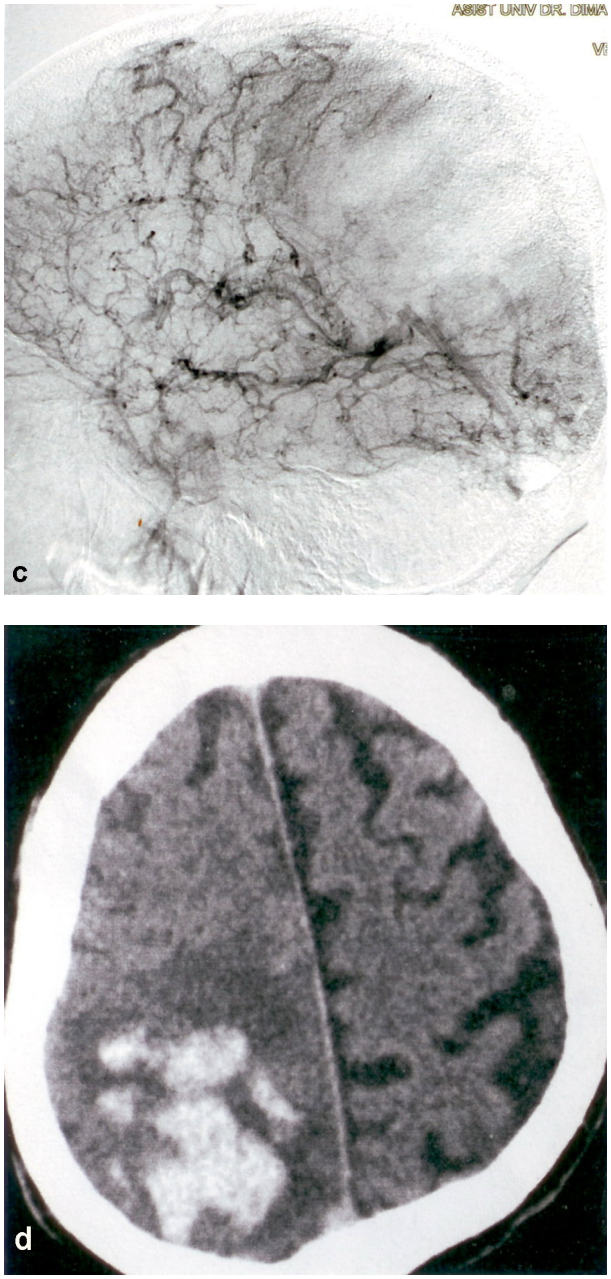


Fig. 1. The anteroposterior (a) and the lateral (b) views of the right carotid angiography, as well as the lateral (c) view of the venous phase of the left carotid angiography reveal the thrombosis of the superior sagittal sinus. The computed tomography (d) show a right parietal cerebral hemorrhage.

Case No. 2

A aged 41-years-old man, plethoric and with high blood pressure, had a thrombophlebitis of the lower extremities and a pulmonary embolism developed following a surgical intervention for prostate adenoma. He had been admitted in our vascular neurosurgery department following the sudden occurence in the ninth day after surgery of motor seizures in the right hemibody, followed by generalized mal, headache and mixed aphasia.

At the admittance in the hospital, the patient experienced general seizures, headaches, right hemiparesis and mixed aphasia.

The late phase of the left carotid angiography had revealed a slowing down of the cerebral circulation, especially in the frontal area, and the lack of visualization of the superior sagittal sinus which had remained permeable only in the area adjacent to the confluence of sinuses, for a length of approximately 1.5 cm.

To be noted the routing of the venous circulation towards the inferior longitudinal sinus and the sylvian valley.

He had received anticoagulant, anticonvulsive and cerebral anti-edematous treatment, which had been followed by the disappearance of all the neurological symptoms.

Case No. 3

A 49-years-old man, with mega-dolichocolon and sigmoid stenosis, had been admitted in the hospital in the 11th day after surgery for headaches, epilepsy and moderate motor deficit in the left side extremities.

The MRI and the magnetic resonance venography demonstrates a thrombosis of the superior sagittal sinus and of the right transversal one, right parietal and occipital venous infarction, the presence of several small bilateral subdural hematomas, diminished ventricles and the flattening of the right parietal and occipital gyri. The cerebral angiography of the 4 blood vessels had confirmed the quasi-complete thrombosis of the superior sagittal sinus, of the right transverse one and of the confluence of sinuses. The cortical veins, more numerous than usual, had demonstrated a larger caliber. Following the anticoagulant, anticonvulsive and cerebral depletive treatment, the neurological condition had improved to a great extent during a period of 2 weeks, and at the control visit performed two months after the admittance it had been found out that the patient had recovered completely.

Case No. 4

A 24-years-old women, eight months pregnant, had experienced dysgravidia and the premature delivery of a normal baby.

At the admittance in the hospital, the patient had high blood pressure, generalized seizures, attention and memory disorders and a moriatic syndrome.

The native MRI examination with contrast media and the magnetic resonance venography with diffusion measurements had revealed the picture of a superior sagittal sinus thrombosis which included also several cortical veins (the delta sign and the absence of the blood flow, respectively, on the venographic measurements). Associated with these, there had been ascertained peripheral infarctions in the right parieto-occipital region and bilaterally in the precentral area, with discrete hemorrhagic transformation. It had also been present a moderate generalized oedema at the level of both cerebral hemispheres, with the effacement of the gyral pattern and the narrowing of the ventricles. The angiography of the 4 cerebral blood vessels had confirmed the thrombosis of the superior sagittal sinus which had remained permeable only in the area adjacent to the confluence of sinuses, for a length of approximately 3 cm. To be noted the filling up of multiple drainage veins, especially of Trolard-Labbé type, and the routing of the venous circulation towards the sylvian valley and the inferior longitudinal sinus.

Following the anticoagulant therapy administered with great caution, the anticonvulsive and the cerebral anti-edematous treatment for 18 days, the patient's health condition had shown an evident improvement.

Case No. 5

BC, a female patient aged 26-years-old, with a personal history of ureteral lithiasis treated surgically a month previously through lithotrities and the mounting of an ureteral stent, had been admitted in the hospital for occipital headaches, paresthesias and the inability to use the left side extremities. At the neurological examination it had been ascertained the presence of a left hemihypoesthesia and of a slight left hemiparesis.

The axial computed tomography had revealed the presence of a right parietal haemorrhagic softening of the brain and of a left posterior superior temporal ischemic area (Figure 2a).

The late phase of the cerebral angiography of the 4 blood vessels had rendered evident the absence of the physiological rapid flow hypersignal of the superior sagittal sinus (thrombosis of the superior sagittal sinus) (Figure 2b).

Under conservative treatment with antibiotics, anticoagulants and cerebral depletive therapy, the evolution had been favorable, with the evident improvement of the symptoms.

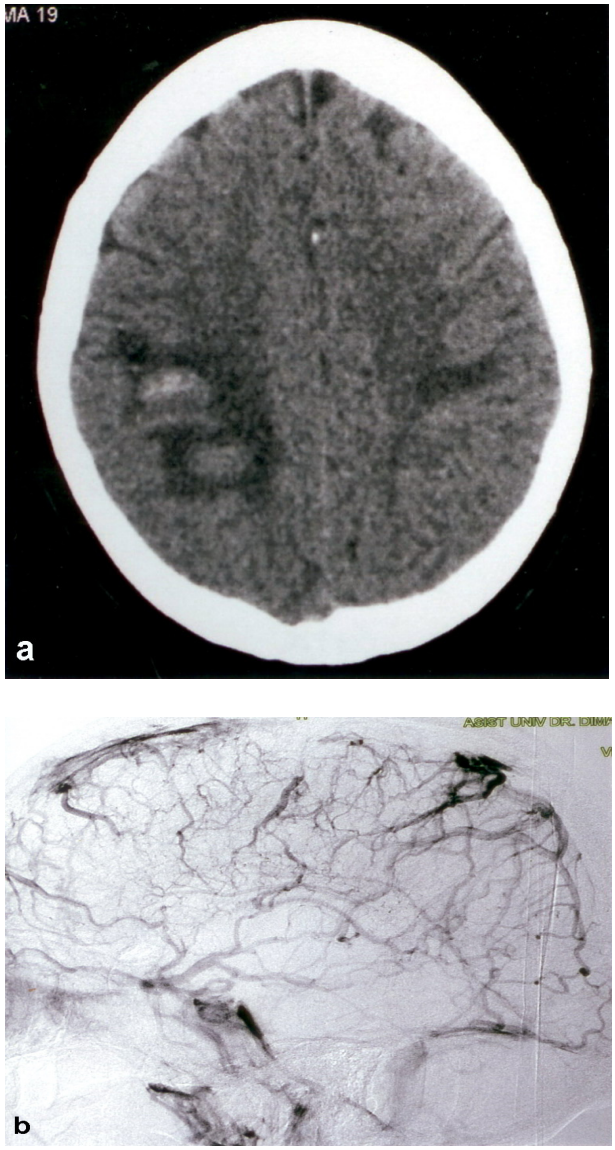


Fig. 2. The computed tomography reveals the presence of a right parietal hemorrhagic softening of the brain and of a left posterior superior temporal ischemic area (a). The late phase of the carotid angiography of the 4 blood vessels (we only show the right lateral image) reveals the thrombosis of the superior sagittal sinus (b).

The repeated computed tomography had revealed the resorbtion of the hemorrhagic focus. 4 months after the moment of admittance, the symptoms had disappeared completely.

Case No. 6

DI, a female patient aged 51-years-old, without a personal history of cardiac events and normal blood pressure, had been admitted in our neurosurgery department with headaches and left crural hemiparesis. The computed tomography had revealed the presence of a right frontoparietal haemorrhagic softening of the brain, while the late venous phase of the carotid angiography had rendered evident the thrombosis of the longitudinal sinus. Gradually, the paresis had expanded to the left upper extremity, and the patient had become somnolent.

Eleven days after the admittance, namely on the 16th of September 2012, it had been performed the surgical intervention for the evacuation of the right frontoparietal haemorrhagic softening area. Postoperatively, the left hemiparesis and the left hemihypoesthesia had improved, but there had been added generalized seizures. 17 days after surgery it had occured a subacute ischemia, with intense cyanosis of the left foot up to the level of the ankle, akinesia and the absence of the pedal pulse.

She had been transferred to a vascular surgery department where it had been ascertained a thrombophilia resistant to activated C protein (PS 62%, PC 81%, AT 92%, APCR-FV 102 - VN>120).

The arterial Doppler examination of the left lower extremity had revealed the thrombosis of the popliteal artery starting at the articular interline.

After the initiation of the treatment with unfractionated heparin, the evolution had been unfavorable, reason for which, on the 5th of October 2012 it had been decided to perform the thrombembolectomy at the level of the left femoro-popliteal and shank arterial axis using the Fogarty catheter through a distal popliteal approach. Postoperatively, the evolution had been unfavorable, with the irreversible ischemia of the left foot, where the popliteo-pedal pulse had been absent. On the 8th of October 2012 it had been performed the amputation in the middle third of the thigh. The postoperative evolution had been good. She had been discharged with a stable respiratory and hemodynamic condition, balanced hydroelectrolitically, with supple amputation stump, the presence of the left femoral pulse, in process of surgical healing.

Case No. 7

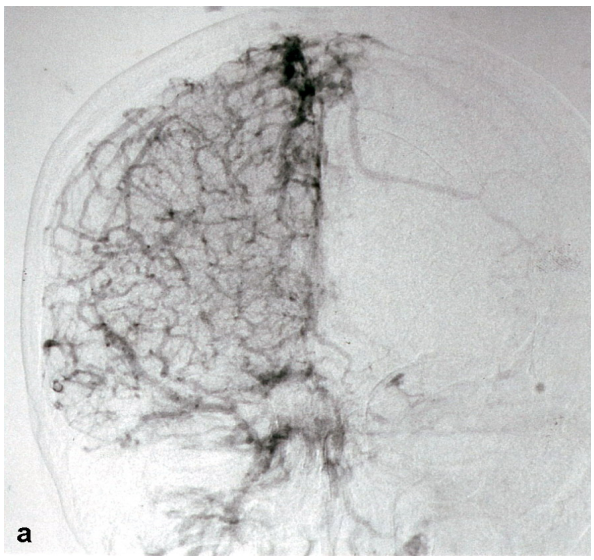
BV, a female patient aged 39-years-old who had been admitted in our cerebrovascular neurosurgery department with the suspicion of subarachnoid haemorrhage. The patient had active pulmonary tuberculosis for which she had received treatment for 3 years.

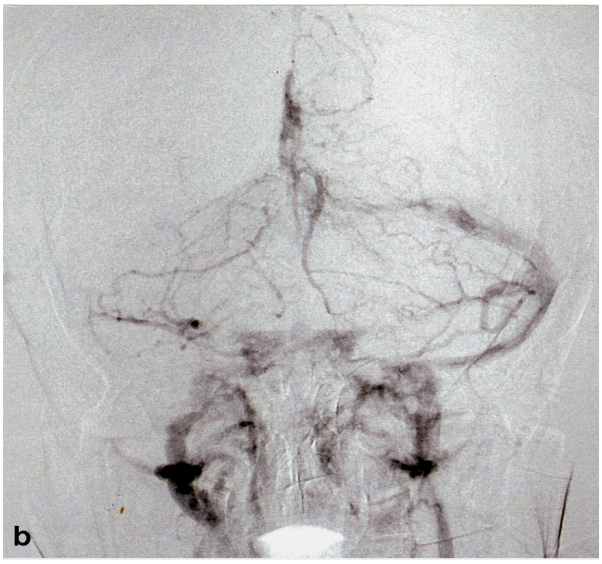
At the admittance, the patient had complained of headache, nausea, vomiting and had moderate left hemiparesis and papillary edema.

The brain CT scan had not revealed any signs of subarachnoid haemorrhage. At the lumbar puncture it had been found a xanthochromic cerebrospinal fluid with 68 leukocytes/cmm, 320 red blood cells/cmm and 0.62 g of proteins /L.

The cerebral angiography of the 4 blood vessels had rendered evident the lack of opacification of the superior sagittal sinus, of the transverse sinus on the left side and of the straight sinus (Figure 3).

Under treatment with antibiotics, anticoagulants, cerebral depletives and symptomatic therapy administered for 2 months, the patient had recovered completely.





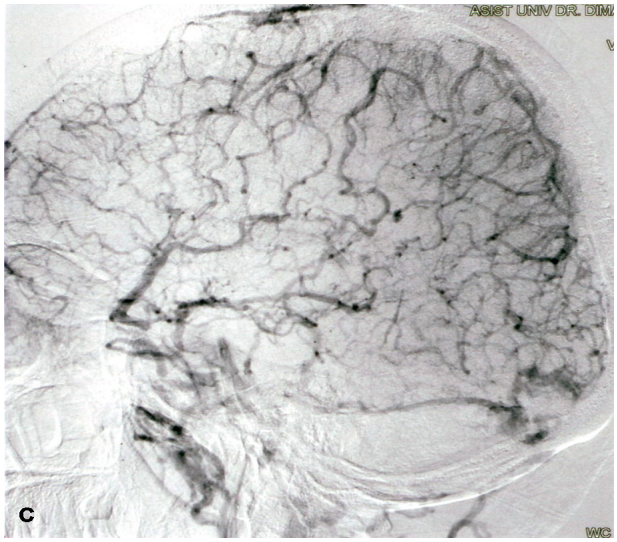


Fig. 3. The anteroposterior views of the late (a) and very late (b) phase of the right carotid angiography, as well as the lateral (c) one reveal the lack of opacification of the superior sagittal sinus, of the straight sinus and of the transverse sinus on the right side.

Case No. 8

BC, a female patient aged 37-years-old, smoker and user of oral contraceptives for several years, who had began to complain of rapidly progressive headaches, vomiting, generalized epileptic seizures and fever, without any detectable infectious cause.

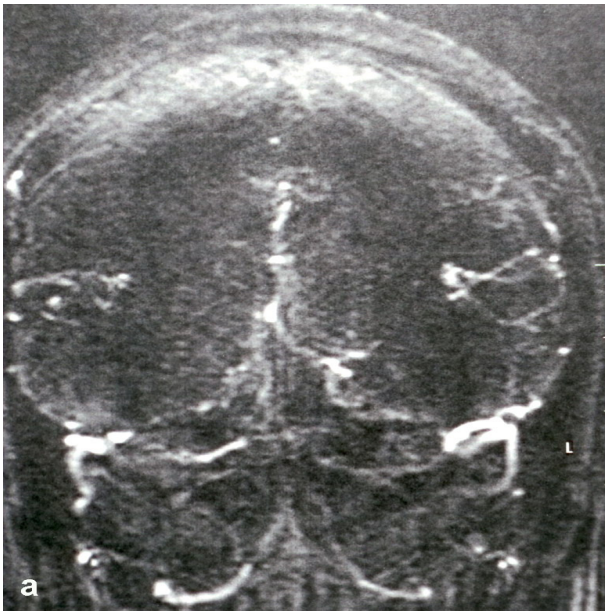
She had been admitted in the hospital because with the generalized tonico-clonic seizures, a suddenly installed left hemiparesis and of a state intense psychomotor agitation.

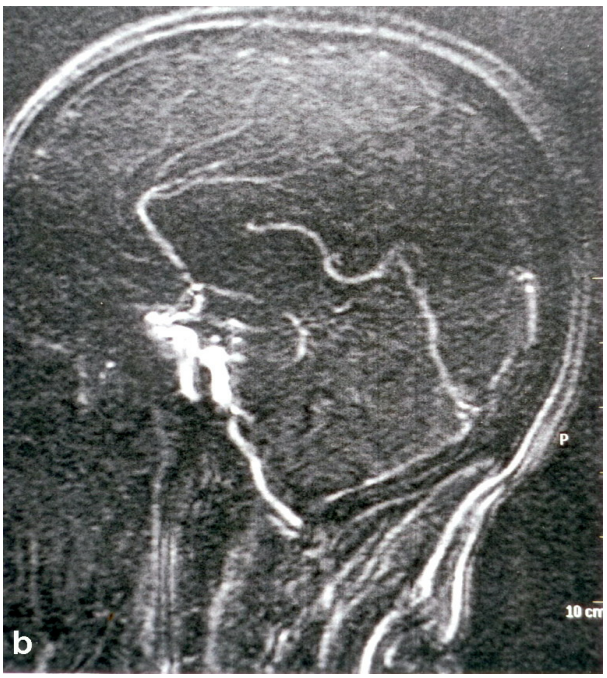
The MRI angiography had rendered evident the absence of the blood flow at the level of the superior sagittal and transverse sinuses, as well as at the level of the intraosseous segments of the internal jugular veins, with the visualization of several hypersignal thrombi in the T1 and T2-weighted sequences at their level. There had been revealed bilateral posterior parietal subarachnoid blood suffusions in hypersignal T1 şi T2 in extracellular methemoglobin stage. Consequently, the MRI angiography had revealed an excessive thrombosis of the superior sagittal and transverse sinuses (Figure 4a, 4b).

The right lateral carotid angiography had rendered evident the complete, massive thrombosis of the superior sagittal sinus, of the right transverse sinus and of the right sigmoid sinus, as well as the presence of a frontal venous malformation (Figure 4c).

Following the treatment with antibiotics, anticoagulants, anticonvulsivants and cerebral anti-edematous agents, the neurological and the general health condition had greatly improved during a 2 week period. The patient had been transferred to a neurology department for the continuation of the treatment.

At the control visit after 3 months it had been found out that the patient had recovered completely.





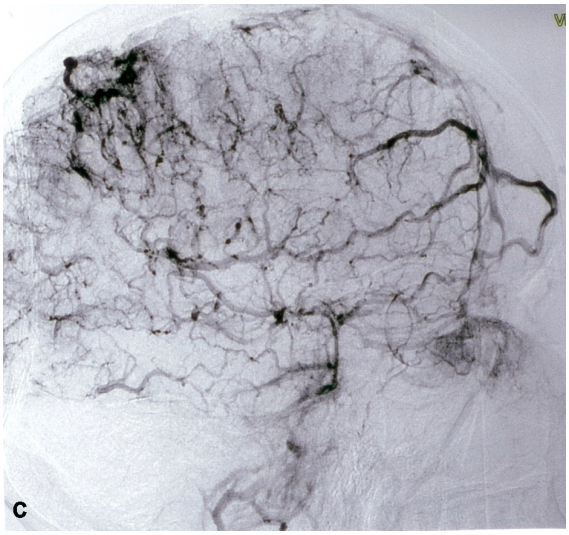


Fig. 4. The magnetic resonance angiography had rendered evident bilateral hypersignal thrombi in the T1 and T2 weighted sequences at the level of the superior sagittal and transverse sinuses, as well as at the level of the intraosseous segments of the internal jugular veins. There can be seen bilateral posterior parietal subarachnoid blood suffusions in hypersignal T1 şi T2 (a, b). The late phase of the right lateral carotid angiography had revealed the massive thrombosis of the superior sagittal sinus, of the right transverse sinus and of the right sigmoid sinus, as well as the presence of a diffuse frontal venous malformation (c).

Case No. 9

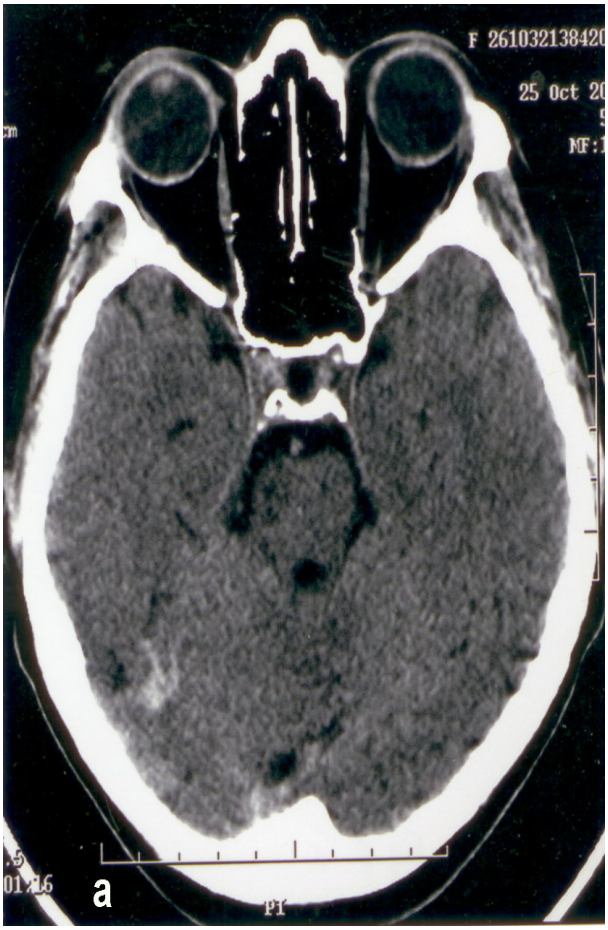
GC, a female patient aged 27-years-old, had started to complain of progressive headaches and to experience vomiting, balance disorders, epileptic seizures and left hemiparesis beginning with the 15th day after an eutocic delivery and papillary edema.

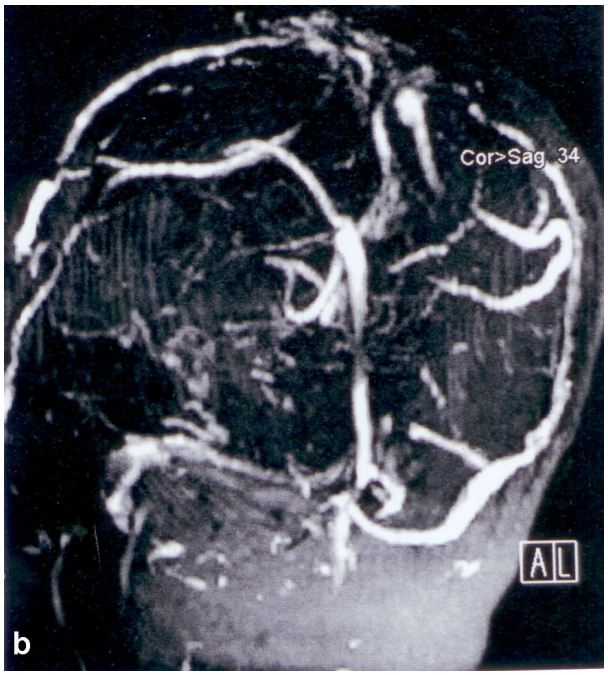
At the hematological examination it had been found leukocytosis with polymorphonuclear cells, hyperfibrinemia and hypertriglyceridemia.

The computed tomography had revealed a small right occipital cerebral hemorrhagic area (Figure 5a).

The magnetic resonance angiography had rendered evident subacute and chronic thrombosis at the level of the transverse, sigmoid, straight and superior sagittal sinuses, with the dilatation of the cortical veins (Figure 5b).

The late venous phase of the right lateral carotid angiography had revealed the lack of opacification of the superior sagittal and right sigmoid dural venous sinuses and of the right side of the transverse sinus, as well as the presence of a parietal venous malformation (Figure 5c).





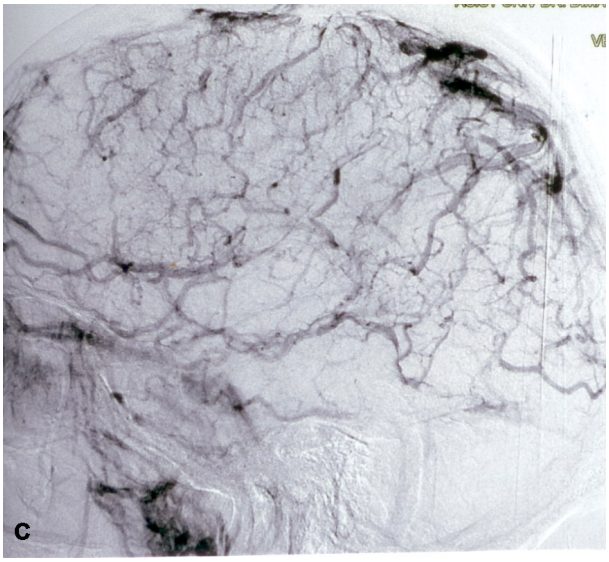


Fig. 5. The computed tomography shows on the axial view (a) a small right occipital hemorrhagic area. The magnetic resonance angiography (b) had rendered evident subacute and chronic thrombosis areas at the level of the superior sagittal sinus and the dilatation of the cortical veins. The right lateral carotid angiography (c) reveals the lack of opacification of the superior sagittal, right transverse and right sigmoid sinuses.

Under the treatment with anticoagulants and antiepileptic therapy administered for 36 days, the patient had recovered completely.

Case No. 10

PM, a female patient aged 34-years-old, had been admitted in the hospital because three days previously she had began to experience right facial-brahial motor seizures, receptive aphasia, headaches, vomiting and gait and balance disorders. At the neurological examination it had been detected right hemiparesis, receptive aphasia, and a right cerebellar syndrome with balance disorders. The medical history of the patient revealed that she had lymphoid leukemia, disseminated lupus erythematosus and recurrent transient ischemic cerebral events. The MRI and the magnetic resonance angiography had revealed subacute thrombosis of the upper longitudinal, inferior sagittal and left transverse sinuses, with subacute hemorrhagic venous infarctions which were more evident in the left parietal area (Figures 6a, 6b).

Following the treatment with anticoagulants, antiepileptic therapy and anti-edematous agents, the neurological condition had improved. The patient had been transferred to a hematology clinic.

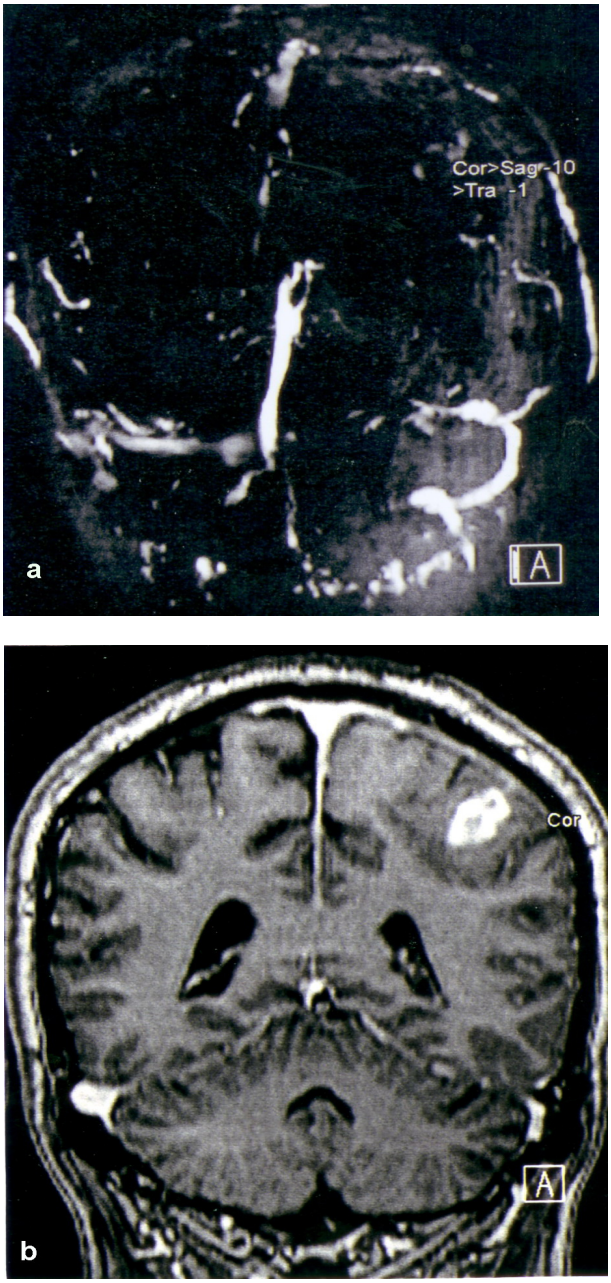


Fig. 6. The magnetic resonance angiography (a), as well as the MRI reveals areas with subacute thrombosis of the superior sagittal, inferior sagittal and left transverse sinuses, with a left parietal subacute hemorrhagic venous infarction (b).

Case No. 11

VR, a female patient aged 53-years-old, had been admitted in our vascular neurosurgery clinic because in the previous three hours she had complained of right hemiparesis, receptive aphasia, headaches, nausea and vomiting.

At the ophthalmic fundus examination it had been ascertained the presence of papillary edema in the left eye. The electroencephalogram had revealed bilateral slow theta waves, but more

pronounced on the left side. The computed tomography had rendered evident small ventricles and the presence of a hypodensity in the superior temporal and cortico-subcortical parietal regions on the left side.

The angiography of the 4 cerebral blood vessels had rendered evident the thrombosis of the upper longitudinal sinus and of the left transverse sinus, the presence of a severe circulatory retardation, as well as the absence of the left upper cortical veins.

Following the anticoagulant and cerebral depletive treatment, the patient's health condition had improved, but she had remained with moderate motor and linguistic sequelae.

Case No. 12

TM, a female patient aged 32-years-old, user of oral contraceptives for several years, had a sudden onset 29 hours previously with headaches, vomiting, balance disorders and aphasia.

The cerebral CT-scan had revealed a posterior and inferior interhemispheric hypertensive band with hematic density.

The cerebral angiography of 4 blood vessels had revealed: the thrombosis of the superior sagittal sinus in its posterior region, the thrombosis of the confluence of sinuses, the thrombosis of the straight sinus, the thrombosis of both transverse sinuses and of the sigmoid sinuses.

At the occipitopetrosal junction the transverse sinus curves downward and backward as the sigmoid sinus, which drains into the internal jugular vein. In this case, the venous drainage had been done anteriorly and posteriorly only through the inferior sagittal sinus, towards the cavernous sinuses.

Under the anticoagulant and neuroprotective treatment, the evolution had been very good.

Case No. 13

AA, a male patient aged 67-years-old, with diabetes mellitus, who had been diagnosed 13 months previously with deep venous thrombosis in both inferior extremities, treated with anticoagulants.

In the day of admittance in hospital, he had fever, headaches, vomiting, gait and balance disorders, a right cerebellar syndrome and papillary edema. Previously, the patient had been hospitalized several times for haemorrhage, because he had antithrombin III deficit. The electroencephalogram had been normal. In the cerebrospinal fluid (CSF) there had been found 113 leukocytes/cmm, 320 red blood cells/cmm and 1.06 g of proteins /L. The venous phase of the angiography of the 4 cerebral blood vessels had revealed the incomplete and irregular opacification of the right transverse sinus and the absence of the sigmoid sinus (Figure 7).

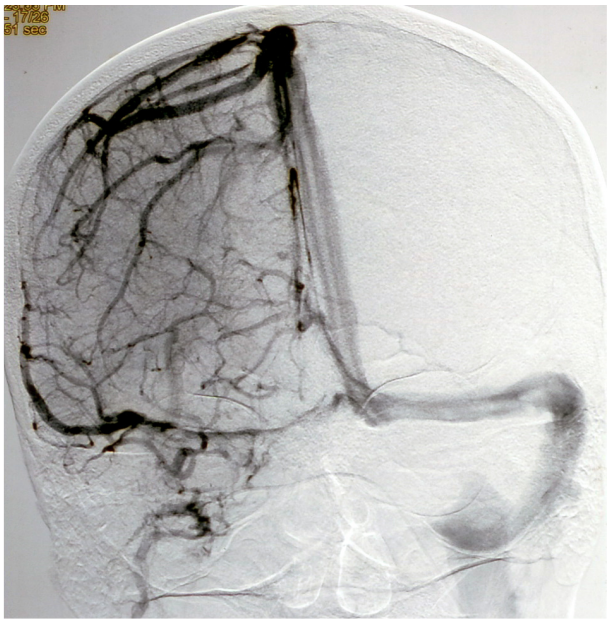


Fig. 7. The anteroposterior view of the venous phase of the right carotid angiography reveals the incomplete and irregular opacification of the right transverse sinus and the absence of the right sigmoid sinus.

In spite of the treatment with antibiotics, anticoagulants and cerebral depletive therapy, the patient's health condition had worsened, and he died 16 days after the admittance.

Case No. 14

HI, a female patient aged 70-years-old, with chronic otitis and neglected left suppurative mastoiditis, had experienced headaches, gait and balance disorders, right hemianopsia, ataxia and fever for 18 days.

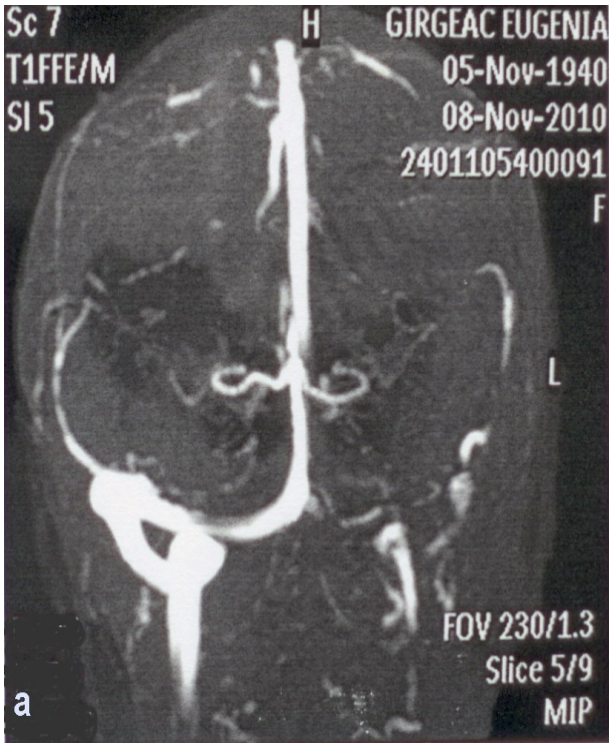
The electroencephalogram had revealed left parieto-occipital slow theta and delta waves.

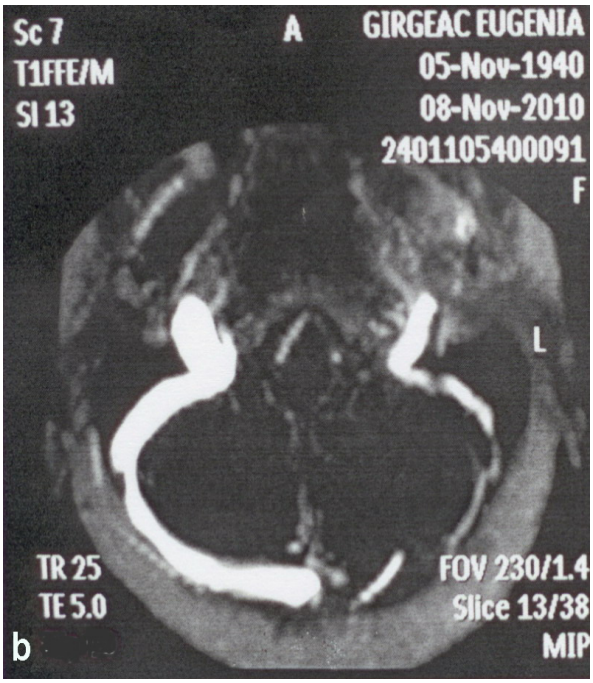
In the CSF there had been found 346 lymphocytes/cmm, 12 red blood cells/cmm and 1 g of proteins /L.

The venous phase of the magnetic resonance angiography had revealed the thrombosis of the transverse and of the sigmoid sinuses on the left side (Figures 8a, 8b).

The computed tomography had rendered evident the presence of relatively small ventricles and of a left temporoparietal cortico-subcortical hypodense area (Figure 8c).

She had received treatment with antibiotics and anticoagulants for three month, and then the patient had been transferred to the otorhinolaryngology department. At the control visit after 4 months it had been found out that the patient had recovered completely.





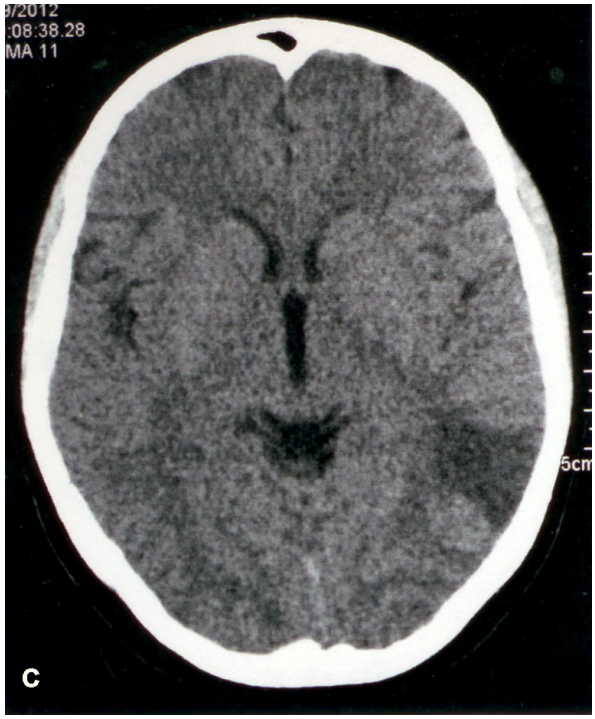


Fig. 8. The venous phase of the magnetic resonance angiography reveals the thrombosis of the transverse and of the sigmoid sinuses on the left side (a, b). The computed tomography renders evident the presence of relatively small ventricles and of a left cortico-subcortical hypodense area (c).

Case No. 15

NC, a female patient aged 36-years-old, with an incorrectly treated right suppurative otomastoiditis, had been admitted in our neurosurgery clinic after 9 days in which she had suffered generalized seizures, headaches and left hemianopsia. In the cerebrospinal fluid collected through lumbar puncture there had been found 138 leukocytes/cmm, 78 red blood cells/cmm and 0.46 g of proteins /L, as well as hypoglycorrhachia.

The late phase of the bilateral carotid angiography had revealed the presence of the thrombosis of the transverse sinus on the right side, and circulatory retardation in the right parieto-occipital arterial network.

The MRI had rendered evident in T2 a right cortico-subcortical parieto-temporo-occipital hypersignal, without arterial systematization.

Following the treatment with antibiotics, anticoagulants and antiepileptic therapy, the neurological condition had improved after three weeks. The patient had been referred to an otorhinolaryngology clinic for specialized medical care.

At the control visit after 5 months it had been found out that the patient did not have any symptoms.

Case No. 16

EA, a female patient aged 51-years-old, had been admitted in our hospital because she had complained for 4 months of headaches, dizziness, and unsystematized balance disorders. At the neurological examination it had been ascertained the presence of a right cerebellar syndrome associated with gait and balance disorders. There was nothing relevant in the patient's medical history.

The ophthalmic fundus, the CSF examination and the EEG had been within the normal limits. The cerebral angiography had revealed the partial thrombosis of the right side of the transverse sinus and of the sigmoid and straight ones, with irregularities of the caliber (Figure 9).

Following the anticoagulant treatment, the symptomatology had a rapid improvement, so that at the control visit performed 72 days after the admittance it had been found out that the patient had recovered completely.

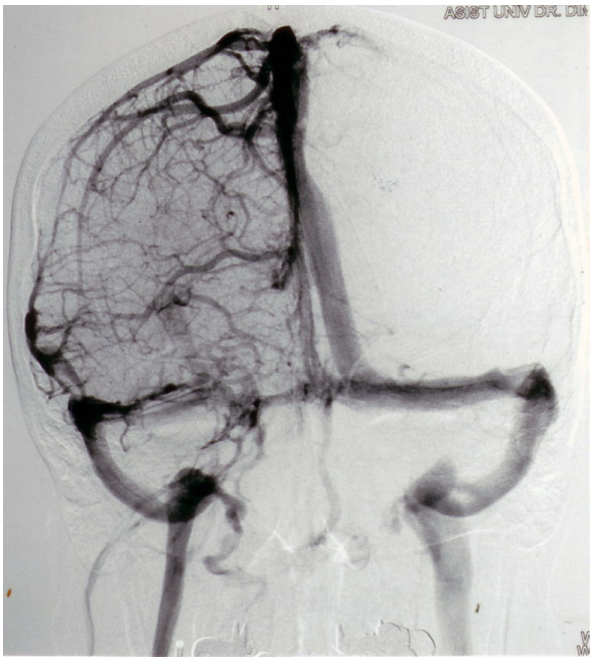


Fig. 9. The anteroposterior view of the late venous phase of the right carotid angiography reveals the partial thrombosis of the right transverse sinus and its caliber irregularities.

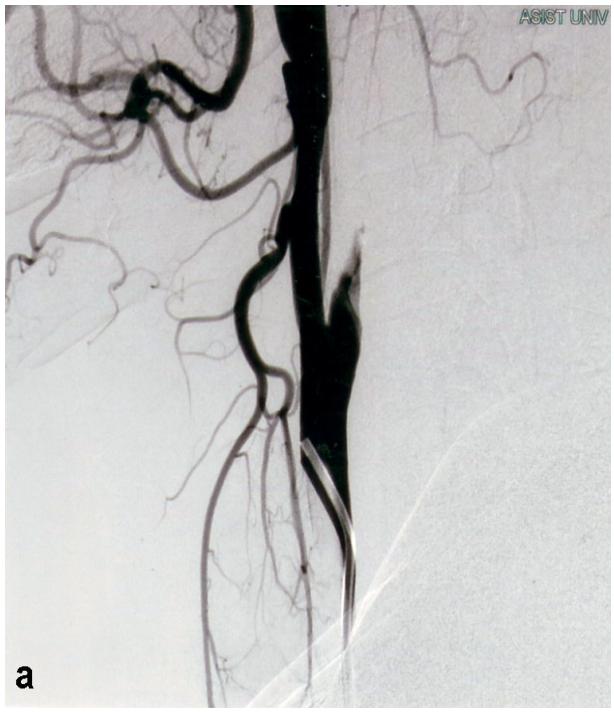
Case No. 17

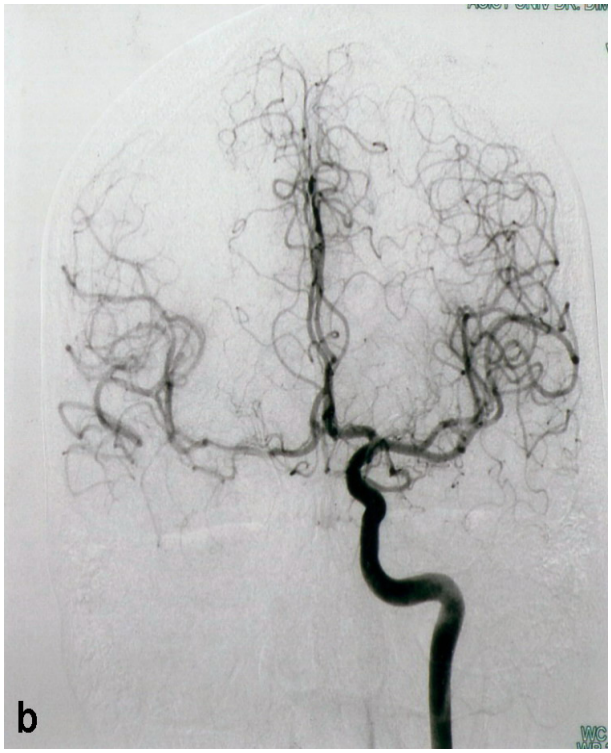
IG, a female patient aged 38-years-old, obese and user of oral contraceptives, had been admitted in our hospital because she had complained for 4 months of progressive headaches, nausea, hyperthermia, an episode of loss of consciousness, left facial paresis and left homonymous hemianopsia. At the ophthalmic fundus examination it had been ascertained bilateral papillary edema.

The hematological examinations she had polynuclear leukocytosis, hyperlipidemia, hypertriglyceridemia and antithrombin III deficit. This antithrombin III deficit had been ascertained in the hematology department where she had been hospitalized several times. The EEG had revealed the presence of several groups of right occipital slow theta waves.

The computed tomography had rendered evident, after contrast, a right temporo-occipital hypodense region, with point-like hyperdense areas and the thickening of the pia mater. In the CSF had been found 16 lymphocytes/cmm, 69 red blood cells/cmm and 0.76 g of proteins /L.

The angiography had revealed a circulatory retardation in the posterior part of the right cerebral hemisphere, the presence of several veins with discontinuous trajectory, and the lack of visualization for the most part of the transverse sinus on the right side and of the right sigmoid sinus (Figure 10).





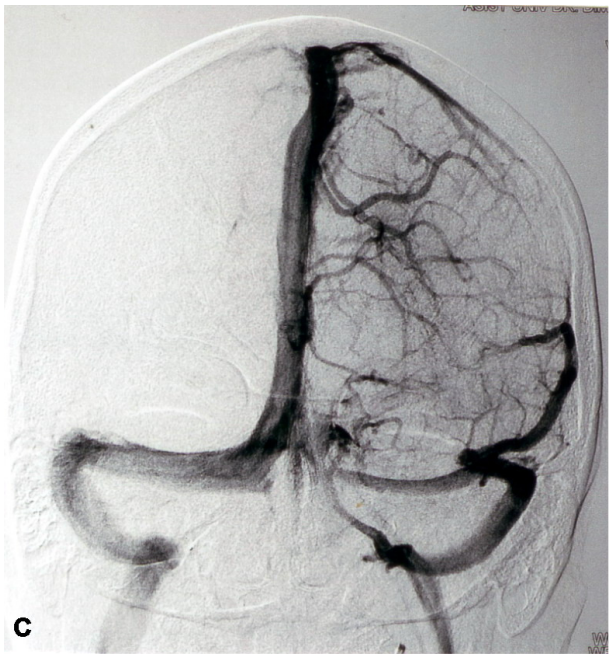


Fig. 10. The anteroposterior view of the right carotid angiography reveals the thrombosis of the internal carotid artery at the level of the neck, approximately 0.5 cm above the bifurcation (a) and the presence of a good collateral circulation through the anterior communicating artery, which is visible on the left carotid angiography (b). The late venous phase of the left carotid angiography renders evident the partial thrombosis of the left transverse sinus (c).

Following the administration of anticoagulant treatment and antiepileptic therapy for 37 days, the patient had recovered completely.

Case No. 18

MS, a male patient aged 42-years-old, had been admitted in the hospital for headaches, vomiting, left hemiplegia, drowsiness and generalized epilepsy which had began 6 days previously.

His medical history had revealed an inadequately treated dyslipidemia.

The angiography of the 4 cerebral blood vessels had revealed the presence of a thrombosis of the right internal carotid artery at the level of the neck and the presence of a good collateral circulation through the anterior communicating artery (Figures 10a, 10b). The venous sinuses had been normal.

The CT scan of the brain had rendered evident a right temporoparietal ischemic area with localized hemorrhage. After 2 weeks, the neurological condition had improved to a great extent.

In the 22nd day after the discharge from hospital, the patient had to be readmitted with headaches, vertigo, fever, tetraparesis and epileptic seizures. The repeated angiography of the 4 cerebral blood vessels had revealed, besides the right internal carotid artery thrombosis, the severe circulatory retardation at the level of the left distal arterial network and the partial visualization of the transverse sinus on the left side (Figure 10c).

Following the anticoagulant and cerebral depletive treatment, the neurological condition had improved.

Case No. 19

PR, a female patient aged 15-years-old, with an inadequately treated chronic frontal sinusitis, had been admitted in our clinic with fever, headaches, nausea, vomiting, and balance disorders which had occurred 8 days previously.

At the neurological examination it had been found out the presence of a right cerebellar syndrome with bilateral papillary edema. The electroencephalogram had revealed a focus of right temporal slow delta waves.

In the CSF there had been found 80 leukocytes/cmm, 240 red blood cells/cmm and a quantity of protein of 0.68 g/L.

At the hematological examination it had been found a moderate polynuclear leukocytosis.

The venous late phase of the angiography of the 4 cerebral blood vessels had revealed the partial thrombosis of the transverse sinus (Figure 11).

Following the treatment with anticoagulants and antibiotics, the neurological and the general health condition had been notably improved. She had been referred to an otorhinolaryngology clinic for the treatment of the sinusitis.

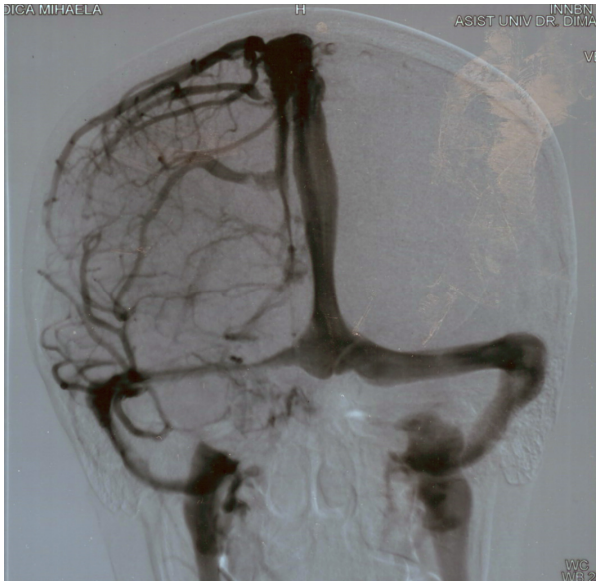


Fig. 11. The anteroposterior view of the late phase of the right carotid angiography reveals the partial thrombosis of the right transverse and sigmoid sinuses.

At the control visit 3 months after the initiation of the treatment, we had found out that the patient had recovered completely.

Case No. 20

TN, a female patient aged 35-years-old, obese and with high blood pressure, who had a thrombophlebitis at the level of the left lower extremity and pulmonary embolism after a cholecystectomy performed for gallbladder lithiasis 21 days before.

At the admittance in the hospital, the patient had gait and balance disorders, a left cerebellar syndrome, fever and headaches.

The angiography of the 4 cerebral blood vessels had revealed the lack of visualization of the left transverse sinus, which was free only for a length of 1 cm from its origin (Figure 12).

Following the anticoagulant and antibiotic treatment, the neurological condition had improved.

The patient had been transferred to a neurology department for the continuation of the treatment.

Case No. 21

ND, a female patient aged 25-years-old, had been admitted in our clinic for headaches, nausea and vomiting which had occured suddenly 2 days after the cesarean section. At the neurological examination it had been found out the presence of a left neocerebellar syndrome and papillary edema.

At the MRI it had been ascertained a normal appearance in the T1 and T2 weighted sequences and after the administration of gadolinium. In the venous flow sequences it had been rendered evident the absence of the venous flow at the level of the left transverse sinus, a suggestive appearance for the thrombosis of the sinus in question.

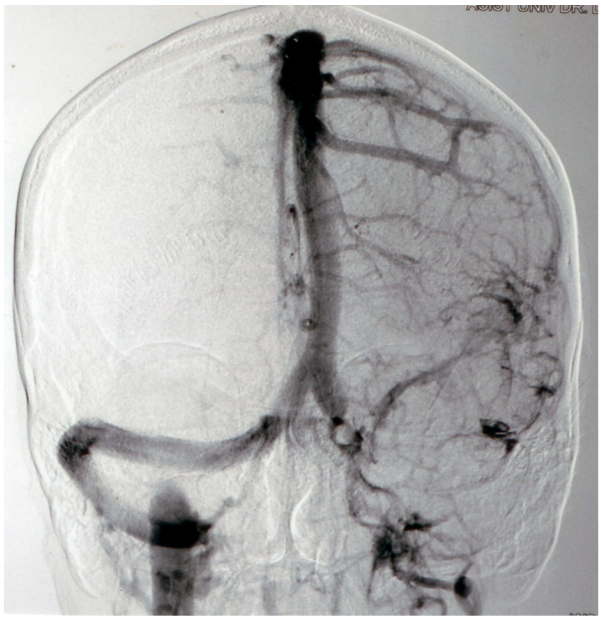


Fig. 12. The anteroposterior view of the late venous phase of the left carotid angiography demonstrates the almost total thrombosis of the left transverse and sigmoid sinuses. It is visualized only an internal segment of approximately 1 cm.

The computed tomography of the brain had been normal.

Under the anticoagulant treatment which had been administered for 10 days, the neurological condition had improved.

The control MRI examination had revealed the same appearance as the previous one, without evidence of the repermeabilization of the left transverse sinus.

The patient had been transferred to a neurology department for the continuation of the treatment.

At the control visit after 6 months, we had found out that the patient had recovered completely.

Case No. 22

GE, a female patient aged 63-years-old, with an inflammatory rheumatic disease, had been admitted as an emergency in the rheumatology department on the 23rd of September 2010 with the diagnosis of rheumatoid arthritis, hepatic steatosis and fracture without displacement of the distal

epiphysis of the right radius. Following the anti-rheumatic drug treatment and the immobilization of the left arm in a plaster cast for 4 weeks, the health condition had improved.

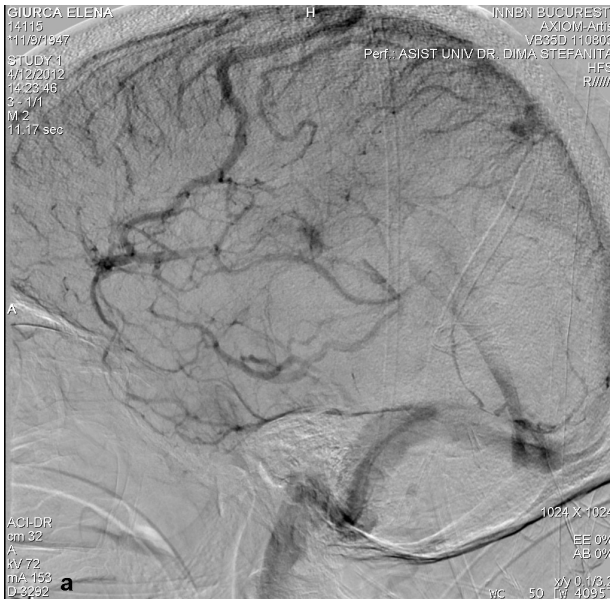
In October 2012 she had all of a sudden presented a left hemiplegia and a confusional state, reason for which she had been admitted in a local hospital.

After a week she had been transferred to our neurosurgery department in coma, and papillary edema because of on the ischemic cerebrovascular accident with hemorrhagic transformation. The computed tomography had confirmed the diagnosis of right temporo-parietal cerebral ischemia with hemorrhagic transformation, while the angiography had revealed the thrombosis of the vein of Labbé (Figures 13a, 13b).

Six days after the admittance, the patient had entered in a coma.

It had been performed an emergency surgical intervention during which it had been excised the hemorrhagic softening of the brain. The brain was edematiated, with a black coloration, while the vein of Labbé was indurated, with a pencil-like appearance. It had been resected together with the temporal cerebral cortex and the hemorrhagic softening of the brain.

The second day after the surgery, the patient had regained consciousness, but the motor deficit in both extremities on the left side had remained unchanged. Due to this fact, the patient had been transferred to a motor recovery department.



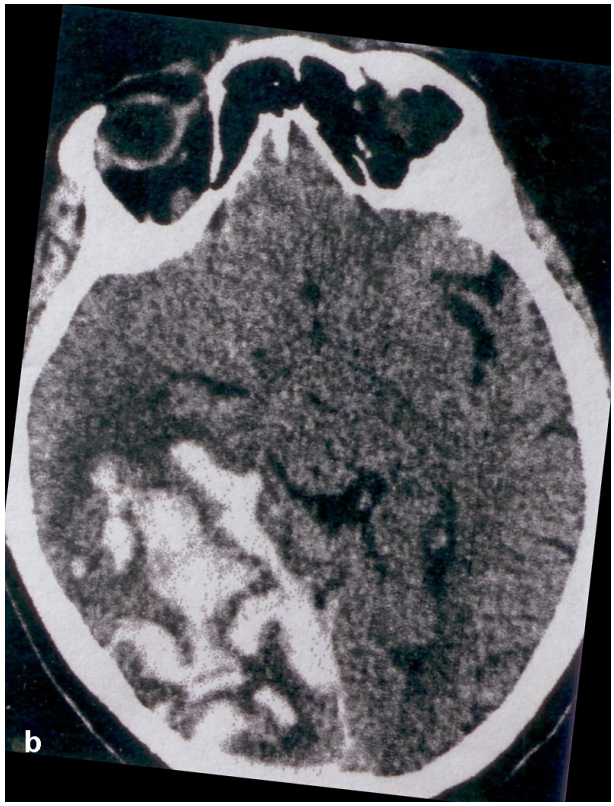


Fig. 13. The right carotid angiography (the late phase) shows the thrombosis of the vein of Labbé (a). The computed tomography reveals the presence of an extensive left temporo-parietal hemorrhagic softening of the brain which had been evacuated surgically (b).

The diagnosis and comments about the cases

We had a number of 22 patients with cerebral venous thrombosis. In 12 of them it had been affected the superior sagittal sinus. The average age of all the 22 patients had been 44 years, with extremes of 15 and 70 years. The average age in question, as well as that of the patients in the literature, shows that the thrombosis of the dural sinuses and that of the cerebral veins occurs in younger subjects, and its incidence in older people is underestimated.

In our cases, we had observed that the women had been more affected than the men.

Hence, out of the 22 cases, 17 (77.27%) had been women, and only 5 (22.72%) men (See Table 1).

The deficiencies concerning the clinical diagnosis of our patients had been dependent on their symptomatic polymorphism, age, sex, the moment of the first occurrence of the symptoms, the type and the localization of the parenchymal lesion, the affected sinus and the severity level of the thrombosis.

Table 1

Our own cases with thrombosis of the dural sinuses

The dural sinus involved	No. of cases	Gender M	Gender F	Age in years	Average age	Results		
						Cured	Improved	Deceased
SSS	6	3	3	56, 41, 49, 24, 26, 51	41 years old	3	3	0
SSS and TS	5	0	5	39, 37, 27, 34, 53, 32	35 years old	4	2	0
TS and SS	5	1	4	67, 70, 36, 51, 38	52 years old	4	0	1
ST	4	1	3	42, 15, 35, 25	29 years old	4	0	0
Vein of Labbé	1	0	1	63	63 years old	0	1	0
Total	22	5 (22.72%)	17 (77.27%)	_	44 years old	15 (68.18%)	6 (27.27%)	1 (4.54%)

SSS = superior sagittal sinus
TS = transverse sinus
SS = sigmoid sinus

Thus, we had two categories of patients in our clinic. For the patients in the first category, the diagnosis of cerebral venous thrombosis could be established from the beginning. We are talking here of young people with an evocative context which consisted of infections of the otorhinolaryngology sphere, postpartum conditions, repeated systemic phlebitis, or various surgical interventions. The onset of the disorders under the more or less complete appearance of an intracranial hypertension syndrome had been associated with signs of focal cerebral lesions manifested through epileptic seizures or deficitary symptoms (cases No. 2, 3, 4, 5, 6, 9 10, 21, 22, etc.).

The papillary edema had been present in 7 (31.81%) of our patients (cases No. 7, 9, 11, 13, 17, 21, 22).

In most of the cases, the differential diagnosis had to be done with meningo-encephalitis.

For the patients in the second category, the ascertaining of the diagnosis of cerebral venous thrombosis had been much more difficult, due to the mode of onset and to the atypical symptoms which had been in the foreground (cases No. 7, 8, 11, 16, 17, etc.)

The onset had been **acute**, in less than 48 hours, in 5 (27.72%) of our patients (cases No. 1, 2, 4, 11 and 12) with obstetrical or infectious problems; **subacute**, between 48 hours and 30 days, in 13 (59.09%) of the patients (cases No. 3, 5, 7, 8, 9, 10, 14, 15, 18, 19, 20, 21 and 22), and insidious or **chronic**, between 30 days and several months, in 4 (18.18%) of our patients with a clinical picture of intracranial hypertension and a predominantly inflammatory etiology (cases No. 6, 13, 16 and 17).

The cerebral venous thrombosis had a brutal onset with coma in 2 (9.09%) of the patients (cases No. 1 and 6) or, most frequently, with headaches in 18 (81.81%) patients, motor deficits in 11 (50%), epileptic seizures in 9 (40.90%), cerebellar disorders in 6 (27.27%), homonymous hemianopsia in 3 (13.63%), aphasia in 4 (18.18%) and moriatic syndrome in 1 (4.54%) patient (See Tables 2, 3, 4, 5 and 6).

The onset had also the appearance of subarachnoid hemorrhage (cases No. 7 and 8) or that of an arterial cerebrovascular accident, especially when it had been accompanied by carotid thrombosis (case No. 18), tumors, or cerebellar abscess (cases No. 19, 20, 21).

These forms of thrombosis of the dural sinuses and of the cerebral veins must be differentiated from the benign intracranial hypertension. A special alteration, with particular symptomatic characteristics had been represented by the psychiatric disorders of moria type (case No. 4).

Table 2

The clinical signs, diagnosis and treatment of the patients with thrombosis of the superior sagittal sinus (SSS)

Case No.	Age in years	Gender	Symptoms	Diagnosis	Etiological and risk factors	Cerebral complications	Treatment	Results
1	56	M	Coma, Left hemiplegia	Angio MRI, Classical angiography	Chronic alcoholism	Right parietal hemorrhagic softening of the brain	Surgical evacuation of the hemorrhagic softening of the brain	Improved
2	41	M	Epilepsy, Right hemiparesis and mixed aphasia, Headaches	Classical angiography	Operated prostate adenoma Thrombophlebitis of the inferior extremities	_	Anticoagulant Antiepileptic	Cured
3	49	M	Headaches, Epilepsy, Left hemiparesis	Classical angiography	Operated sigmoid stenosis	Small bilateral subdural hematomas	Anticoagulant Antiepileptic	Cured
4	24	F	Epilepsy, Moriatic syndrome	MRI, Classical angiography	Premature delivery	Bilateral parieto-occipital infarctions and hemorrhages	Anticoagulant Antiepileptic	Improved
5	26	F	Headaches, Left hemiparesis	CT scan, Classical angiography	Operated kidney lithiasis	Cerebral oedema with right parietal hemorrhagic foci	Anticoagulant Antibiotic	Cured
6	51	F	Left hemiparesis Headaches	CT scan, Classical angiography	Thrombophilia resistant to C protein	Hemorrhagic softening of the brain	Surgical evacuation of the hemorrhagic softening of the brain	Improved

Table 3

The clinical signs, diagnosis and treatment of the patients with combined thrombosis of SSS and TS

Case No.	Age in years	Gender	Symptoms	Diagnosis	Etiological and risk factors	Cerebral complications	Treatment	Results
7	39	F	Headaches, Vomiting, Left hemiparesis Papillary edema	Classical angiography, CT scan	Active pulmonary tuberculosis	Subarachnoid hemorrhage	Anticoagulant Antiepileptic	Cured
8	37	F	Headaches, Fever, Vomiting, Epilepsy, Left hemiparesis	Classical angiography, Angio MRI	Oral contraceptives	Subarachnoid hemorrhage	Anticoagulant Antiepileptic	Cured
9	27	F	Headaches, Vomiting, Epilepsy, Left hemiparesis Papillary edema	Angio MRI	Eutocic delivery	Right occipital cerebral hemorrhage	Anticoagulant Antibiotic Antiepileptic	Cured
10	34	F	Right facio-brahial motor seizures, Aphasia, Balance disorders	MRI, Classical angiography	Acute lymphoid leukemia Lupus erithematosus	Left parietal cerebral infarction	Anticoagulant Antiepileptic	Improved
11	53	F	Right hemiparesis, Aphasia, Headaches, Vomiting Papillary edema	Classical angiography, CT scan	_	Left rolandic hemorrhagic softening of the brain	Anticoagulant	Improved
12	32	F	Headaches, Vomiting and balance disorders, Aphasia	Classical angiography, CT scan	Oral contraceptives	Interhemispheric hemorrhage	Anticoagulant	Cured

Table 4

The clinical signs, diagnosis and treatment of the patients with thrombosis of the transverse and sigmoid sinuses

Case No.	Age in years	Gender	Symptoms	Diagnosis	Etiological and risk factors	Cerebral complications	Treatment	Results
13	67	M	Headaches, Fever, Gait and balance disorders, Right dysmetria Papillary edema	Classical angiography	Diabetes mellitus, Thrombophlebitis of the inferior extremities, Antithrombin III deficit	CSF with white blood cells and red blood cells	Anticoagulant Antibiotic	Deceased
14	70	F	Headaches, Gait and balance disorders, Right hemianopsia	Angio MRI, CT scan	Left otomastoiditis	CSF with white blood cells and increased albumin	Anticoagulant Antibiotic	Cured
15	36	F	Headaches, Left hemianopsia, Epilepsy	Classical angiography, Angio MRI	Right suppurated otomastoiditis	CSF with white blood cells, red blood cells and hypoglycorrhachia	Anticoagulant Antiepileptic Antibiotic	Cured
16	51	F	Headaches, Vertigo, Balance disorders, Right cerebellar syndrome	Classical angiography	_	_	Anticoagulant	Cured
17	38	F	Headaches, Fever, Epilepsy, Left hemianopsia Papillary edema	Classical angiography	Oral contraceptives, Hyperlipidemia, Antithrombin III congenital deficit	Hemorrhagic softening of the brain	Anticoagulant Antibiotic	Cured

Table 5

The clinical signs, diagnosis and treatment of the patients with thrombosis of the transverse sinus

Case No.	Age in years	Gender	Symptoms	Diagnosis	Etiological and risk factors	Cerebral complications	Treatment	Results
18	42	M	Headaches, Fever, Tetraparesis, Epilepsy	Classical angiography	Dyslipidemia, Right carotid thrombosis	Right temporoparietal ischemic softening of the brain	Anticoagulant Cerebral anti-edematous	Deceased
19	15	F	Headaches, Fever, Right cerebellar syndrome	Classical angiography	Chronic frontal sinusitis	Polynuclear leukocytosis, CSF with white blood cells and red blood cells	Anticoagulant Antibiotic	Cured
20	35	F	Fever, Gait and balance disorders, Left cerebellar syndrome, Headaches	Classical angiography	Thrombophlebitis of the left lower extremity, Pulmonary embolus, Cholecystectomy	_	Anticoagulant Antibiotic	Cured
21	25	F	Headaches, Vomiting, Left neocerebellar syndrome Papillary edema	Angio MRI	Cesarean section	_	Anticoagulant	Cured

Table 6

The clinical signs, diagnosis and treatment of the patients with thrombosis of the vein of Labbé

Case No.	Age in years	Gender	Symptoms	Diagnosis	Etiological and risk factors	Cerebral complications	Treatment	Results
22	63	F	Left hemiplegia, Confusional state Coma Papillary edema	CT scan, Classical angiography	Rheumatoid arthritis, Hepatic steatosis	Ischemic cerebrovascular accident with hemorrhagic transformation	Surgical evacuation of the hemorrhagic softening of the brain	Improved

Consequently, the various clinical signs (headaches, epileptic seizures, focal deficits, disorders of the consciousness and papillary edema) encountered in these patients could be grouped in the following classical acute clinical pictures: intracranial hypertension, focal neurological signs, epilepsy, acute encephalopathy, or a combination of the four elements listed before.

The diagnosis was frequently delayed due to the wide spectrum of clinical symptoms and to the often subacute and chronic onset. However, the early diagnosis of the cerebral venous thromboses had been crucial since the early anticoagulation had greatly reduced the severe disabilities and even the risk of a fatal outcome, without promoting intracranial hemorrhage.

Risk factors

As etiological or risk factors, in our cases we had registered the surgical interventions in 5 (22.57%) patients, the thrombophlebitis of the lower extremities in 3 (13.63%), the use of oral contraceptives in 3 (13.63%), otomastoiditis in 2 (9.09%), the eutocic or premature delivery in 2 (9.09%), dyslipidemia in 2 (9.52%), the antithrombin III deficit in 2 (9.09%), obesity in 1 (4.76%), carotid thrombosis in 1 (4.76%), frontal sinusitis in 1 (4.54%), chronic alcoholism in 1 (4.54%), lymphoid leukemia in 1 (4.54%), active pulmonary tuberculosis in 1 (4.54%) and the resistance to the protein C in 1 (4.54%). In some of the patients there had been present more than one etiological or risk factor.

Etiology

The multiple causes of the thrombosis of the dural sinuses and of the cerebral veins can be divided in three categories: **primary, secondary** (which cover the situations where the production mechanisms of the thrombosis in question can not provide a complete explanation) and **idiopathic**.

Among the main etiologies there are the cranial **traumatisms**, their frequency being very difficult to appreciate. The direct traumatisms of the SSS are known to be part of the war pathology[24]. Nevertheless, the less intense traumatisms can, even without fracture, also cause cerebral venous thromboses which are probably the consequence of the endothelial lesions[25,26,27].

The thrombosis of the dural sinuses and of the cerebral veins associated to the **arterial occlusions** (case No. 18) are very rare, and they have the appearance of a septic or neoplasic occlusion of the cavernous sinus complicated by the occlusion of one of the internal carotid arteries[28,29], or that of an aseptic arterial occlusion which is accompanied by the thrombosis of the drainage veins in the area affected by the arterial occlusion[30,31].

The incidence of the septic etiology of the thrombosis of the dural sinuses and of the cerebral veins is in continuous decrease due to the use of antibiotics and to the developments of the medical care in the otorhinolaringology area of interest. In 1967, these causes were representing 41% of the observations of Krayenbühl (1967)[32], while in the series of Rousseaux *et al.* from 1985[33], the frequency had been between 10% and 15%. The most frequent localization of this kind of the cerebral venous thrombosis had been the cavernous sinus.

The involvement of the superior sagittal sinus had represented 15% of the cases reported by Southwick *et al.* (1986)[34].

The infections in the otorhinolaringology area of interest had been the origin in 3 of our cases (cases No. 14, 15 and 19), they being the main cause of the dural sinuses thrombosis.

The central nervous system (CNS) infection secondary to the frontal and mastoid air sinus infection was a common entity in the past.

The spectrum of the intracranial complications of the frontal air sinus or mastoid infection at that

time is in contrast with our experience today, although the principal infectious agents remain familiar.

Cairns (1930)[35] emphasized the role of the dura mater in blocking the infection, in addition to its role in preventing the leakage of cerebrospinal fluid[36].

The infection remains a perennial problem in the neurosurgical practice, as it had been throughout the history of surgery. According to Becham and Tyler (1912)[37], the annual incidence of encephalitis, pyogenic bacterial meningeal infections, and brain abscess may be as high as 17 cases per 100 000 of the population. These cases are treated with aggressive regimens of antibiotics which often accompany the surgical drainage (Chalif et al., 2013)[38].

Otitis media was estimated by Cairns (1930)[35] to account for 50% of the cerebral abscess in the pre-antibiotics era.

By 1945, the casualties due to brain abscess decreased to 3%, compared with 27% in the pre-penicillin era[36].

The existence of an antibiotic substance associated with moldy bread has been known about by physicians since antiquity. The Edwing Smith Papyrus, a summary of the Egyptian medical knowledge written circa 1500 BC, provides details on the treatment of traumatic wounds using moldy bread to stave off or treat the infection[39]. The fungal species responsible for the antibiotic effect of the moldy bread is penicillium. However, the antibiotic effect of penicillium was reported to the Royal Society of Medicine in London in 1875 and had been published in 1877 by the polymath John Tyndall[40].

It was Alexander Flemming who had recognized the potential clinical applications of penicillium in 1928, at St Mory's Hospital in London[41].

Cairns wrote more than once about the use of the sulfonamide drugs as antimicrobial agents, and the successes achieved with them[42].

However, his lack of success with them for particular infections, including the purulent pachymeningitis, spurred on his further research into penicillin[43–45].

The epileptogenic side effects of penicillin had not been recognized until later. The first report of the neurotoxicity of penicillin was published in 1945[46].

The success of the antibiotics in the treatment of infections has led to their excessive use in the clinical practice, leading to the evolution of the antibiotic-resistant organisms.

However, reviewing Cairns' work and reflecting on the discovery[41] and on the early clinical use of penicillin reminds us how devastating were the infections in the pre-antibiotics era, and how fortunate are the neurosurgeons to have antibiotics available for both the prevention and the treatment of the neurological infections[38].

During the evolution of acute leukemia (case No. 10) and of the lymphomas, the thrombosis of the dural sinuses or of the cerebral veins can complicate the therapeutic induction phase, which involves the action of the cytolytic compounds and/or the direct effect of the chemotherapeutic agents[47].

Observations of venous thrombosis had been also reported in the myeloproliferative syndromes (Vaquez' Disease and essential thrombocythemia), which cause hyperviscosity of the blood, functional platelet disorders and general factors, such as dehydration [49–54].

Fujimaki *et al.* (1986)[55] had observed a similar case during the evolution of high altitudes polyglobulia.

The constitutional disorders of hemostasis are a rare etiology of the thrombosis of the dural sinuses and of the cerebral veins which occur during the recurrent and familial multiple phlebitis. In the respective individuals it can be found a deficit of antithrombin III[56,57], (our cases No. 13 and 17), of protein S[58] or of protein C[59] (case No. 6).

Feldenzer *et al.* (1987)[60] had considered drepanocytosis to be a cause of the cerebral venous thrombosis, while Gettelfinger and Kokmen (1977)[61] and Donhowe and Lazaro (1984)[62] thought that the hemolytic process, as that in the paroxysmal nocturnal hemoglobinuria (Evans' Syndrome), also played a role[63].

The systemic diseases which can be complicated by the thrombosis of the dural sinuses and of the cerebral veins are dominated by the Behçet's disease[64,65]. Behçet's disease is an affliction of the mucous membranes characterized by thrush-like ulcerations accompanied by general symptoms, erythematous skin eruptions, ocular manifestations (iritis, retinal hemorrhages), and occasionally, nervous symptoms (clinical picture of disseminated encephalomyelitis).

The presence of a perivascular infiltrate at the level of the venous sinuses attests the existence of a vasculitic process[66].

Here, the thrombosis simulates the clinical picture of a cerebral pseudotumor, and for this reason, its frequency can be underestimated.

The thrombosis of the dural sinuses and of the cerebral veins is rare in association with other systemic diseases, such as lupus erythematosus[67,68] (our case No. 10).

There had also been observed cerebral venous thrombosis during the evolution of nephrotic syndromes associated with blood clotting disorders[69].

Finally, the cerebral venous thrombosis can also be associated with a cerebral vascular pathology, such as dural arteriovenous fistulas[70,71], which can generate drainage shunts.

The superficial craniocerebral tumors (tumors of the cranial vault, metastases, meningioma etc.) can also lead to the thrombosis of the dural venous sinuses through direct or infiltrative compression[72,73].

The presence of an extra-cranial solid tumor is often considered to be a determining factor, but the physiopathology of the thrombosis of the dural sinuses or of the cerebral veins can seldom be determined in such cases. The presence of metastatic emboli is often debatable[29], as is sometimes debatable the individualization of the intravascular coagulopathies and of the hypercoagulability states[74].

Within the framework of secondary etiologies, we bestow a special importance to the thrombosis of the dural sinuses and of the cerebral veins during pregnancy. Carroll *et al.* (1966)[75] had estimated their incidence to be 1 case for 2.5×10^2 pregnancies, while the study conducted by Cross et al. (1968)[76] had reported only a single case in 6×10^5. This complication continues to remain valid today, fact demonstrated by our cases No. 4, 9 and 21.

Anyhow, the thrombosis of the dural sinuses, of the cerebral veins and the other vascular malformations[77,78] are rare complications of pregnancy itself.

It usually occurs during the second week after delivery[79].

The multitude of pathogenic hypotheses ventured had attributed this complication to the endogenous hyperestrogenemia, the intravascular coagulopathies and the paradoxical embolisms favored by the labor.

It had also been established that the thrombosis in question occurs as the result of a pregnancy complicated with systemic phlebitis[80,81], after a dystocic delivery, and especially after a pregnancy toxemia[47]. The latter etiology had been found in 107 cases out of the 129 in the series of Srinivasan (1983)[79].

The same pathogenic uncertainties are also applicable for the thrombosis of the dural sinuses and of the cerebral veins developed postoperatively, as well as for those which are attributed to the oral contraceptives[8,81]. We had two such cases (cases No. 8 and 17).

In these patients, the identification of the anti-ethinyl-oestradiol antibodies had remained a delicate interpretation process[82].

The idiopathic etiology refers to the thrombosis of the dural sinuses and of the cerebral veins where, in spite of a thorough etiological analysis and an extended follow up, the causes remain uncertain. Anyhow, they are for a long time the first and only manifestation of a subjacent disease, such as the carcinomatous meningitis[11]. They can be found, as an average, in 10% to 20% of the cases. In our series of 22 patients, we had two such cases, representing 9.52% of the analyzed patients.

In the cerebrospinal fluid we had found occasionally pleiocytosis which had been mostly lymphocytic, hyperproteinemia, hypoglycorrhachia and red blood cells.

In some of the patients there had been encountered more than one risk factor.

In summary, the risk factors for the thrombosis of the dural sinuses and of the cerebral veins can be permanent and transient[7,83].

In the category of the transient risk factors we include: sarcoidosis, intestinal inflammatory diseases, malignant tumors of the viscera and of the CNS (meningiomas, metastases, malignant infiltrations), leukemias (lymphomas and myeloproliferative sindromes), polycythemia, thrombocythemia, anemias (drepanocytosis, paroxysmal nocturnal hemoglobinuria, hemorrhage, etc), coagulation disorders (protein C or S deficiency, antithrombin III deficiency, factor V Leiden mutation, factor II mutation (G2021A), hyperhomocysteinemia and homocystinuria, disseminated intravascular coagulation, excess of factor VIII, nephrotic syndrome, antiphospholipid syndrome, lupus anticoagulant, cardiopathies, especially the congenital ones, arteriovenous fistulas, collagen diseases (Behçet's disease and Sjögren's sindrome, Wegener's disease, lupus erythematosus)[84].

In the category of the permanent risk factors there are included the infectious diseases of the

CNS (meningitis, empyema, abscesses) and of the face and neck (sinusitis, mastoiditis, cellulitis), as well as the systemic ones (septicemia, endocarditis, etc.), the pregnancy and the postnatal confinement, mechanical factors (jugular catheterization, head injuries, neurosurgical interventions, lumbar puncture), certain medicines (androgens, tamoxifen, thalidomide, erythropoietin, heparin, epsilon aminocaproic acid, intravenous immunoglobulins, lithium, ecstasy, vitamin A, steroids, the hormone replacement therapy, oral contraceptives), severe dehydration, the plasminogen deficiency, hemorrhagic strokes, space replacing lesions, heparin-induced thrombocytopenia[83–85].

In some of the patients there are present multiple risk factors. According to Guenther and Arauz (2011)[4], in 85% of the patients is encounter a single risk factor, while in 40% of them there are at least two. The congenital or genetic thrombophlebitis is encountered in at least 20% of the cases[4,7,10].

The most commonly encountered risk factor is represented by the oral contraceptives.

The etiology in other cases is dominated by the genetic and acquired prothrombotic conditions[4].

The neuroradiological diagnosis

The diagnosis of the thrombosis of the dural sinuses and of the cerebral veins had been ascertained with the aid of the **neuroradiological explorations**.

The earliest attempt to demonstrate occlusion of the sinus were by direct sinography, the contrast medium being introduced directly into the sinus through the anterior fontanelle[86–93] or injected directly into the venous channels e.g. superior longitudinal sinus, ophthalmic or diploic veins.

Because of the high degree of risk this method had been abandoned.

The cerebral radioisotope investigation, which is a non-invasive procedure, had been proved to be useful occasionally, for the diagnosis of this condition[94–96].

The perfusion imaging technique had brought forth some new data. Thus, Schaller *et al.* (2003)[97], who had used the positron emission tomography, had rendered evident the reduction of the brain blood flow and the concomitant development of the venous infarction of the cerebral parenchyma after the ligature of the superior sagittal sinus. Kawai *et al.* (2005)[98] had observed a regional increase of the cerebral blood volume in a young adult with superior sagittal sinus thrombosis. Keller *et al.* (1999)[99] and Mullins *et al.* (2004)[100] had showed that the prolongation of the mean transit time and the increase of the cerebral blood volume suggest a venous congestion, as opposed to the pattern that had been observed in the patients with ischemic arterial stroke (the prolongation of the mean transit time and the reduction of the cerebral blood volume).

The investigative modalities mentioned above did not dispose of pathognomonic characteristics for the ascertaining of an accurate diagnosis.

The definitive diagnosis of the thrombosis of the dural sinuses and of the cerebral veins had been ascertained with the aid of the neuroradiological investigations.

So, in the past it had been alleged that the diagnosis can be confirmed only by the carotid angiography[101,102], assertion that had been refuted in the present day.

However, in the last two decades, the neuroradiological study played an important role in the diagnosis and the management of the cerebral venous thrombosis[6,103–111].

The purpose of the noninvasive and of the invasive investigative modalities is that of ascertaining the vascular and parenchymal changes associated to the medical conditions. Further on, we discuss radiological findings, and the utility of various imaging modalities to confirm the diagnosis of CVT in suspected cases.

Head Computed Tomography (CT) is the most frequently performed imaging study for the evaluation of the patients with recently installed headaches, focal neurological abnormalities, seizures, or changes in the mental status.

According to Saposnik *et al.* (2011)[5], the CT scan may detect an alternative diagnosis or demonstrate the existence of venous infarcts or hemorrhages. However, it has a poor sensitivity and it reveals the direct signs of cerebral venous thrombosis in only one third of patients.

The signs of cerebral thrombosis on the CT scans include the hyperdensity in the area of a sinus or cortical vein and the filling defects, especially in the superior sagittal sinus (empty delta sign), in the contrast-enhanced studies[5–7].

The thrombosis of the posterior portion of the superior sagittal sinus may appear as a dense triangle, the dense or filled delta sign.

Cerebral computed tomographic venography (CTV) provides a rapid method for the detection of the cerebral venous thrombosis, especially in patients with contraindications to MR imaging[5].

Compared with the density of the adjacent brain tissue, a thrombus may be isodense, hypodense, or of mixed density[5,7,109, 110,112–123].

The magnetic resonance imaging (MRI) of the head combined with the magnetic resonance venography (MRV) are the most sensitive investigations for the detection of the cerebral venous thrombosis in the acute, subacute and chronic phases[2,7]

The cerebral intraarterial angiography with venous phase imaging and direct cerebral venography are less commonly used to ascertain the diagnosis of cerebral venous thrombosis, given the availability of MRV and CTV (Lafite *et al*., 1999; Bousser, 2000; Yoshikawa et al., 2002; Saposnik et al., 2011; Piazza, 2012)[5,7,15,124,125].

The thrombophilia testing

Because of the high frequency of thrombophilias among the patients who develop cerebral venous thrombosis, it should be performed a screening for hypercoagulable conditions[5,7]. According to Piazza (2012)[7], the thrombophilia testing should include the evaluation for factor V Leiden mutation, prothrombin 20210 gene mutation, lupus anticoagulants, anti-cardiolipin antibodies, hyperhomocysteinemia, protein C, protein S and antithrombin deficiencies.

In the case of a young patient who complains of persistent headaches or who develops a stroke in the absence of the known risk factors, there can be raised the suspicion for thrombosis of the dural sinuses and of the cerebral veins. The confirmation can be achieved only through imaging investigations that show the absence of the flow at the level of dural sinus or of a cerebral vein and render evident the intraluminal thrombosis, with the involvement of the cerebral parenchyma[2,5-7,10]. We had only a single case with pure cerebral venous thrombosis (case No. 22).

The post-thrombotic cerebral complications had been manifested through hemorrhagic softening of the brain in 8 (36.36%) of our patients, subarachnoid hemorrhage in 4 (18.18%), and through the softening of the brain in the absence of hemorrhagic foci in 1 (4.54%) patient.

Treatment

In the acute phase, all our patients had received anticoagulant therapy, and for those with an infectious etiology it had been added antibiotic treatment.

The anticoagulant medication administered immediately after the admittance in the hospital had consisted of unfractionated heparin administered intravenously or low-molecular-weight heparin administered subcutaneously, as a preparation for the oral anticoagulation with antivitamin K.

During the acute phase, all our patients had also received pain relief treatment for the amelioration of the pain and agitation, as well as treatment for the reduction of the intracranial hypertension using osmotic diuretics, positioning and hyperventilation, accompanied by the monitoring of the intracranial pressure, and antiepileptics.

According to Einhäupl et al. (2006)[1], Guenther and Arauz (2011)[4] and Saposnik *et al.* (2011)[5], the use of glucocorticoids has not been proved beneficial.

Occasionally (cases No. 1 and 6) it had been necessary to evacuate a hematoma or a hemorrhagic softening of the brain. In other situations, the use of the decompressive craniectomy is a mandatory therapeutic procedure[1, 5, 85].

In the patients with associated cortical lesions or in those with inaugural seizures, the use of antiepileptics had reduced the frequency of the crises.

The antibiotics, the antiepileptic and the cerebral antiedema drugs require little discussion.

With several exceptions[126,127] the surgical solution had been rarely used. In our cases we used surgery only in 2 patients.

The objectives of the anticoagulant treatment target are to prevent extension of the thrombus, to facilitate recanalization of the occluded sinuses and cerebral veins, and to prevent deep vein thrombosis and pulmonary embolism[5].

For this reason, the anticoagulant treatment must be initiated as soon as possible, using unfractionated heparin or low-molecular-weight heparin.

The dosages are established depending on the body weight.

The international guidelines recommend the use of the heparin as primary treatment in the adult patients with cerebral venous thromboses, regardless of the presence of the basic hemorrhagic lesions[128].

For the patients with cerebral venous thrombosis caused by transient risk factors, Caprio and Bernstein (2012)[129] recommend 36 months of anticoagulant treatment.

For the patients with cerebral venous thrombosis caused by severe or combined trombophilias, the same authors recommend the permanent administration of the anticoagulant treatment for the prevention of the recurrences.

With respect to **aspirin,** there are no observational studies or control trials to evaluate its role in the treatment of cerebral venous thrombosis.

The steroids can play a role in the patients with cerebral venous thromboses by reducing the vasogenic edema, though they can also accentuate the hypercoagulability.

In a case-control study conducted by the ISCVT[130], 150 patients treated with steroids at the discretion of the investigator had been compared with 150 patients who did not receive steroid medications.

At the end it had been ascertained that the patients treated with steroids had the same characteristics as the control subjects, but the latter had not developed vasculitis. After 6 months, it had been observed that the subjects treated with steroids had a higher risk of dependency and death. However, after the exclusion of those with vasculites, malignancies, inflammatory diseases and infections, the difference had disappeared. For the patients with lesions of the cerebral parenchyma that had been visualized on the CT scan or on the MRI, the tendency for death or dependency in the steroids treated patients had been 4.8 times higher than in the control group[5].

Other studies, which had also resorted to various analytical approaches, had revealed the same.

The antibiotics must be used in the patients with local or systemic infections (otitis, mastoiditis, etc., or meningitis, septicemias etc., respectively) because they can cause thromboses of the dural sinuses and of the cerebral veins, both adjacent and at a distance.

In the patients in whom there is the suspicion of infection and cerebral venous thrombosis, there must be initiated the adequate antibiotic treatment accompanied by the surgical drainage of the suppurative focus (subdural empyema or purulent collections of the paranasal sinuses).

The local or systemic venous **thrombolytic therapy** with urokinase and rt-PA, as well as the mechanic thrombectomy, can be attempted in the patients with unsatisfactory results in spite of an adequate anticoagulant treatment[131].

However, in the absence of randomized clinical studies, it is difficult to allege if the interventional radiology is safer and more effective than the heparin therapy. A potential benefit refers only to the severe cases where the heparin therapy had failed (Einhäupl *et al.*, 2006; Caso *et al.*, 2008; Guenther and Arauz, 2011; Saposnik *et al.*, 2011; Caprio and Bernstein, 2012; Coutinho *et al.*, 2012)[1,4,5,128,129,131].

We had used the heparin treatment in the first 48 hours for the patients with acute, dramatic onset, followed by the administration of low-molecular-weight heparin fractions (for approximately 2–3 months), and then we had prescribed oral anticoagulant treatment (Plavix, Aspenter) for several months.

The treatment after the acute phase

According to Ferro and Canhao (2004)[10] the antithrombotic treatment must be continued to prevent the recurrences in 2 to 7% of the cases, and the extracerebral venous thrombosis in up to 5% of the cases. In these patients it must be maintained an INR between 2 and 3. It is not known the definite duration of the treatment. According to Caprio and Bernstein (2012),[129] in the case of the transient risk factors, the duration of the anticoagulant treatment must be between 3 to 6 months. In the cases with idiopathic cerebral venous thromboses, or in those with moderate thrombophilia, the anticoagulant treatment will be administered for a period of 6 to 12 months. In the patients with severe thrombophilia, as well as in those with an associated cancer, the respective treatment must be permanent.

The pregnancy, especially during the third trimester and in the puerperial period is a frequent thrombogenic conditions.

For the cerebral venous thromboses which occur during pregnancy, it is indicated the use of low-molecular-weight heparin from the moment of diagnosis until birth.

Likewise, the anticoagulant treatment with low-molecular-weight heparin or with antivitamin K will continue to be administered for at least 6 weeks after birth[5].

Saposnik *et al.* (2011)[5] and Caprio and Bernstein (2012)[129], do not counter-indicate a future pregnancy in a woman with a history of cerebral venous thrombosis because the risk of recurrence is very low.

Nevertheless, if during the pregnancy and the postpartum period it is absolutely necessary to use low-molecular-weight heparin, the teratogenic risk of warfarin has to be taken into account.

The use of estrogen oral contraceptives must be stopped[5,129].

The epileptic seizures had been reported in 5–16% of long-term survivors[132.133,134]. The epileptic

seizures in the chronic phase are encountered in some of the patients who already had seizures during the acute phase, in those with cerebral hemorrhage and in those with focal deficits. These seizures occur more than 2 weeks after the confirmation of the diagnosis of CVT[135–136]. In the respective cases, the optimal duration of the antiepileptic treatment should be at least one year.

The headaches afflict between 25%[137] and up to 60% of the survivors[18], although Baumgartner *et al.* (2003)[138] have reported lower percentages of up to 14%.

The most frequent headaches are benign, of a migraine or high pressure type.

The severe headaches requiring bed rest or admittance in hospital had been reported in 14% of the patients[10].

In such cases, it must be excluded the possibility of a recurrence.

In the cases with chronic ICH (intracranial hypertension) and a normal MRI, there had been proposed repeated lumbar punctures, acetazolamide (500–1000 mg/day) and a lumbar-peritoneal shunt[3].

The focal neurological deficits had been reported in 12 to 18% of the patients who had been followed-up for a long period of time after a cerebral venous thrombosis[18,137,138]. These include visual field defects, cognitive impairment, motor deficits and aphasia[18,138]. **The vision** had been affected in 6.7% of the patients in the ISCVT study, while the severe visual loss had been encountered in < 1% of the patients[85].

In the Lille study, 3 patients (5.5%) had visual field defects, while 2 (3.6%) of them had optic atrophy with an important decrease of the visual acuity ([18]).

The visual field defects had been observed in 10% of the patients that had survived the cerebral venous thrombosis[18]

However, the visual loss is not a frequent symptom in the acute phase of the cerebral venous thrombosis because only 30% of the patients may have papillary edema[10] which, if not treated adequately, can lead to optic atrophy and even blindness. The permanent optic atrophy in the acute phase may be caused by the increased intracranial pressure[132].

Biousse *et al.* (1999)[13] had systematically examined 59 out of the 160 patients with cerebral venous thrombosis and isolated intracranial hypertension syndrome, and had observed in the end that only 3 (5%) of them had developed optic atrophy with severe visual loss.

Although it is a rare complication, the vision impairment must be actively investigated and treated.

If the vision continues to deteriorate in spite of the administration of acetazolamide and the repeated lumbar punctures, it should be resorted to either a lumboperitoneal shunt, or an optic nerve sheath fenestration[1].

The cognitive disorders which had remained after the cerebral venous thromboses had been very frequent. After 18.5 months of evaluation, de Bruijn et al. (2000)[139] had observed that 35% of their patients had cognitive impairment, while 40% of them had lifestyle restrictions. Out of these, 20% had memory disorders, 28% had speech disorders, 28% of them had various degrees of impairment of the constructional abilities while 33% had visual-spatial orientation and planning disorders.

After a follow-up of 46 months, Appenzeller *et al.* (2005)[137] had found out the presence of cognitive disorders in 17% of the patients. Buccino *et al.* (2003)[140], had additionally observed the presence of depression and working memory deficits.

After 12 months from the onset of the cerebral venous thrombosis, Madureira *et al.* (2001)[141] had investigated the professional status, the cognitive performance, the depressive symptoms and the quality of life in 15 patients. At the end of this study, the above mentioned authors had found out that 46% had resumed their previous occupation, 33% had changed their job for a period of time, while 20% of them had retired. Only 2 patients had demonstrated neuropsychological impairment. One of them had remained aphasic, while another had memory and verbal fluency impairment.

However, half of the survivors of a cerebral venous thrombosis had complained of pain, while 2/3 of them suffered from anxiety or depression, despite a seemingly good general health condition.

Out of the 34 patients with cerebral venous thrombosis, studied by Buccino *et al.* (2003)[140] after one year from the acute event, 3 had non-fluent aphasia, 6 of them working memory deficits, while another 6 suffered from depression.

The thrombosis of the deep venous sinus represented by the internal cerebral veins, the basal veins of Rosenthal, the straight sinus and the great vein of Galen, causes bilateral panthalamic infarctions, abulia, executive function disorders, amnesia, disorders of consciousness and coma. The recovery degree is variable, but the memory

deficits, the executive function disorders and the behavioral problems can persist[142–145].

The dural arteriovenous fistulas in the patients with cerebral venous thromboses are not frequent. The cerebral venous thromboses and the arteriovenous fistulas can develop simultaneously, both in the acute and in the chronic phases. Tsai *et al.* (2004)[146] had reported the presence of cerebral venous thrombosis in 39% of the patients with dural arteriovenous fistulas. However, the thrombosis of the cavernous, lateral or sagittal sinuses can also induce the development of dural arteriovenous fistulae[147], while the pial fistulae can develop after the thrombosis of the cortical veins[148].

RESULTS

The evolution of our cases, with all their clinical and etiological polymorphism, had been towards complete recovery in 15 (68.18%) of the patients, towards improvement in 6 (27.27%) patients, while 1 (4.54%) had died. Therefore, the results had been generally good and very good, because 21 (95.45%) of our cases had improved or had recovered completely, while only one (4.54%) had died. The patient in question, aged 67 years old, had diabetes mellitus, deep venous thrombosis of the inferior extremities and antithrombin III deficit.

In the literature, the mortality rates had been between 10 and 20%[11,33,149], while in the 1960s it had been between 35 and 50%[32,150,151,152].

After the introduction of the modern diagnostic and treatment methods, the percentage of the improved cases and of those with complete recovery had remained relatively stable, fact that demonstrates that many of the thromboses of the dural sinuses and of the cerebral veins can have a spontaneous benign evolution.

The invalidating sequelae which are manifested through focal and epileptic neurological deficits are encountered in approximately 15% to 30% of the cases.

Prognosis

The 21 patients who survived had been followed up between 6 and 18 months. Within this time period, we had found out that 1 (4.76%) patient, who had been declared completely recovered, had epileptic seizures.

The other neurological sequelae had been manifested through headaches, in 7 (33.33%) cases, unilateral motor deficits in 2 (9.52%), visual field deficits in 1 (4.76%), cognitive disorders in 1 (4.76%) and the emergence of new thrombotic events in 1 (4.76%).

Consequently, the prognosis is good under treatment[4,83].

However, during the acute phase, in 23% din of the patients it can be observed an aggravation which is often caused by the development of a focal hemorrhagic lesion in 39% of the cases[10,21].

Masuhr and Einhäupl (2008)[153] had found out that the mortality is three times higher in the patients with epileptic seizures.

Other factors that worsen the prognosis are the age over 37 years, the male gender, the state of consciousness evaluated using the Glasgow Coma Scale under 9, the changes in the mental functions, the deep cerebral venous thromboses, the intracranial hemorrhage, the papillary edema, the worsening of the anterior lesion, the new deficits, neuroinfection and the malignant tumors[4].

In a retrospective study conducted on 91 patients, Putaala *et al.* (2010)[154] had found out that the residual headaches had been more frequent in those patients in whom the recanalization did not occur during the first 6 months.

With regard to the functional evolution, Girot *et al.* (2007)[21] had observed that only 5.1% of the patients who had been enrolled in the ISCVT study had been left with severe residual disabilities, while 70–80% of the cases had recovered completely within 2 months.

In 3.7% of the cases there had been reported venous thromboses outside the brain[155].

The patients with post-thrombotic conditions or with deep vein thromboses of the lower extremities had a higher risk to develop recurrent thromboses.

The relapse rate had varied between 0% in the first year and up to 12% after 6.5 years[16,156].

The great majority of the patients heal without sequelae, but the individual prognosis is difficult to ascertain. The identification of the hereditary higher risk factors for the thrombotic diseases helps in taking measures for the avoidance of the acquired risk factors.

The morbidity and the mortality rates associated with the cerebral thrombotic diseases are low (between 3 and 9,4%) due to their early diagnosis and to the efficient antithrombotic drugs.

Therefore, the long-term prognosis of the patients with thrombosis of the dural sinuses and of the cerebral veins is generally very good. The survival rate after the acute phase of cerebral venous thrombosis is quite high, but some of the patients remain with neurological and psychiatric problems. These include focal neurological deficits, headaches, epileptic seizures, cognitive impairment and depression. It is therefore important to follow up these patients, even if they initially seem to have recovered completely.

DISCUSSIONS

In our cases, the transverse sinus and the superior sagittal sinus had been the most frequent seat of the cerebral venous thrombosis. The inferior sagittal and the petrosal sinuses had not been affected, while the cavernous sinus had been always thrombosed only together with other sinuses or cerebral veins.

The most frequent location of thrombosis had proved to be the veins on the convexity, without the involvement of the profound veins.

In case of bilateral involvement of the veins in question, regardless of the extension, their injury had been asymmetrical. Anyhow, the thrombosation of the isolated cortical veins, or even of the circumscribed ones, causes irreversible circulatory and cerebral disorders. In such cases, the type and the intensity of the secondary brain lesions (for ex., the haematoma) had proved to be more important for the evolution of the primary disease than the spreading of the thrombosis.

In rare cases, the veins on the convexity had been obstructed together with the great cerebral vein of Galen. The lethal isolated thrombosis of the great vein of Galen and of the internal cerebral veins occurs only in children.

In the majority of the cases, the thrombosis of the veins in question occurs in combination with the obstruction of one or both lateral sinuses.

The thrombosis of the pial veins occurs in the context of obliterating thrombophlebitis, while the thrombosis of the great vein of Galen occurs as a consequence of migrans thrombophlebitis or of certain processes which involve severe coagulation disorders.

The lateral sinuses are involved in the context of vicinity inflammatory processes, posttraumatic thrombosis and thrombophlebitis migrans.

In one of our patients (case No. 18) the internal carotid artery had also been obstructed. In other cases from our group, the venous thrombosis had occurred following the obliteration of the visceral veins and of those in the extremities (caused by the thrombophlebitis migrans), certain postpartum and postoperative thrombosis, as well as certain severe general coagulation disorders.

In all the patients we had described, the thrombotic process had begun in the superior sagittal sinus, in the transverse sinuses, or in the vein of Labbé. It had been difficult to ascertain the primary localization of the thrombosis in the processes expanded over a large number of sinuses and cortical veins.

Martin (1941)[157] had drawn the conclusion that the thrombosis occurs at the point of communication between two ascendant pial veins, a short distance before the emergence of the venous trunks from the subarachnoid space. According to the concept of Claude (1911)[158], following the fresh sinus thrombosis, besides the obstruction of the cortical veins, the primary thrombosation of the cerebral veins also occurs.

Depending on the expansion of the cerebral venous thrombosis, the tissular alterations spread across variable territories of the convexity.

According to Huhn (1965)[159], in most of the cases it is involved the cerebral cortex of one, or even both hemispheres. These authors had ascertained a preponderance of the involvement of the parasagittal and biparietal regions, followed by, in descending order, the parieto-temporal, parieto-frontal and parieto-occipital convexities.

The subcortical bleedings had occurred mostly in the corresponding areas of the parietal lobes.

The topography of the hemorrhagic cerebral infarctions had depended less on the position of the thrombosed segment of the sinus, and more on the trajectory of the thrombosed cerebral vein which drains in the sinus in question. The hemorrhagic infarctions, often confluent, spread from the parieto-temporal cortex into the depth of the white matter. Massive bleedings also occur with a similar frequency at the limit between the parietal, temporal and occipital lobes.

In the frontal lobe there is a less frequent occurrence of hematomas.

In the not very complicated cases, it is frequently involved only the cortical layer.

As the process is more severe, the more deeply the big hemorrhagic infarctions, the massive bleedings and the necroses penetrate in the white

matter. Surprisingly, the internal cerebral veins and the great vein of Galen are only seldom thrombosed.

The basal ganglia and the brainstem are involved only in the cases where there is the thrombosation of both the internal cerebral veins and of the great vein of Galen.

Consequently, the pathological changes are dependent on the number of thrombosed cerebral veins.

As the blood in the cerebral white matter crosses repeatedly the internal cerebral venous system, the right sinus, the connexions with the lateral sinus and the Sylvian fissure veins, the first tissular changes occur usually at the limit of the cerebral cortex, that is, in the border region between the internal and the external venous systems. The white matter changes become pronounced when the thrombosis also involves the intracerebral veins, or when the evolving cerebral oedema leads to phlebostasis in the internal cerebral venous system.

The thrombosation of the internal venous system leads infrequently to the development of cerebellar and brainstem tissular changes.

CONCLUSIONS

The thrombosis of the dural sinuses and of the cerebral veins, which is rarely encountered in our days, generally afflicts the younger people, but especially the women in association with the specific hormonal factors.

In two thirds of the patients with cerebral venous thrombosis it can be identified a direct cause or a prothrombotic factor.

This disease must be suspicioned in all the people with atypical vascular manifestations, as well as in those with headaches of unspecified causes. The diagnosis of thrombosis of the dural sinuses and of the cerebral veins is easy to be ascertained with the aid of computed tomographic venography (CTV), of magnetic resonance venography (MRV) and of angiography. The cerebral angiography and the direct cerebral venography are required only in the cases where the CTV and the MRV are inconclusive, or whenever an endovascular procedure is considered. The management of the cerebral venous thrombosis includes the treatment of the basal conditions, the symptomatic treatment, the prevention or the treatment of the complications of the increased intracranial pressure and of the venous infarction, as well as the anticoagulant treatment. The correctly administered anticoagulant treatment reduces the incidence of the neurological and psychiatric sequelae, the death rate and that recurrences.

The duration of the anticoagulation therapy must be individualized and adapted to the risk factors that predispose to recurrences.

The endovascular procedures and the hemicraniectomy must be rigorously evaluated before they are put into practice.

For the accurate evaluation of the efficacy and the duration of the alternative treatments for cerebral venous thrombosis there are required large randomized prospective trials.

The prognosis is generally favourable for the patients with the occlusion of the lateral sinus, while it is pejorative for those with the thrombosis of the superior sagittal sinus.

Of the 22 patients treated by us, 15 (68.18%) had been cured, 6 (27.27%) had improved, while 1 (4.54%) had died.

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