VERTEBRAL ARTERY DISSECTION – CLINICAL ASPECTS

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ABSTRACT

We report the cases of four patients under age of 55 years hospitalized in the Neurology Clinic of the Emergency University Hospital, Bucharest, for occipital headache, vertigo, nausea and vomiting, simptomatology installed after neck injury. Cerebral MRI revealed ischemic stroke in the posterior circulation due to an important dissection of a vertebral artery underlying the importance of this pathology as a cause of ischemic stroke in young patients.
Key words: dissection, vertebral artery, cerebral MRI

Arterial dissection is one of the many non-atherosclerotic vasculopathies that can cause brain ischemia and accounts 2% of all ischemic strokes and up to 25% of ischemic strokes in patients younger than 55 years. Dissection of an artery occurs when blood under pressure finds its way into the vessel wall along a line of cleavage which is usually near the endothelial surface. It either leads to a luminal narrowing and/or occlusion (subintimal dissection) or to the formation of a pseudoaneurysm with potential risk of bleeding (subadventitial dissection). Most cases of vertebral artery dissection are encountered in the age group on 25-55 years with a slight female preponderance (1).

CAUSES OF VERTEBRAL ARTERY DISSECTION

The most important causes of vertebral artery dissection are (2):

1. clear cut neck trauma (motor vehicle accidents, strangulation, fall followed by neck injury)
2. relatively trivial mobilization of the neck (hair washing, chiropractic manipulation)
3. spontaneously - without any precipitant factor

The last two scenarios probably reflect an underlying structural weakness of the arterial wall, inasmuch as dermal connective tissue abnormalities have been detected in up to one third of the cases.

Approximately 5% of spontaneous dissection can be attributed to inherit disorders of collagen structure among which, the most important is the Ehlers-Danlos syndrome type IV.

Others to be mentioned are Marfan's syndrome, autosomal dominant polycystic kidney disease, osteogenesis imperfecta type I and fibromuscular dysplasia.

PREDISPOSING FACTORS FOR VERTEBRAL ARTERY DISSECTION

It has been reported that 5% of patients have a family history of arterial dissection. Various predisposing factors have been mentioned in the literature such as (3):

1. arterial hypertension
2. migraine
3. recent infections
4. hyperhomocysteinemia

ANATOMY AND PATHOPHYSIOLOGY OF THE VERTEBRAL SYSTEM

Pathophysiological mechanisms underlying neural damage following an arterial dissection may be diverse. Stenosis or occlusion of the vertebral artery results in direct ischemia of the brainstem or spinal cord. It may result from an endoluminal thrombus or by compression of the true lumen due to the presence of blood in the vessel wall (4). Progressive thrombosis or artery to artery embolisation can cause distal ischemia and branch artery occlusion (4,5,6). The process can obstruct distal basilar artery flow, compress cranial nerves

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or cause a subarachnoid hemorrhage (5). Spinal cord infarction is possibly related to hypoperfusion and watershed infarction, embolisation or anterior spinal artery occlusion (6).

Anatomically the vertebral artery is divided into four arbitrary parts designated V1 to V4. Most of the traumatic dissections involve the atlanto-occipital segment. It is likely that increased mobility, poor anchoring into the neighboring tissue and increased mechanical torsion and stretch at C1-C2 region predispose to mechanical injury (5,7).

THE DIAGNOSE OF THE VERTEBRAL ARTERY DISSECTION (4,6,8,9,10)

The diagnose of the vertebral artery dissection can be established by both non-invasive and invasive means.

1. The ultrasound examination of the neck vessels can suggest the presence of dissection but the sensitivity to detect vertebral artery disease is low. However the duplex examination has been used to monitories the healing of dissections in order to guide the long-term therapy. The typical findings includes: increased arterial diameter, decreased pulsatility, cerebrovascular abnormal echoes and haemodynamic evidence of decreased or reversed flow (11).
2. The cerebral MRI is the preferred non-invasive technique for both diagnose and follow-up of the vertebral artery dissection.
3. The catheter angiography remains the gold standard for diagnose of the vertebral artery dissection, showing the intramural flap and haematoma.

TREATMENT OF THE VERTEBRAL ARTERY DISSECTION (3,4,6,8,11,13)

The actual treatment of the vertebral artery dissection involves:

1. Anticoagulation with heparin followed by oral warfarin therapy (INR target = 2-3) as soon as possible after the diagnose. The duration of the anticoagulant therapy can be decided by following-up the patient on ultrasound or MRI. Whenever the vascular stenosis persists after three months of therapy, a continuation of another three months is imposed. After six months, if the stenosis is still persisting the switch on antiplatelet agents is necessary.
2. The general stroke therapy with monitorising the vital functions in a well equipped stroke unit.
3. Endovascular stent reconstruction whenever the medical management failed.
4. Surgical treatment of the dissections consisting of an in-situ interposition graft or an extracranial-intracranial bypass. The last one is indicated only for patients with persisting symptoms refractory to maximal non-invasive management and where the endovascular therapy is not indicated.

PROGNOSIS

As reported in the literature, the maximal improvement of the simptomatology occurs the first three months followed an arterial dissection. Risk of spontaneous dissection approximates 2% in the first month and decreases to about 1%/year (14, 15). Dissection usually does not recur in the same vascular territory (3,15).

CASE PRESENTATION

All cases to be presented consists in a simptomatology of nuchal/head pain followed by symptoms suggestive of brainstem ischemia, predominantly in the distribution of postero-inferior cerebellar artery and therefore simulating a lateral medularry syndrome.

The first case is of 51-year-old women with no personal medical history, admitted in the Neurology Clinic for neck pain, vetigo, nausea and vomiting. The neurological exam revealed Wallenberg's syndrome. The cerebral MRI showed in diffuse weighted image sequence a hyperintense signal localized in the posterior part of the right medulla and pons; in the T1 weighted image the double lumen on the right vertebral artery level was visible with no flow on this level.

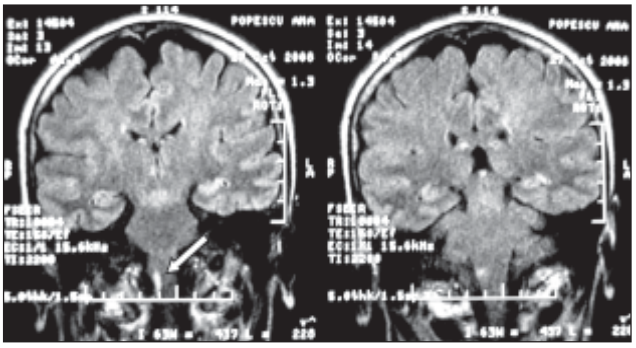

Figure 1. *Cerebral MRI – FLAIR image – hyperintense signal in the posterior part of the right medulla and pons*

The second case reports a 53-year-old male with no medical history, hospitalized for vertigo and equilibrium troubles. The neurological exam

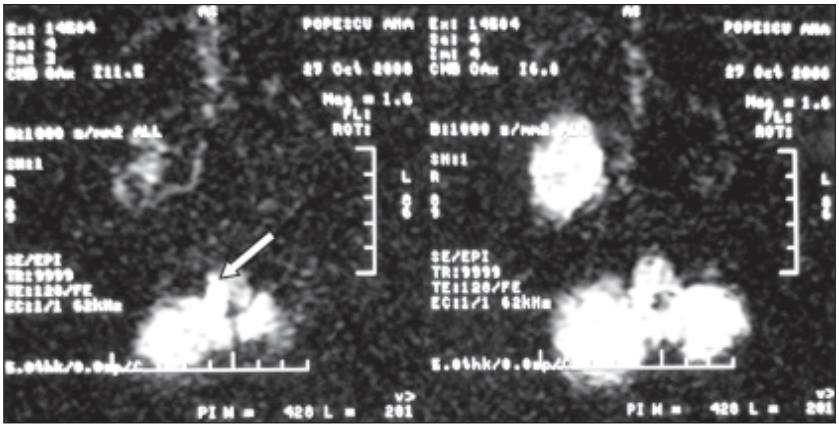

Figure 2. *Cerebral MRI – DWI sequence – hyperintense signal in the posterior part of the right medulla and pons*

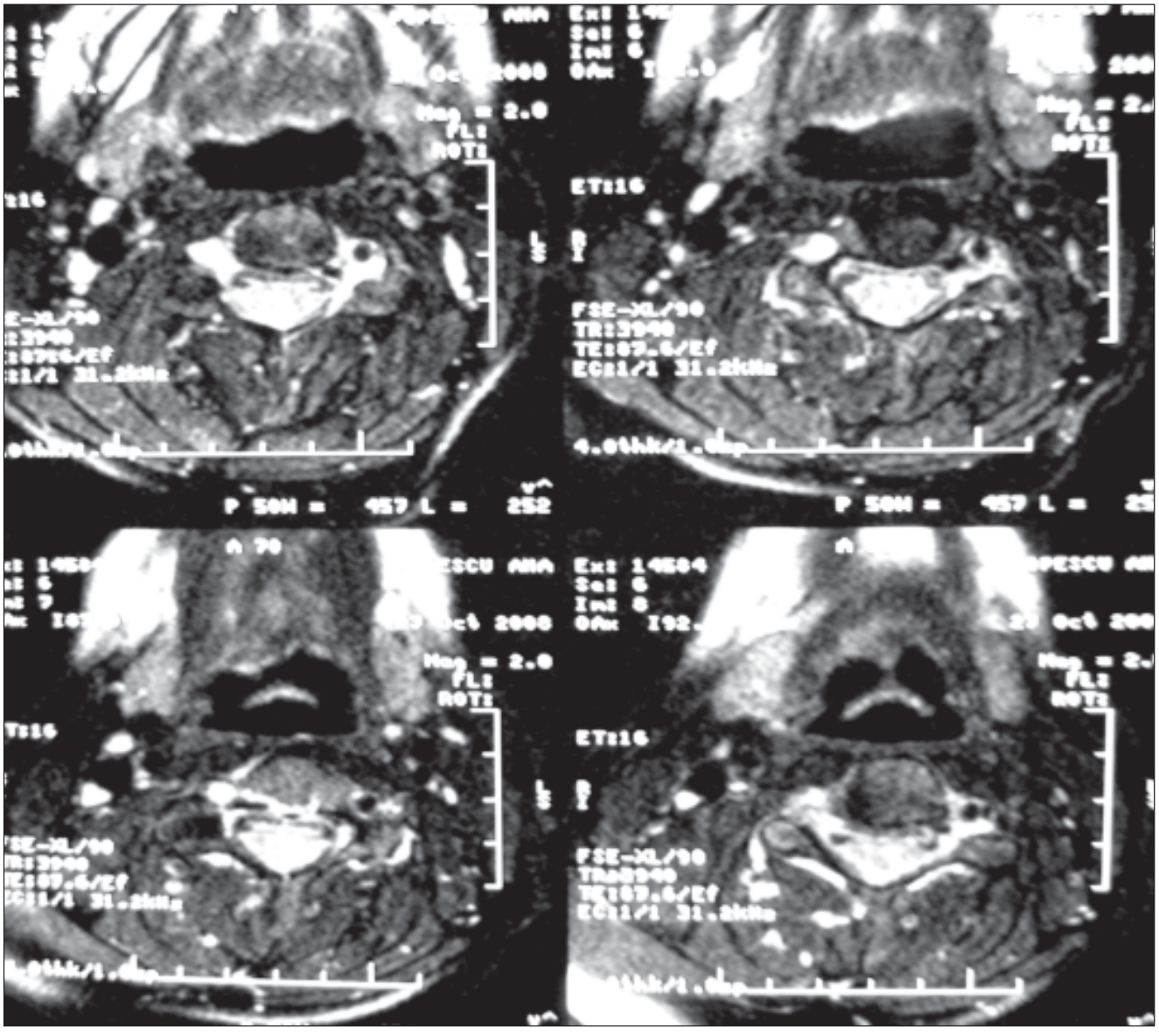

Figure 3.
Cerebral MRI - T1 weighted image - the double lumen on the right vertebral artery level with no flow on this level.

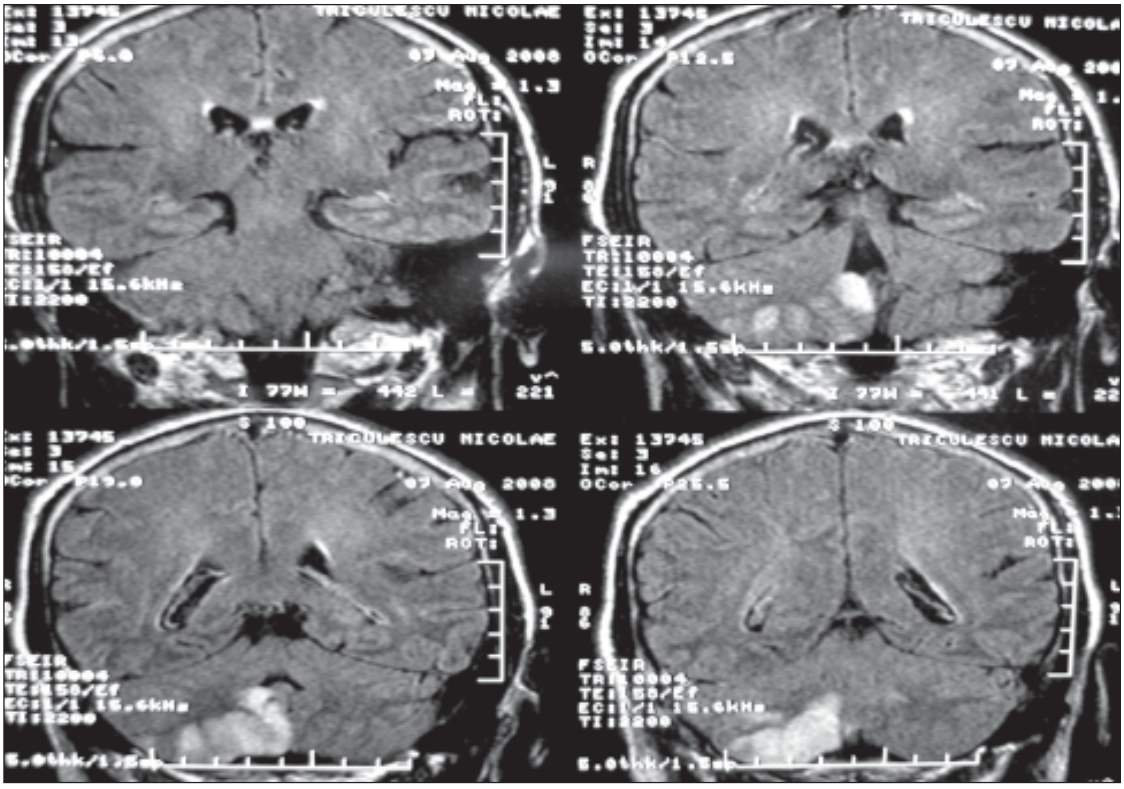

Figure 4. *Cerebral MRI – FLAIR image – hyperintense signal localitzed in the right cerebellum, in the territory vascularized by PICA*

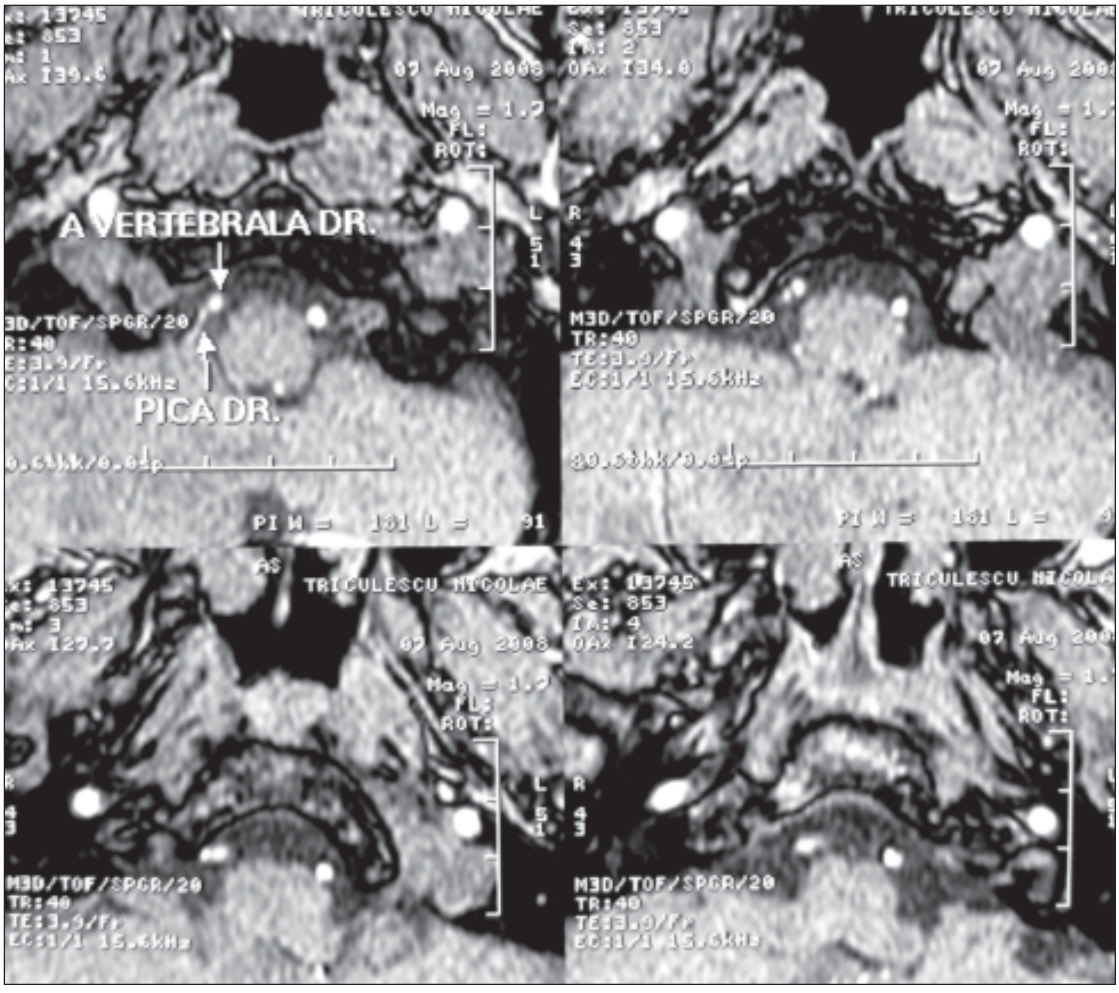

Figure 5. *Cerebral MRI - T1 weighted image – the intramural haematoma at the level of the right PICA*

revealed nothing more than astazo-abasia, no ataxia of the trunk or limbs was present. On the FLAIR sequence of the cerebral MRI a hyperintense signal localized in the right cerebellum, in the teritorry of vascularization of postero-inferior cerebellar artery was visible.

Another case is of a 47-year-old man, with no medical history, that after a prolonged cervical hyperextension position presents, suddenly, vertigo and equilibrium troubles. Cerebral MRI on T1 sequence shows bicerebellar hypointense signal and a double lumen at the level of the right vertebral artery.

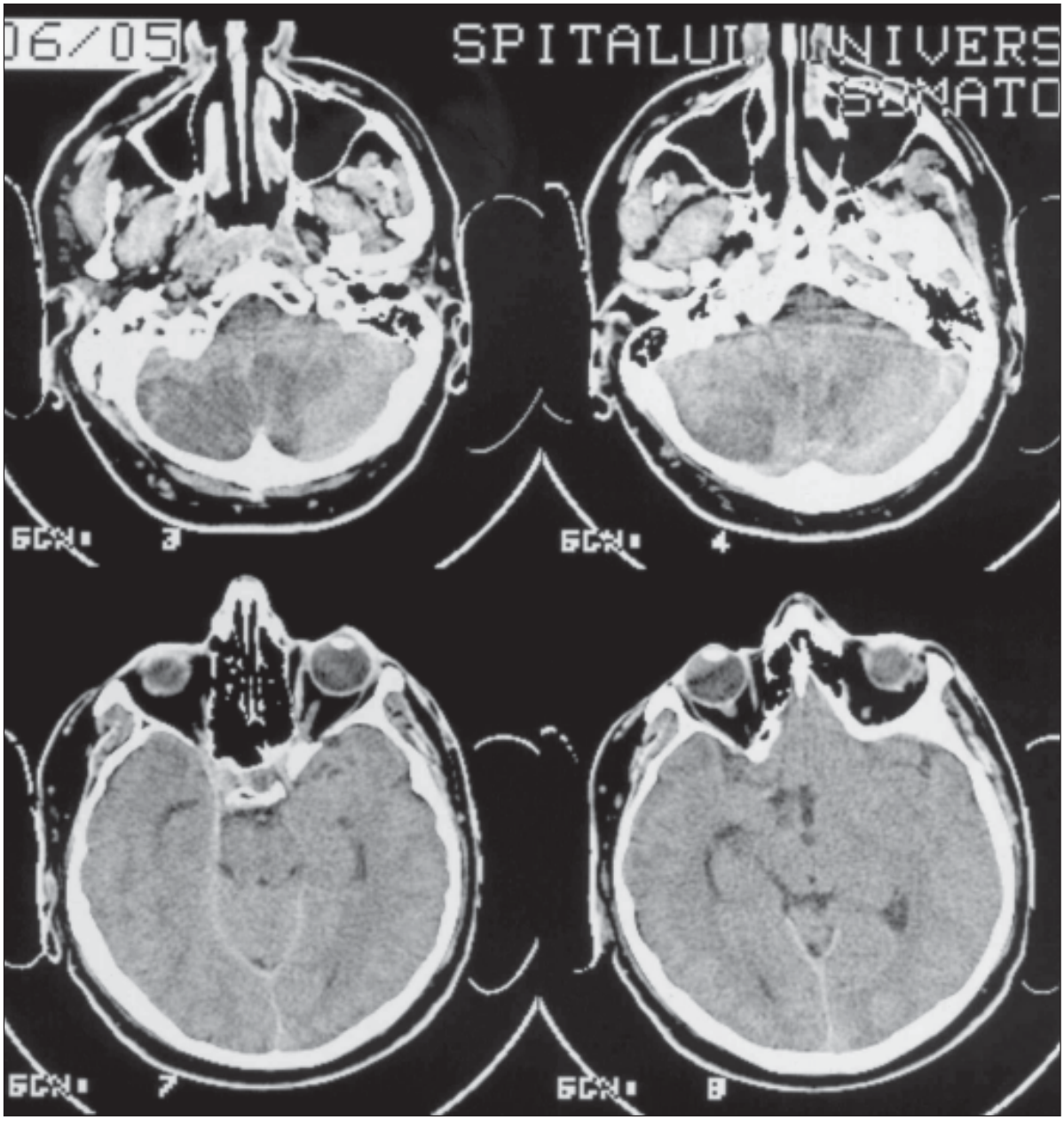

Figure 6. *Cerebral MRI - T1 weighted image – bicerebellar hypointense signal and a double lumen at the level of he right vertebral artery*

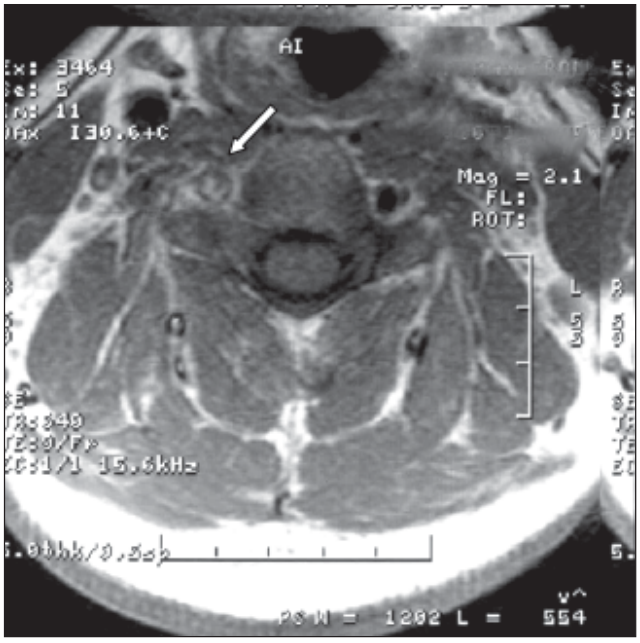

Figure 7. *Cerebral MRI - T1 weighted image – a double lumen at the level of the right vertebral artery*

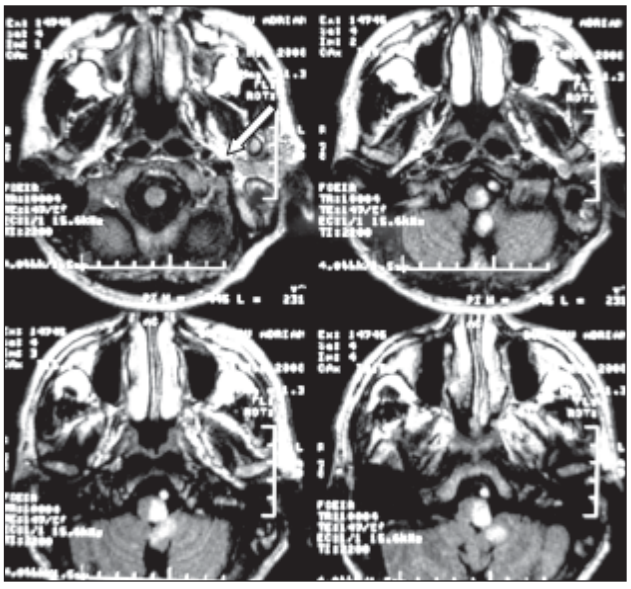

Figure 8. *Cerebral MRI – FLAIR image – hyperintensity localized in the left part of the medulla, the cerebellar vermis and the cerebellar amigdala; lack of the flow in the left vertebral artery (haematoma) and right vertebral artery hypoplasia*

The last case present a 39-year-old male with no personal medical history that after a fall installed vertigo, nausea and vomiting. The clinical exam revealed left Wallenberg syndrome. The cerebral MRI - T1 weighted image - showed hyperintensity localized in the left part of the medulla and the cerebellar vermis with lack of the flow in the left vertebral artery.

All of our patients received during the acute phase heparine treatment followed by administration of oral anticoagulants (acenocumarol) for another 3 to 6 months. Chronically, all patients received treatment with antiplatelet agents (aspirine).

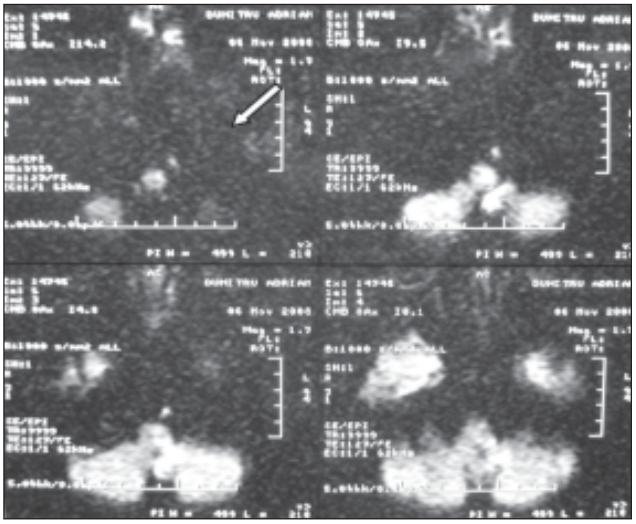

Figure 9. *Cerebral MRI – DWI – hyperintensity localized in the left part of the medulla, the cerebellar vermis and the cerebellar amigdala*

CONCLUSION

The purpose of the paper is to point out the importance of the vertebral artery dissection as a significant cause of ischemic stroke in young patients under the age of 55 years. The cerebral MRI and the MR angiography are the standard methods to apply for the diagnose of this pathology as well as for the follow up of this category of patients (class II level B of evidence) (3). The cervico-cerebral ultrasound exam is useful for noninvasive monitoring of vessel recanalization and for determining the duration of therapy (3).

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