The 21-year-old patient presented to the closest emergency department at 8 PM for nausea, numbness of the left limbs, incoordination of walking and dysarthria, with the symptomatology installed in a progressive mode 4 hours before presentation.

The nausea was the initial symptom, starting at 4 PM (without headache, nuchal pain, or vomit) and then, after 5.30 PM, the numbness of the left limbs and the incoordination of walking and by 7 PM dysarthria was added to the entire clinical constellation.

The patient did not have a medical history or a current medication.

He was a student.

He did not smoke and did not use recreational drugs.

He only drank alcohol occasionally.

There was no family history of thromboembolism, cardiovascular problems, or hematologic diseases.

He was first evaluated by a neurosurgeon in the emergency unit and had a brain CT scan that highlighted infracentimetric hypodensity in the thalamic nucleus, on the left side and at the level of the right cerebellar hemisphere in the PICA territory, and also a chest X-ray that revealed a widened projection area of the right side hilum, without any other pathological findings.

Following this evaluation, he was sent to the National Institute of Neurology and Cerebrovascular Diseases for consultation and special treatment.

During the ambulance transportation, he had repeated episodes of vomiting.

The initial physical examination (at 10 P.M) showed normal vital signs: blood pressure 130/80mmHg, heart rate 100 beats per minute and normal body temperature 36,8°C.

The cardiopulmonary exam was normal.

No carotid bruits were registered.

The neurological examination was notable for normal orientation.

The patient had equal, reactive pupils, divergent strabismus to the left eye, horizontal nystagmus, normal deglutition, mild paresis of the left arm, ataxia of all limbs, with an accent on the left extremities, numbness of the left limbs, bilateral response present at all deep tendons reflex, Babinski sign on the left side, stereotypical language.

The patient was able to repeat a few words and fragment of phrases and to perform simple commands of medium complexity.

The laboratory analysis highlighted a normal hemoleucogram, normal blood glucose level 9,11 mmol/ L (normal range 3,9-5,8mmol/ L), serum creatinine 0,69mmol/ L (range 0,5-0,9mmol/ L), total serum cholesterol 5,2mmol/ L (normal range < 5,2 mmol/ L).

The tests for thrombophilia were negative.

He also presented a normal sinus rhythm on the ECG.

The brain CT performed at the first emergency unit, as mentioned above, revealed a infracentimetric hypodensity at the level of the left thalamic nucleus and at the level of the right cerebellar hemisphere in the PICA territory.

Doppler ultrasound for cerebral and cervical blood vessels highlighted the marked frena in the flow velocities of the V2 segment of the right vertebral artery, an accentuated aspect in the high V2 segments with the complete disappearance of the flow in the V3 segment, without carotid atheromatous lesions or modifications of the trajectories or the arterial caliber.

Overall, there was a possibility of a dissection of the right vertebral artery in the higher segments with indication of cerebral and angio MRI.

Cerebral and angio MRI showed: areas with T2, FLAIR hypersignal, with an important restriction of diffusion and low ADC, situated in the right cerebellar area of the postero-inferior territory, anterior bilateral cerebellar territory, left cerebellar peduncle, both right > left thalamic nuclei, as well as subcortical occipital bilateral, with an ischemic aspect that was recently constituted in the vertebrobasilar territory.

T1 FS hypersignal was present on the entire intracranial segment of the right vertebral artery and the cranio-spinal junction.

On the angiographic arterial 3D TOF sequence, the presence of the rapid flow of the right vertebral artery could be viewed, up to the C2 segment, where there was a progressive pinch out with an aspect of flute beak shape.

Distal of this level, there was an absence of the rapid flow signal and also a lack of right PICA visualization.

The MRI aspect suggested a subacute dissection of the right vertebral artery.

The cerebral ventricular system had normal dimensions, shape, and topography.

The pericerebral fluid spaces and the basal cisterns were normal.

Conclusions: there were some ischemic lesions recently constituted in the vertebrobasilar territory and also a subacute dissection of the right vertebral artery.

Heparin administration was started immediately in the process of hospitalization because of the dissection.

The APTT value was kept in the range of 50-70 seconds.

Also, the prophylactic therapy for stress ulcer was performed.

After 7 days from the debut, the patient was transitioned to acenocumarol for long-term anticoagulation.

The treatment with acenocumarol was instituted with the heparin overlap until getting the INR therapeutic in the 2-3

intervals.

The evolution was favorable, the patient regaining the verbal fluency and the capacity for autonomy in movement. The ataxia was remitted at the right limbs and persisted at the left extremities, but registered a lot of improvement. The patient was discharged home with the recommendation to continue the treatment with Acenocumarol for 6 months with clinical, paraclinical and treatment reevaluation at 6 months.

The follow up was not possible because the patient did not return to the hospital after 6 months.