

Basilar artery thrombosis in a 20-year-old pregnant patient – difficulties in establishing the aetiology

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Dear Editor,

I would like to present a case of a 20-year-old pregnant patient with basilar artery thrombosis and our struggle to establish its aetiology in this particular patient.

Pregnancy constitutes a risk factor for both arterial and venous cerebral thrombosis. The reported incidence of ischaemic stroke in young adults is 10.8/100,000/year [1]. During pregnancy the incidence of ischaemic stroke is estimated at 12.2/100,000 pregnancies [2]. The most common causes of non-haemorrhagic stroke in pregnancy are cardioembolism, coagulopathy and preeclampsia/eclampsia. Nevertheless, establishing the aetiology of stroke in pregnancy can be challenging.

A 20-year-old pregnant patient in the 8th week of gestation was admitted to Department of Anaesthesiology and Intensive Care (ICU) of a tertiary medical centre following ischaemic stroke of the left cerebellar hemisphere. The patient was initially admitted to emergency department (ER) of a district general hospital with symptoms of headache, vertigo and impaired balance. These symptoms were present from the morning hours of the previous day, however the patient did not seek medical attention at that time. On admission to ER the patient was alert, non-responsive, meningeal signs were negative, pupils were equal, right 3rd cranial nerve paresis was present, vertical movements of eyeballs were observed, flexion of right limbs and extension of left limbs to pain was elicited and the Babinski sign was present bilaterally. Due to the risk of aspiration, the patient was

intubated and mechanical ventilation was commenced. Computed tomography (CT) revealed a 9 mm thrombus in the basilar artery and signs of left cerebellar hemisphere ischaemia. Head and neck angio-CT confirmed a thrombus in the basilar artery with no signs of vertebral artery dissection. Medical and drug history were negative. The patient underwent uncomplicated caesarean section 3 years earlier. Family history revealed very short height in the mother and grandmother. On admission to ICU the neurological status of the patient did not change compared to initial evaluation. Both neurological and interventional radiology consultations were requested, however the patient was disqualified from intravenous thrombolysis and percutaneous thrombectomy due to late presentation (over 24 hrs). Medical treatment of cerebral oedema was commenced (mannitol) and the patient was started on antiplatelet (acetylsalicylic acid 150 mg) and anticoagulant (dalteparin 200 IU kg⁻¹) medication. The ultrasound (US) examination revealed uterus corresponding to 10 weeks of gestation, gestational vesicle with a foetus corresponding to 9 weeks of gestation, normal foetus heart beat and normal decidual-chorionic reaction. Repeat head CT examination on day 4 confirmed ischaemia of left cerebellar hemisphere, upper part of pons, left cerebral peduncle, moderate oedema of cerebellum and brainstem, lower intensity of hyperdense signal in the basilar artery. On day 5 the patient regained consciousness, started opening eyes to voice, however was not following commands. On day 13 repeat

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head CT revealed reduced ischaemic area, reduced cerebellar oedema, and lower intensity of hyperdense signal in the basilar artery. On day 18, after receiving a consent from the court of law, dilatational percutaneous tracheostomy (Griggs method) was performed, respiratory support was gradually decreased and the weaning process was completed on day 21. On day 22 neurological rehabilitation consultation was requested and confirmed bulbar syndrome with dysphagia, immobile tongue, diminished palatine-pharyngeal reflexes, quadriplegia and suspicion of locked-in syndrome. Due to the risk of aspiration and need for prolonged enteral nutrition, percutaneous gastrostomy was inserted on day 32.

DIFFERENTIAL DIAGNOSIS, INVESTIGATIONS AND TREATMENT

Along with usual treatment and management of the patient, efforts were undertaken to find the cause of basilar artery thrombosis. The initial evaluation included an echocardiographic examination – no interatrial septum defect or thrombus were detected. US Doppler of lower extremities was also normal. After cardiac abnormalities and arterial dissection were ruled out, the next most likely cause of thrombosis was unspecified coagulopathy. The initial evaluation of this suspected coagulopathy included the standard laboratory coagulation panel (Table 1) and antiphospholipid syndrome panel (Table 2).

The results of standard laboratory coagulation tests were normal with the exception of mildly elevated D-dimers (1711 ng mL⁻¹). The results of the antiphospholipid syndrome panel were also negative. The last step in establishing the cause of thrombosis was determination of activity of protein C, free protein S, anti-thrombin, factor V Leiden and prothrombin gene 20210A mutation, homocysteine concentration, activity of factor VIII, IX, XI and plasminogen – the results are presented in Table 3. Determinations of these coagulation and anticoagulation factors were performed 4 weeks

after presentation in order to obtain the most objective results.

Activity levels of coagulation factor VIII and IX were moderately and mildly elevated, but activity increased to such a degree is normal during physiologic pregnancy. The diagnostic imaging and laboratory tests performed did not reveal the cause of ischaemic stroke in this particular patient.

The patient gradually started mobilizing upper and lower extremities, communication with the patient was maintained through nonverbal measures. In the 14th week of pregnancy the patient was transferred to Department of Neurological Rehabilitation. Haematology consultation was requested and as the final diagnostic step, histological examination of bone marrow and tests for Janus kinase 2 mutation and paroxysmal nocturnal haemoglobinuria were suggested.

The presented case report describes a basilar artery thrombosis in a young, apparently healthy, pregnant patient. There is increasing incidence of stroke in younger patients [3]. Although strokes in young adults are less frequent than in older patients,

TABLE 1. Standard laboratory coagulation panel

Parameter	Value	Reference range
Prothrombin time (s)	12.6	9.4–12.5
INR	1.11	0.80–1.20
Prothrombin activity (%)	85	80–120
Fibrinogen (mg dL ⁻¹)	314	200–393
aPTT (s)	28.1	25.4–36.9
D-dimers (ng mL ⁻¹)	1711	< 500
Platelets (G L ⁻¹)	290	130–400

aPTT – activated partial thromboplastin time, INR – international normalised ratio

when faced with acute neurologic symptoms in this age group, stroke should be considered in differential diagnosis. In our case the patient was not a candidate for thrombolysis/thrombectomy due to late presentation, what significantly impacted on neurologic recovery of the patient. There is high variability in aetiology of stroke in this population [4]. There is also a wide variety of uncommon causes of stroke in young patients. This variability is even higher in pregnant patients, as pregnancy is associated with several additional risk factors for stroke, particularly

TABLE 2. Antiphospholipid syndrome laboratory panel

Parameter	Value	Reference range
Lupus anticoagulant	0.76	0.80–1.20
β_2 -glycoprotein I IgM (RU mL ⁻¹)	6.32	< 20 negative result
β_2 -glycoprotein I IgG (RU mL ⁻¹)	< 2	< 20 negative result
Anti-cardiolipin antibody IgM (MPLU mL ⁻¹)	1.7	< 12
Anti-cardiolipin antibody IgG (GPLU mL ⁻¹)	2.9	< 12

IgG – immunoglobulin class G, IgM – immunoglobulin class M

TABLE 3. Additional laboratory panel

Parameter	Result	Reference range
Protein C activity (%)	> 149.9	70–140
Protein S free (%) ELISA	66	64–126
Anti-thrombin activity (%)	118	75–120
Factor V Leiden mutation	Negative	–
Prothrombin gene 20210A mutation	Negative	–
Homocysteine (μ mol L ⁻¹) CMIA	5.36	4.44–13.56
Factor VIII activity (%)	212.1	70–150
Factor IX activity (%)	130.3	70–120
Factor XI activity (%)	115.7	65–150
Plasminogen (%)	152	80–120

CMIA – chemiluminescent magnetic microparticle immunoassay, ELISA – enzyme-linked immunosorbent assay

preeclampsia/eclampsia, reversible cerebral vasoconstriction syndrome and cerebral venous sinus thrombosis [5]. Physiologic changes attributable to pregnancy might predispose to stroke, particularly dehydration, haemoconcentration or upregulation of clotting factors. Despite more accurate diagnostic tools becoming available, the most common type of stroke in young patients is a stroke of undetermined aetiology [6].

CONCLUSIONS

When faced with pregnant patients presenting with acute neurologic deterioration one should always consider stroke in a differential diagnosis. Prompt diagnosis and treatment is crucial for satisfactory neurological recovery. Establishing aetiology of stroke in this population is challenging and often remains undetermined due to numerous possible causes.

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