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Case Report

Cerebral Venous Thrombosis Due To Fasting: Case Report

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Abstract

Cerebral venous sinus thrombosis is a rare condition that sould be considered in indivudials with persistant and intractable headache. A 62 year-old male was admitted with a complaint of headache after 3 days of fasting. Neurological examination was normal except bilatetal Grade IV papiledama. Magnetic resonance venography revealed thrombosis of sagittal, transverse and sigmoid sinuses. In conclusion; dehydration due to fasting has an important factor especially polycytemic patients to develop cerebral venous thrombosis.

Keywords: Cerebral venous thrombosis, fasting

Oruç Sonrası Gelişen Serebral Venöz Sinüs Trombozu: Olgu Sunumu

Özet

Serebral venöz sinus trombozu, nadir rastlanan süregen ve tedaviye dirançli başağrılarında ayırıcı tanıda düşünülmesi gereken bir durumdur. 62 yaşında erkek hasta 3 günlük oruç sonrası başağrısı şikayeti ile başvurmuş, nörolojik muayenesinde bilateral Grade IV papilödem dışında bir muayene bulgusu saptanmamıştır. Hastanın yapılan Magnetik rezonans venografisinde sagital ve transvers sinüslerin tromboz ile tıkalı olduğu görülmüştür. Sonuç olarak; oruca bağlı dehidratasyon özellikle polisitemisi olan hastalarda serebral venöz tromboz gelişmesi açısından önem taşımaktadır.

Anahtar Kelimeler: Serebral venöz sinüs trombozu, oruç

INTRODUCTION

Cerebral venous thrombosis (CVT) is a rare and potentially fatal condition. Common etiologies include hypercoagulable diseases, dehydration, low flow states, adjacent infectious processes, malignancies, systemic diseases, oral contraceptives, hormonal replacement therapy, pregnancy and puerperium. The most common presenting symptoms include headache, seizure, nausea, focal neurological deficit and coma [(1)].

The following case demostrates a severe venous sinus thrombosis due to dehydration.

CASE PRESENTATION

We describe a patient with marantic cerebral venous thrombosis. This's a rare condition caused by increased blood viscosity secondary to malnutrition and dehydration. A 62 years-old male patient admitted to the hospital with headache after 3 days of fasting.The headache started from the neck, expanded through the occipital region and partially diminished with non-steroid antiinflammatuary agents. Within 10 days the headache worsened, nausea and vomitting accompanied. His physical examination was normal. Bilateral Grade 4 papiledama was the only finding in neurological examination. Laboratory findings were as follows; Hb:17.7 g/dl, Hct:52 %, MCV:81.8 fL, WBC:9600/ ìl , PLT:190000/iL, LDH:642 u/L, with normal biochemical values. Cranial Computerized Tomography didn't show any pathology. Cranial Magnetic Resonance Venograpy (MRV) showed blood flow loss due to thrombus in transvers, sigmoid and sagittal sinuses. Thrombus extended through straight sinus. There was no flow except deep venous

sinuses (see Figure 1). We applied lomber punction (LP) to monitor intracranial pressure. Cerebrospinal Fluid (CSF) was acellular with opening pressure 450 mmH2O, closing pressure 165 mmH2O, CSF protein was 39 mg/dl, CSF glucose was 67 mg/dl while blood glucose was 103 mg/dl at the meantime. ANA and anti-DNA results were negative. ACA IgM, ACA IgG, CRP, fibrinogen, homocystein, protein C, protein S values, thyroid function and lipid profile tests were in normal ranges. Anti-Hbs antibodies were pozitif while other serologic markers were negative. Factor V Leiden mutation, Prothrombin II mutation, Metyl Tetra Hydro Folat Reductase (MTHPR) mutation were negative and there were no Bcr-Abl gene products. The patient was diagnosed as marantic CVT. Despite hydration and 5 units of phlebothomia Hb remained 17.7g/dl. He was consultated by haemotology and blood marrow aspiration biopsy was applied. The biopsy result was normocellular and showed no clues of chronical myeloproliferative disease. Multipl LPs were done to reduce intracranial pressure. Heparin infusion was started to provide recanalisation. Acetazolamide was added to decrease intracranial pressure. After 14th day of heparin infusion, no recanalisation was detected. Infusion was stopped and oral warfarin treatment was started, planning to keep INR between 2.0-3.0. The last LP showed 170 mmH2O opening pressure. The patient was externated with Grade I papilledema.

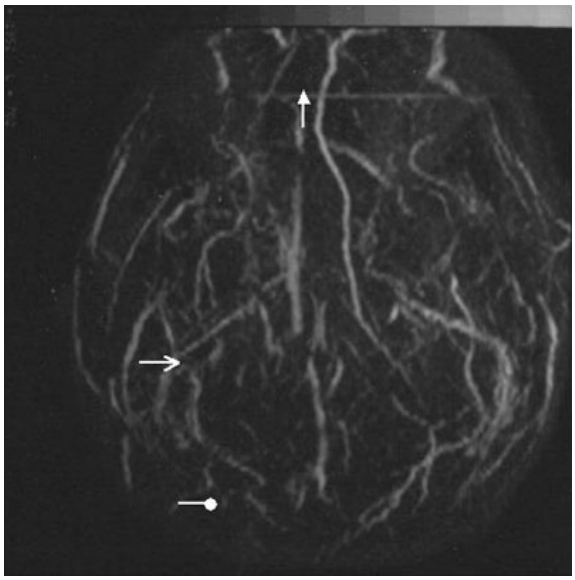

Figure 1: *Axial MPI of cranial MR venography shows no flow in all venous sinuses (superior sagital, transvers and sigmoid sinuses) consisted with venous sinus thrombosis. The original locations of the sinuses have been shown by different arrows. Arrow: Superior sagital sinus; Open arrow: Transvers sinus; Oval arrow:Sigmoid sinus*

Table 1: Conditions that increase blood viscosity.

1) Dehydration
2) Polycythaemia vera
3) Thrombocytosis
4) Antithrombin III deficiency
5) Protein C or Protein S deficiency
6) Pregnancy
7) Puerperium

DISCUSSION

Cerebral venous thrombosis CVT is a rare cause of stroke [2-4]. The estimated annual incidence is 3 to 4 cases per million population and up to 7 cases per million among children [5]. Many etiological factors can be responsible for this clinical situation [6]. It can occur in conditions that increase blood viscosity (See Table 1)[7-12]. Polycythaemia and dehydration are among these conditions and may cause marantic CVT.

Cerebral venous thrombosis has various clinical and radiological manifestations [13]. It can be diagnosed in its early phase by MR and MR angiography [2,3,8,14-15]. Time-of-flight (TOF) MRV is often used to examine the intracranial dural sinuses [3].

Treatment options are systemically delivered anticoagulation therapy , chemical (tPA) or mechanical thrombolysis [16]. Targeted endovascular delivery of thrombolytic agents may be a choice, but it carries an increased risk of hemorrhage [16]. When, neurological deterioration occurs in sigmoideus and/or transversus dural sinus thrombosis with unilateral edema, despite adequate anticoagulation therapy, a decompressive craniectomy should be considered especially in young patients[4].

In our patient the only etiological factor was dehydration after 3 days of fasting. The patient was already polycythaemic and fasting probably increased blood viscosity. In Moslem countries fasting is a kind of religious exercise. Individuals and doctors should be aware about dehydration, electrolyte imbalance while fasting. Especially polycythaemic individulas are at risk and should be warned by doctors.

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