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Case Report

Cerebral venous sinus thrombosis in a 30-year-old woman on medroxyprogesterone acetate: a case report

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ABSTRACT

About 80% of patients diagnosed with cerebral venous sinus thrombosis (CVST) exhibit a prothrombotic risk factor or an identifiable direct cause. The use of combined oral contraceptives (COC) is well recognized as a significant risk factor among women. Furthermore, it has been demonstrated that progestins, whether administered alone or in conjunction with ethinyl estradiol, are also linked to an increased risk of venous thrombosis. This study presents the first documented case in Ivory Coast of cerebral venous thrombosis in a young woman without any other identifiable prothrombotic risk factors, who was receiving depot medroxyprogesterone acetate (DMPA), a long-acting injectable progestin contraceptive. The patient, a 30-year-old right-handed female, was utilizing a progestin-based contraceptive method (medroxyprogesterone acetate, PROVERA DEPO) administered quarterly. At the time of her admission to the neurology department, she was receiving her second injection and presented with recent, atypical headaches that escalated rapidly. The neurological examination upon admission revealed signs of intracranial hypertension and a spastic pyramidal syndrome affecting the left side. Neuroimaging studies, including CT and MRI, identified cerebral venous thromboses in the right sinus and the superior sagittal sinus. Further investigations did not reveal any abnormalities associated with thrombophilia, autoimmune disorders, neoplasms, or infections. The patient received anticoagulant therapy at an effective dosage for a duration of six months, resulting in a reduction of headaches and the resolution of motor deficits. Any neurological event in a patient receiving Medroxyprogesterone Acetate, necessitates a neuroimaging evaluation to rule out the presence of CVST.

Keywords: Cerebral venous sinus thrombosis, Progestative contraception, Depot medroxyprogesterone acetate, Case report, Ivory coast

INTRODUCTION

Cerebral venous sinus thrombosis (CVST) is a distinct form of stroke that involves the development of a thrombus in the cerebral venous sinuses or veins. Once considered rare, this condition is now more frequently identified, largely due to improvements in neuroimaging, especially magnetic resonance imaging.[1,2] In sub-Saharan Africa, the prevalence of CVST in hospital settings is reported to be between 0.47% and 3%.[3] This condition primarily impacts younger populations, particularly individuals under 50 years of age, and is observed to be three times more common in females than in males.[2] Approximately 80% of patients diagnosed with CVST exhibit a prothrombotic

risk factor or an identifiable direct cause.[4] However, in 20 to 25% of cases, no cause can be determined despite extensive investigations. The prothrombotic risk factors associated with CVST include various thrombophilic abnormalities, which may be either genetic or acquired.[5-7] Specific risk factors for women include pregnancy, the postpartum period, phytoestrogens, and the use of hormonal treatments, including contraceptives, all of which contribute to an increased risk.[4,7,8]

The use of combined oral contraceptives among women is identified as a significant risk factor, with estimates indicating that the risk may be two to six times higher compared to women who do not use these contraceptives. Initially, it was believed that the estrogen component in contraceptive pills was responsible for the increased risk of thrombus formation.[9] However, research has shown that progestins, whether administered alone or in conjunction with ethinyl estradiol, are also linked to a heightened risk of venous thrombosis.[9-11] The highest risk is associated with third-generation progestins, such as desogestrel, as well as newer progestins like cyproterone acetate and drospirenone.[5,6]

The depot medroxyprogesterone acetate (DMPA) is a long-acting injectable progestin contraceptive. In 1998, the World Health Organization reported a slight increase in the risk of venous thrombosis among women using injectable contraceptives composed solely of progestins (OR), 2.2; 95% (CI), 0.7 to 7.3).[11] Conversely, Goldstein et al, found that this injectable progestin contraceptive did not exhibit adverse effects on markers of thrombosis risk.[12] The most recent large-scale clinical study, which included 446 patients and 1,146 controls, established a 3.6-fold increased risk (95% CI) of deep vein thrombosis associated with injectable contraceptives containing medroxyprogesterone acetate.[9] However, despite these discussions and controversies, there remains a lack of large-scale studies to further elucidate this risk. Nonetheless, there are some global case reports, of cerebral and/or systemic venous thrombosis occurring in women using progestin contraceptives.[4,13-15] This observation marks the first reported case in Ivory Coast of cerebral venous thrombosis in a young woman using DMPA without any other thromboembolic risk factors.

CASE REPORT

A 30-year-old female patient, who is a midwife and immunocompetent, presents with a medical history of four pregnancies, three of which were voluntarily terminated, the most recent occurring over a year ago. She underwent a cesarean section in 2021. The patient was placed on progestin contraception (medroxyprogesterone acetate, DMPA, PROVERA DEPO), administered every 12 weeks, and she was receiving her second injection at the time of admission. There is no history of neurological events, and she was admitted to our neurology department due to recent and unusual headaches that developed rapidly over a span of five days. The symptoms commenced approximately seven days prior to her admission, characterized by unilateral headaches on the right side, localized in the temporal region. These headaches were described as pulsating, radiating towards the orbit and forehead on the same side, with an increasing intensity reaching around 8 out of 10.

The patient also experienced paroxysmal episodes accompanied by symptoms such as sneezing, tearing, and photophobia, which were exacerbated by any movement. Notably, there were no instances of projectile vomiting or fever. This clinical presentation prompted a consultation at a peripheral hospital, where an infectious workup was conducted. Initially, the patient was treated for uncomplicated malaria; however, the lack of improvement in her headaches necessitated her transfer to our neurology department for more comprehensive evaluation and management.

Upon admission, the neurological examination revealed signs of intracranial hypertension and left-sided hemiparesis rated at 4/5 on the Medical Research Council (MRC) muscle strength scale, accompanied by diffuse hyperreflexia on the same side. The patient exhibited difficulty in ambulation, dragging the left foot. No evidence of meningeal syndrome, cranial nerve involvement, sensory disturbances, or coordination issues was identified, nor were there any signs of an infectious syndrome. Bilateral papilledema was noted during the ophthalmological assessment.

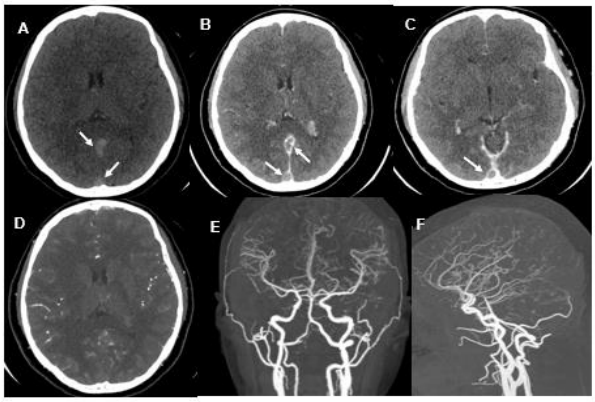

Figure 1: Brain computed tomography angiography (CTA) conducted upon the patient's admission, approximately seven days following the onset of headaches. In the axial section without contrast agent (A), a spontaneous hyper density is identified within the right sinus and the superior longitudinal sinus, indicative of the "dense triangle" sign, which suggests the presence of a thrombus. Following the administration of the contrast agent (B and C), enhancement of the sinus wall is noted, outlining a central hypo density in the right sinus and the superior sagittal sinus, corresponding to the "Empty delta" sign. No abnormalities are detected during the arterial examination (D, E, and F).

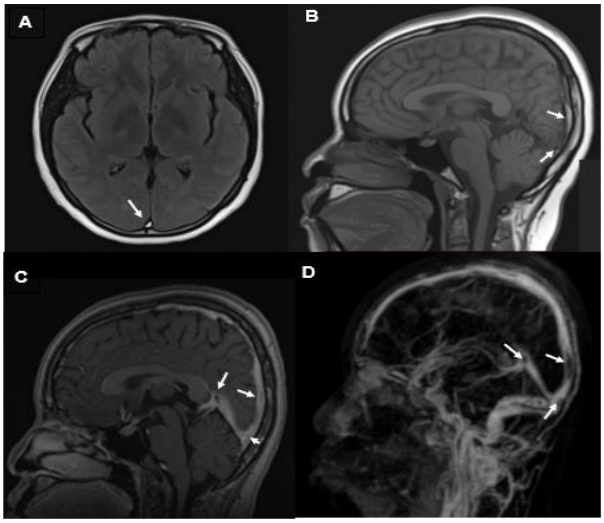

Figure 2: Brain magnetic resonance angiography (MRA) carried out three weeks after symptom onset. (A, B, C, D) The thrombus is observable as a hyper intense signal in the T2 FLAIR spin-echo (SE) sequence and in T1 SE without gadolinium at the location of the superior sagittal sinus. Conversely, it exhibits a hypo intense signal in T1 SE with gadolinium and during the magnetic resonance venography (MRV) at the superior sagittal sinus, the right sinus, and the Torcular Herophili.

The Brain CT-Angiography performed at the time of admission showed extensive cerebral edema, accompanied by indirect signs that were suggestive of cerebral venous thrombosis. This diagnosis was later confirmed through magnetic resonance imaging and venography. An etiological workup was conducted, which included a lumbar puncture and tests for thrombophilias and autoimmune diseases, all of which were negative.

An established treatment protocol involved the administration of low molecular weight heparin at a dose of 8000 IU, given twice daily for five days. This was followed by a regimen of Rivaroxaban, starting at 15 mg twice daily for three weeks, and then adjusted to 20 mg once daily for five months. In addition, analgesics were prescribed to alleviate headaches, and Mannitol was administered to control cerebral edema. The patient's clinical evolution was marked by a significant improvement in headache symptoms and a reduction in motor deficits within one week. After two weeks, she was discharged from the hospital and continues to receive follow-up care in an outpatient setting. She reported no additional complaints and was advised against renewing her prescription for medroxyprogesterone acetate, opting instead to pursue natural contraceptive methods with her partner.

DISCUSSION

CVST represents 0.5% of all strokes, with an annual incidence of three to four cases per million in the general population, and as high as seven cases per million among younger individuals, particularly affecting young adult women more than their male counterparts.[17] The recent development of non-invasive diagnostic methods, notably magnetic resonance imaging (MRI) has greatly improved the diagnosis of CVST.[17,18] In sub-Saharan Africa, the prevalence of CVST in hospitals ranges from 0.47% to 3%.[19-21] This condition significantly contributes to disability, resulting in dependence for approximately 5% to 10% of patients, with an acute mortality rate of about 10% in this region.[3,11]

The risk factors associated with prothrombotic conditions leading to cerebral venous thrombosis encompass hereditary disorders such as ant thrombin deficiency, deficiencies in proteins C and S, the factor V Leiden mutation, and Hyperhomocysteinemia. Additionally, acquired prothrombotic states include nephrotic syndrome, antiphospholipid syndrome, neoplasms, traumatic brain injuries, infections of the central nervous system and adjacent structures, hematological disorders, cirrhosis, systemic vasculitis, and certain medications such as steroids, methotrexate, and cisplatin.[4,22,23]

Pregnancy, the postpartum period, and the use of contraceptives are significant risk factors for women. In the analysis conducted by Yanina et al, on 287 cases of cerebral venous thrombosis in sub-Saharan Africa, it was found that infection was the most prevalent risk factor, accounting for 63.1%. This was followed by oral contraceptives, which represented 7.3%, while pregnancy or the postpartum period was associated with 6.2%, and genetic abnormalities related to thrombophilias accounted for 2.2%.[3]

The association between the use of combined contraceptives by women and the risk of thromboembolic disease has been substantiated by numerous studies.[11,24] According to the research conducted by Lidegaard et al, the absolute risk of venous thrombosis is 3.01 per 10,000 woman-years among non-users of oral contraceptives, whereas it increases to 6.29 for users. This risk is influenced by factors such as the estrogen dosage, the type of progestin, and the duration of contraceptive use.[11]

There are ongoing debates regarding the risk of venous thrombosis associated with contraceptives that contain only progestogens. The available data lacks sufficient statistical power to identify minor differences, primarily due to the relatively small number of users of these contraceptive method.[11] The first study to investigate the relationship between progesterone-based injectable contraceptives and cardiovascular diseases, including strokes, cerebral venous thrombosis, and myocardial infarction, was conducted by the World Health Organization (WHO) through the "World Health organization collaborative study of cardiovascular disease and steroid hormone contraception" expert group.

This study found that the adjusted odds ratio for all combined cardiovascular diseases, when compared to non-users of steroid hormonal contraceptives, was 1.02 (0.68 – 1.54) for oral progestogen contraceptives and 0.95 (0.49 – 1.86) for injectable contraceptives. Conversely, the risk of cerebral venous thrombosis among users of injectable progestogens was higher in developing countries, with an odds ratio of 1.23 (0.58–2.61), compared to 0.90 (0.09–9.46) in European countries.[11]

The case-control study conducted by Astrid and her colleagues focused on a cohort of premenopausal women aged 18 to 50, comprising 446 cases and 1,146 controls. The findings indicated that the use of injectable contraceptives containing medroxyprogesterone acetate was associated with a 3.6-fold increased risk of venous thrombosis (95% confidence interval, 1.8 to 7.1) when compared to women not utilizing hormonal contraceptives. Additionally, case reports from regions outside Africa have suggested a potential link between the use of progestin-only contraceptives and cerebral venous thrombosis, in the absence of other prothrombotic abnormalities, although some studies have reported contradictory findings.[4,12-15,24]

The diagnosis of cerebral venous thrombosis (CVST) poses significant challenges due to its heterogeneous clinical presentation and the lack of specific clinical signs. Symptoms associated with CVST can vary widely, ranging from mild manifestations to potentially life-threatening conditions.[25] This variability is influenced by several factors, including the location and extent of the thrombosis, the degree of venous occlusion, the patient's age, and the underlying disease or risk factors present.[7] Headaches are the most commonly reported symptom in cases of CVST, occurring in approximately 90% of instances, and they may be the sole manifestation in about 25% of patients.[25]

Headaches related to cerebral venous thrombosis do not exhibit distinct diagnostic characteristics, although they are typically progressive, developing over several hours or days. In the case of our patient, the headache was the initial symptom observed, presenting in a subacute manner over a five-day period. Approximately 30% of CVST cases present acutely, with symptoms emerging within 48 hours. In up to 50% of cases, symptoms develop subacutely, appearing between 48 hours and 30 days.

Chronic forms account for 20% of cases, with symptoms evolving over a period exceeding 30 days and potentially lasting up to six months.[26] Up to 40% of patients with cerebral venous thrombosis (CVST) may exhibit focal neurological symptoms that resemble those of a stroke. However, these manifestations are often less sudden than those associated with arterial ischemic strokes or intracranial hemorrhages. Motor symptoms are the most commonly observed, followed by visual impairments and speech disturbances, particularly when the left transverse sinus and Labbé vein are involved. Sensory symptoms, on the other hand, are less frequently encountered.[25] In the case of our patient, a mild motor deficit was noted on the seventh day of symptom progression, with a muscle strength assessment of 4/5 on the medical research council (MRC) scale. Due to the non-specific nature of the clinical signs, it is crucial to perform neuroimaging in any patient presenting with symptoms suggestive of CVST, such as a recent and persistent headache, exacerbation during the Valsalva maneuver, lack of improvement with standard analgesia, or in individuals with typical risk factors for CVST or papilledema.[7,25,26]

Currently, no laboratory test can definitively rule out cerebral venous thrombosis.[28] The guidelines established by the European Stroke Organisation (ESO) and the American Heart Association (AHA) advocate for the use of magnetic resonance venography or computed tomography venography as confirmatory diagnostic methods.[27,28] Non-contrast cranial computed tomography (CT) serves as an initial examination that proves beneficial in approximately one-third of patients. It can reveal specific signs, such as hyperdensity in the venous sinus or deep veins, known as the dense triangle sign in cases of sagittal sinus thrombosis or deep cerebral vein thrombosis, or the cord sign, which indicates a thrombus in the transverse sinus.

However, it is crucial to note that up to 30% of patients may present with a normal CT scan, and even when abnormalities are detected, they are not specific. Therefore, all patients suspected of having cerebral venous thrombosis should undergo further imaging studies beyond standard CT.[25,28] MRI is recognized as the most sensitive technique for detecting a thrombus, employing sequences that exploit the magnetic susceptibility effects of paramagnetic blood components containing iron, such as T2 weighted gradient echo or susceptibility-weighted imaging (SWI).

The signal characteristics of the clot across various MRI sequences differ according to its age, which can aid in determining the timing of the thrombus. In the case of our patient, during the subacute phase, the thrombosis is characterized by hyper intensity on T1, FLAIR, and diffusion-weighted imaging, alongside hypo intensity on gradient echo sequences. Furthermore, MRI is the preferred method for a comprehensive assessment of parenchymal consequences, including ischemia, hemorrhage, and edema.[7,25]

The therapeutic strategy for cerebral venous thrombosis is based on the effective use of anticoagulants, along with the treatment of the identified causative factors or the reduction of relevant risk factors. In our instance, we began anticoagulant treatment through the parenteral route, administering low molecular weight heparin for three days, followed by a switch to Rivaroxaban at a dose of 15 mg twice daily, and subsequently 20 mg once daily for five months, in line with the American guidelines.[28] The results from the SECRET study demonstrate that the immediate

initiation of oral anticoagulant therapy, such as Rivaroxaban, can result in outcomes that are comparable.[29]

The annual risk of recurrent cerebral venous thrombosis (CVST) is estimated to be between 2% and 7%, while the risk of other venous thromboses ranges from 4% to 7% per year. Consequently, long-term anticoagulation is typically advised. However, the optimal duration of anticoagulation for CVST remains uncertain due to the lack of randomized trials or prospective studies. In practice, this duration is determined based on the individual's risk factors for recurrence and bleeding.

Patients who have experienced a CVST episode linked to transient risk factors, such as the use of contraceptives, are recommended to undergo anticoagulation for a period of 3 to 6 months.[25] An ongoing study aims to compare the efficacy and safety of short-term anticoagulation (3 to 6 months) versus long-term anticoagulation (12 months) following a CVST, which may provide insights into the appropriate treatment duration.

In rare instances, endovascular treatment may be considered for severe cases of CVST that do not improve or worsen despite anticoagulant therapy, although this option may not be feasible in resource-limited settings. In addition to the specific treatment, it is essential to incorporate symptomatic management, particularly for headaches, through the use of analgesics, as well as addressing intracranial hypertension via osmotic therapies such as mannitol.[25]

Cerebral venous thrombosis (CVST) is typically linked to a positive prognosis, with around 75% of patients experiencing complete functional recovery. Nonetheless, approximately 15% of those affected may either die or become dependent. In the instance of our patient, she recovered from her hemiparesis after two weeks of treatment, showing no motor deficits. In the acute phase, factors that may contribute to a poor prognosis include male gender, older age, confusion or coma, intracranial bleeding, deep vein involvement, infections, and malignancies.

Importantly, our patient did not present any of these risk factors. Furthermore, individuals with severe thrombophilic disorders and those who discontinue anticoagulant treatment prematurely are deemed to be at higher risk. However, our patient did not have any thrombophilic issues and adhered to her anticoagulant therapy for six months, achieving recovery without motor or cognitive impairments. Despite this, she suffers from anxiety and remains apprehensive about the possibility of recurrence, even after stopping the use of progestin contraceptives, exhibiting a significant fear of hormonal contraception.

CONCLUSION

The onset of any neurological symptoms, especially recent and atypical headaches that exhibit rapid deterioration and do not respond to conventional pain relief, or the presence of focal signs in a patient on Medroxyprogesterone Acetate, warrants a neuroimaging assessment to exclude the possibility of cerebral venous thrombosis. It is also imperative to conduct thorough investigations to uncover additional thromboembolic risk factors. Anticoagulation treatment should be commenced promptly and continued for at least three months. A collaborative approach involving a vascular neurologist, hematologist, and gynecologist is critical to identify the most appropriate contraceptive strategy for this group of patients.

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