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Stroke Secondary to an Intra-Carotid Thrombus in a Context of Antiphospholipid Syndrome and Thrombocytosis: Report of a Case

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Abstract

Common carotid artery thrombosis (CCT) represents 7% of supraaortic artery thrombosis (TSA). CCT is responsible for neurological symptoms in 92% of cases. Antiphospholipe antibody syndrome (APS) is a rare cause, and its association with thrombocytosis is exceptional. We report a case of stenosing TBI secondary to APS associated with thrombocytosis discovered during a stroke. Ultrasound was the means of diagnosis and monitoring of CCT. The diagnosis of APS was biological. The cause of our patient's thrombocytosis remains unknown. The treatment was only medical followed by a good clinical evolution. Anticoagulation has been used for treatment in the acute phase but also for the prevention of stroke recurrence.

Keywords

Thrombus, Anti-Phospholipids, Thrombocytosis

1. Introduction

Common carotid artery thrombosis (CCT) represents 7% of supraaortic artery thrombosis (TSA) (Bhatti et al., 2007). Thrombus migration is associated with a poor prognosis (Koge et al., 2020). TBI is a rare cause of cerebral ischemia often associated with atheromatous plaque (Ukkola-Pons et al., 2012). TBI is responsible for neurological symptoms in 92% of cases (Kazantsev et al., 2023). In 17.1% cases, an ischemic recurrence is recorded within 30 days following the manifestation (Cancer-Perez et al., 2021). Antiphospholipid syndrome (APS)

and thrombocytosis are causes of hypercoagulability, their combination potentially increases the risk of vascular events (Kim et al., 2019; Tufano et al., 2010). Cerebrovascular accidents (CVA) are the second most common clinical manifestation of APS after venous thrombosis, ischemic stroke is the symptom in 13% of cases (Cervera et al., 2002; Sanna, 2003). Reactive thrombocytosis can also lead to carotid thrombosis and ischemia secondary brain injury, particularly in young African-American women (Nelson et al., 2022). There is no therapeutic protocol for CCT to date, although medical and surgical treatment have been effective, no clear superiority has yet been established (Rothwell, 2006). Persistent antiphospholipid antibodies in a patient with a history of thrombosis lead to an increased risk of recurrence, approximately 50% over 5 years (Basili & Violi, 2008). We report an exceptional case presentation of stroke secondary to head trauma in a setting of APS and thrombocytosis.

2. Observation

This was a 58-year-old patient with a medical history of non-insulin-dependent diabetes on metformin 850 mg, overweight (BMI 33) and thrombocytosis. He was admitted for a sudden onset of right brachial hemiparesis with NIHSS 2, regressing in 30 min.

Cerebral angio-CT reveals a small left parietal hypodensity suggesting ischemia of the left superficial sylvian artery, angio-TSA unstable atheromatous plaque, without calcification, sub-occlusive of the left common carotid.

Doppler ultrasound of ASD (**Figure 1**): D1 pedunculated thrombus on atherosclerotic plaque at the distal part of the left internal carotid, an anatomical stenosis of approximately 70%, and a hemodynamic impact of 50% to 60% European Carotid Surgical Trial (ECST) and less of 50% North American Symptomatic Carotid Endarterectomy Trial (NASCET) (Vitesse Systolique Maximale (VSM): 155 cms; Vitesse Tele Diastolique (VTD): 40 cms). Cardiac ultrasound revealed no pericardial effusion, no valvular prolapse or stenosis, regular ejection fractions… and all relevant negative cardiac abnormalities that ruled out cardiac dysfunction. Abdominopelvic ultrasound: steatotic liver, discreetly increased in size without obvious lesion.

The blood test on admission shows: thrombocytosis: 457 × 10.9/l after control on D2 330 × 10.9/l, lymphocyte phenotyping without particularity, V617F mutation of the JAK2 gene negative, positive circulating lupus anticoagulant was persistent even after twelve (12) weeks, Anticardiolipin antibodies and anti-Beta-2-glucoprotein 1 antibodies were negative.

The treatment was as follows: Aspirin 250 mg and Clopidogrel 150 mg as a bolus then respectively 100 mg and 75 mg per day for one month. LMWH 6000 IU every 12 hours after 5 day relay Coumadin 5 mg per day with target INR 2 - 3, Atostatin 40 mg 1/d, Pantoprazole 15 mg 1/d, Metformins 850 mg 2/d.

On the evolutionary level: complete regression of symptomatology on day 2 with NIHSS = 0. Ultrasound of ASD on day 3 shows the persistence of the thrombus

without anatomical or hemodynamic modification. On day 7 (Figure 2) good anatomical regression of the stenosis to 45% without hemodynamic impact. D10 continued regression of the thrombus without character stenosing.

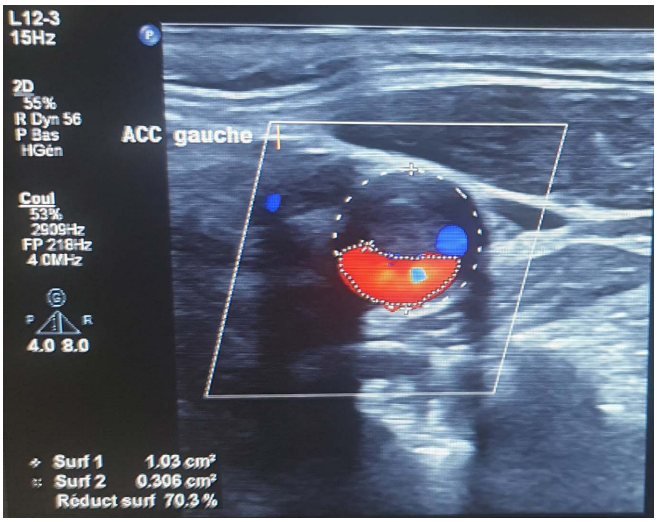

Figure 1. 70% stenosis of the CCG on day 1.

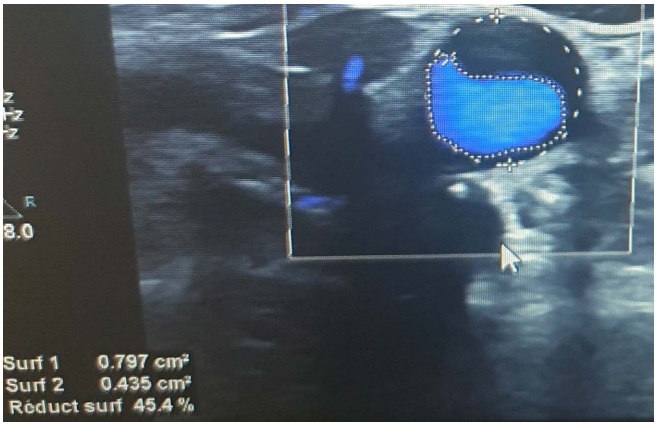

Figure 2. 70% stenosis of the CCG on day 7.

3. Discussion

CCT is a rare entity and its true incidence is unknown. The most common etiology is the complication of an atheromatous plaque; APS is a rare cause (Ferrero, Ferri, Viazzo, Labate, Pecchio, & Berardi, 2011a; Khalfi et al., 2024). However, CCT can be detected in young stroke patients without identifiable arterial disease (Vassileva et al., 2015). These patients are at high risk of stroke and death in the short term (Fridman et al., 2019). CCT is a common carotid artery endoluminal thrombotic material (Bouly et al., 2005). The source of the thrombus is not always known (Silver et al., 2002). CCT is more common in men than in women, it occurs at a younger age and is symptomatic in 92% of patients (Bhatti et al., 2007; Cancer-Perez et al., 2021). According to one study, approximately 70% of patients with symptomatic stroke stenosis of 70% to 99% had no subsequent recurrence for 5

years with better medical treatment (Naylor et al., 2014). Doppler ultrasound is a reference diagnostic examination for the diagnosis of CCT (Dermitzakis et al., 2002). Ultrasound allowed us to confirm and monitor the evolution of the patient's CCT. Doppler ultrasound constitutes an important means of visualizing the thrombus and assessing its hemodynamic impact (Eudo & De Bray, 2000). Multidimensional CT angiography is a new high-resolution imaging modality that allows rapid, simultaneous and minimally invasive visualization of cervical and cerebral vessels, including virtual endovascular views (Busch et al., 2002). The CCT observed in our patient is secondary to APS associated with thrombocytosis. APS is the most common acquired autoimmune thrombophilic disorder and a well-established cause of ischemia in young patients (Di Minno et al., 2012; Mittal et al., 2023). The diagnosis is based on at least one clinical criterion and one biological criterion, as was the case in our patient. A positive antiphospholipid test without clinical events does not indicate the APS as this may be secondary to another condition or treatment such as chlorpromazine (Cervera et al., 2002; De Laat et al., 2004). APS appears to be a significant marker of subclinical atherosclerosis, endothelial dysfunction and cardiovascular risk (Ambrosino et al., 2014). Premature atherosclerosis occurs in thrombotic APS over 30 years of age (Ames et al., 2009). The evaluation of carotid intima-media thickness on ultrasound allows the diagnosis of subclinical atherosclerosis, it has a higher sensitivity for early detection of atherosclerosis compared to angiography (Bots & Grobbee, 2002). When stroke is associated with APS, it is important to exclude CCT in the context of stroke prevention (Hagiwara et al., 2003). According to a large European cohort study, the mortality rate due to APS over a 5-year period was 5.3% (Espinosa & Cervera, 2010). Antiphospholipid antibodies are present in 30% to 50% of lupus patients (De Groot & Derksen, 2005). Antithrombotic treatment appears to be more effective for secondary prophylaxis of APS with arterial thrombosis but New Oral circulation anticoagulant (NOACs) should be avoided (Aibar & Schulman, 2021). In addition to APS, our patient presented persistent thrombocytosis with a high probability of essential thrombocythemia. According to Richard et al, essential thrombosis can be suspected even if the platelet count is not significantly increased. The diagnosis must be confirmed by a hematologist to initiate appropriate treatment (Richard et al., 2011). Essential thrombosis is rare in children and only a third of affected adult patients present a thromboembolic complication; the incidence of thrombosis during essential thrombosis varies from 11% to 25% (Bertrand et al., 2014; Suzuki et al., 2020). Essential thrombosis is a risk factor for stroke, mainly small vessel ischemia, so early diagnosis and treatment can promote the prevention of stroke recurrence (Pósfai et al., 2014). However, the degrees of initial severity of the stroke and the vital and functional prognosis after an acute ischemic stroke are not associated with this pathology (Furlan et al., 2016). The absence of Janus Kinase 2 (JAK2) gene mutation and histological evidence in our patient points us towards reactive thrombocytosis. According to some authors, reactive thrombocytosis can lead to carotid thrombosis and secondary acute ischemic stroke, particularly in young African-American women,

but this opinion is not unanimous (Nelson et al., 2022; Vardiman et al., 2002). The extent of thrombocytosis is not a criterion for distinguishing a primary process from a reactive process (Kim et al., 2019). The role of Platelet anti-aggregant (PAA) in the treatment of secondary thrombocytosis is not well established, which justifies their absence in our therapeutic protocol (Martínez et al., 2018). Screening for JAK2 mutation is complementary to histology for the diagnosis of ET and another chronic myeloproliferative disorder that can lead to thrombocytosis (Tefferi et al., 2007; Vardiman et al., 2002). This nor any gene mutation is specific to essential thrombosis (Vannucchi & Barbui, 2007). Myeloproliferative disorders can be the cause of persistent thrombocytosis in our body, hence the interest in the histological study of the marrow which has unfortunately not been carried out. Bone marrow biopsy as well as cytogenetic analysis are therefore necessary for a definitive diagnosis (Batista et al., 2019). Medical treatment with anticoagulation allowed us to have a spectacular reduction in CCT in our patient, without any clinical worsening or any possible surgical indication. Complete dissolution of CCT without further neurological progression is observed in 86% of medically treated patients (Caplan, 2003). Typically after starting immediate anticoagulation, the thrombus is completely dissolved within two weeks without further deterioration of the patient's symptoms (Yonemura et al., 2003). Our treatment regimen initially consisted of heparin followed by Antivitamin K (AVK). This therapeutic regimen is the most widespread in CCT, some practitioners add antiplatelet agents and others use only antiplatelet agents. However, there are no randomized trials to support these latter hypotheses (Caplan, 2003). Anecdotal evidence supports early initiation of intravenous unfractionated heparin to prevent stroke recurrence in CCT (Elijovich et al., 2013). Some literature data report that initial anticoagulation for symptomatic CCT results in a low rate of recurrent ischemic events and that carotid revascularization, if indicated, can be performed safely and in a delayed manner (Vellimana et al., 2013). The management of CCT by acute thromboendarterectomy remains little practiced, with fewer than 150 cases having been described to date in the world literature (Lane et al., 2010). This endovascular treatment should be encouraged (Li et al., 2016). Endarterectomy or endovascular embolectomy are effective and safe techniques in CBT to limit stroke recurrence, but they carry a real risk of distal embolization and stroke (Ferrero et al., 2011b; Goyal et al., 2016; Kazantsev et al., 2023). Thrombus migration is associated with a poor prognosis, it can cause dramatic neurological deterioration in patients with a mild initial clinical presentation (Koge et al., 2020). It is important to clarify that surgery has some advantages for symptomatic stenosis of 50% to 69% and is very beneficial for symptomatic stenosis of 70% or more (Rothwell et al., 2003). The treatment of CCT is not codified. Anticoagulation seems preferable initially; interventional and surgical treatment can be considered in the event of ineffectiveness or contraindication to anticoagulation (Khalfi et al., 2024). Timely surgical intervention and optimal medical treatment appear to be equally important (Rothwell, 2008). However, hematological disorders represent a small percentage of the etiologies of stroke, more frequent in young people (Crassard & Woimant,

2005).

4. Conclusion

Common carotid artery thrombosis (CCT) is a rare pathology, often discovered during the etiological assessment of stroke. It can occur in young subjects with a male predominance. Although osteoarthritis is the main etiology, it can be secondary to hemopathy. Our patient presents an association of etiology, a presentation rarely reported in the literature. There is no established superiority between medical treatment and surgery, nor any therapeutic protocol. However, initial medical treatment followed possibly by surgery due to lack of effectiveness of medical treatment could be a good therapeutic option. The prognosis is mostly good.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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