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Case Report Open Access

Contrast-Induced Encephalopathy after Endovascular Lower Limb Revascularization: A Rare Cause of Blindness

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Abstract

Background and objectives: Contrast-induced encephalopathy (CIE) after endovascular lower limb revascularization is an uncommon complication caused by the administration of intravascular contrast media. The clinical presentation of CIE is variable, ranging from cortical blindness to encephalopathy, seizures, and focal neurological deficits. It is often an exclusion diagnosis. In this case, CIE remains the most likely diagnosis on the basis of the symptoms and typical radiological findings. The authors want to point up how blindness after surgery led to CIE diagnosis and all the major complications that followed with patient dead.

Case report: We describe a case of a 77 year-old male patient who developed CIE after endovascular lower limb revascularization of the right iliac artery. He had a history of high blood pressure, type II diabetes mellitus, peripheral artery disease and cerebrovascular disease. Surgical procedure under general anesthesia was uneventful. The patient started complaining of bilateral blindness short after arrival at the Post Anesthesia Care Unit (PACU). A vascular event was excluded and the diagnosis of CIE was suggested through a cerebral angiography computed tomography scan. On the return to the PACU the patient developed hypovolemic shock from retroperitoneal hemorrhage. The event proved fatal despite further surgery and all resuscitative efforts.

Conclusion: This case highlights and should raise clinical awareness of a rare but important complication follow endovascular lower limb revascularization. CIE presented as blindness, a very rare complain after surgery. The diagnosis is challenging and requires imaging confirmation. Mobilization of the patient carries risks. In this case the retroperitoneal hemorrhage that followed proved fatal to the patient.

Keywords: Contrast-induced encephalopathy; Endovascular revascularization; Blindness

Introduction

Endovascular surgery is increasing as a complementary or alternative method to bypass graft surgery for treatment of peripheral artery disease [1]. Intravascular contrast administration is essential to these procedures but carries risks, namely nephropathy and anaphylaxis [2,3]. Contrast-induced encephalopathy (CIE) is a rare transient complication [4]. Clinical manifestations include encephalopathy, cortical blindness, seizures and focal neurological deficits [5]. Underlying mechanism of CIE remain unclear [6]. There are cases described in post-coronary angiography [7,8] and in post-neuroradiology interventions [9]. Herein, we report a case of 77-year-old man who developed CIE following peripheral angioplasty. To our knowledge, it is the first description in this type of intervention recognized in the immediate postoperative period.

Case Report

A 77-year-old male patient was scheduled for iliac kissing stent implantation for right lower limb revascularization. He had an increasing intermittent claudication for approximately 30 to 40 meters. Past medical history included controlled high blood pressure (amlodipine 10 mg, once daily, per os), type 2 diabetes mellitus under oral antidiabetics (metformin 500 mg, twice daily; sitagliptin 100 mg, once daily), ischemic stroke without sequelae dated 7 years before and peripheral artery disease with previous revascularization of the left leg. He had no complains suggestive of coronary artery disease and no symptoms of heart failure. The functional capacity was difficult to assess, however evaluation with a transthoracic echocardiography showed a good global biventricular systolic function. Electrocardiogram showed a normal sinus rhythm and the renal clearance was 63,42 mL/min. Contrast induced nephropathy prophylaxis was prescribed 24 hours before surgery (normal saline 41 ml/h and acetylcysteine 600 mg, twice daily, per os). General anesthesia was chosen since the patient was on clopidogrel and enoxaparin therapy. Standard ASA plus invasive arterial blood pressure, Bilateral Bispectral Index (BIS) and cerebral regional oximetry (*INVOS*™) monitors were used. The stent was placed in the

proximal portion of the common iliac artery after several progressive dilations with balloon by a bifemoral arterial approach. Intravenous 70 U/kg unfractionated heparin was administered and a total of 350 ml isosmolar and nonionic contrast medium (Visipaque®, osmolarity 702 mOsm/kg H_2O) was used. The patient remained clinically and haemodynamically stable without changes in the monitoring profile (Figure 1).

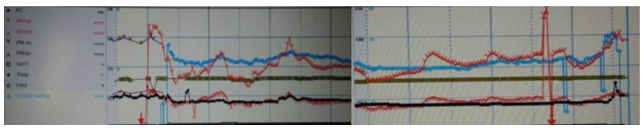

Figure 1: Profile of intraoperative monitoring.

After intervention the patient was taken to the PACU. Minutes after arriving he started complaining of bilateral blindness and became restless and confused. He was eupneic and haemodynamically stable, and had no other neurological deficits. No alterations in the blood gas analysis were observed. After discussion with the Ophthalmology and Neurology teams, an emergency cerebral angiography computed tomography scan was performed. It showed a hyperdensity and an enhancement of the occipital cortex bilaterally, with attenuation of the occipital cortical furrows. Acute vascular changes were excluded (Figure 2).

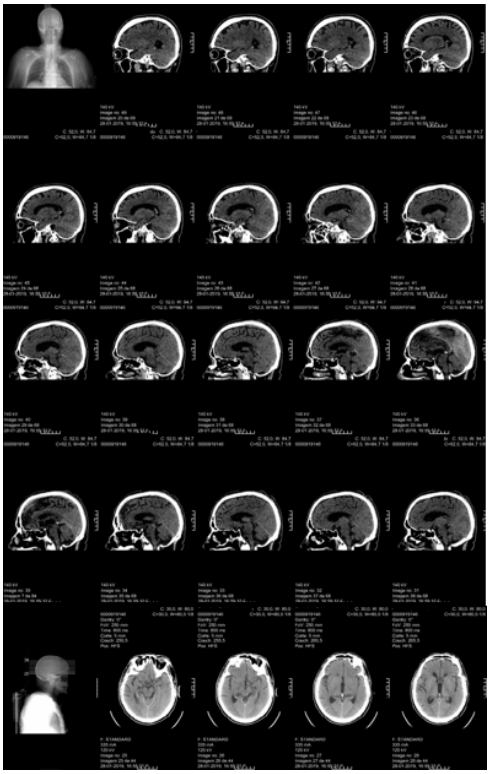

Figure 2: Cerebral angiography computed tomography images (Increased hyperdensity and enhancement of bilateral occipital cortex; Attenuation of occipital cortical furrows; Acute vascular changes excluded).

After mobilization and transport back to the PACU the patient became hypotensive, with a feeling of full bladder (although he had an indwelling catheter) and with signs of poor perfusion without visible hematic losses. A retroperitoneal hemorrhage was suspected and the patient was taken to emergent surgery. The diagnosis was confirmed and iliac arteriorraphy done. The hypovolemic shock revealed fatal hours later despite all resuscitative efforts.

Discussion

CIE is a very rare complication of diagnostic angiography and percutaneous interventions [10]. Clinical manifestations include encephalopathy, seizures, cortical blindness, and focal neurological deficits [11]. Patient's neurological status usually develops within minutes of exposure to the contrast medium. Spontaneous resolution of neurological status usually occurs within 72 hours, as it is cleared by the kidneys [12].

In the literature, all types of iodinated contrast agents, irrespective of their osmolarity or ionic states, can induce CIE [13]. The incidence ranges between 0.3% and 1% [14]. The mechanism is not exactly clear, but one possible explanation is a disruption of the blood-brain barrier [13,14]. The cause of disruption is attributed to the hyperosmolality and chemotoxicity of contrast media [15]. The occipital cortex is one of the regions with higher permeability of the blood-brain barrier. This explains the more frequent occurrence of neurological deficits such as cortical blindness and ophthalmoplegia. Commonly cited risks factors include chronic hypertension, [4,5] transient ischemic attack, [6] impaired cerebral autoregulation, [5,6] large contrast volume, [4,5] impaired renal function [2,5] and male gender [4,5] Due to their similar clinical presentations, it is essential to distinguish thromboembolic and hemorrhagic complications from CIE [13,16]. Brain CT and magnetic resonance values, such as the apparent diffusion coefficient can differentiate CIE from cerebral ischemia and subarachnoid hemorrhage [17]. An occipital cortical or subcortical enhancement is the most frequent finding of CIE in brain CT.

Although there is no specific treatment for this condition, hydration and close observation of the patient in the immediate post procedural period are recommended [6-8,17]. Symptomatic treatments, such as anticonvulsant therapy for seizures, are usually sufficient. In a few cases, patients have been treated with steroids to reduce cerebral oedema with no adverse consequences [9]. In most cases of CIE, the prognosis has been excellent, with rapid recovery with supportive management only [9-12].

Despite the early identification and diagnosis of CIE the outcome was not favorable in this clinical case. Patients with peripheral artery disease are at increased risk of stroke and imaging is essential for diagnosis and treatment [1]. The suspicion of CIE could have prevented a new administration of contrast for brain CT; however the literature is unaware of the risk of re-exposure to the contrast agent [13-15]. In addition, contrast prophylaxis with fluid therapy and acetylcysteine does not seem to prevent this entity [5,7,18]. In this case, male gender, a previous history of stroke and chronic hypertension may have induced changes in the blood brain barrier that, in association with the use of contrast, predisposed to CIE.

Conclusion

Our case highlights a rare but important complication follow endovascular lower limb revascularization that clinicians need to be aware of. The presentation of CIE is variable, ranging from cortical

blindness to encephalopathy, seizures, and focal neurological deficits, which often pose a difficult differential diagnosis, especially in these types of patients. In any acute neurologic dysfunction it is important to carry out the relevant investigations. CIE is often a diagnosis of exclusion. In this case, CIE remains the most likely diagnosis on the basis of the symptoms and typical radiologic findings.

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