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Case Report and Review of the Literature

Contrast-Induced Encephalopathy Post Gadolinium Administration: A Case Report and Literature Review

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ABSTRACT

Background: Contrast-induced encephalopathy (CIE) is an unusual and transient complication of intra-arterially administered contrast media during interventional procedures. We report a case of CIE post-gadolinium-based contrast administration.

Case Description: A 53-year-old female patient who was presented with a long-standing history of headaches and left-sided weakness. The patient had medical history of type II diabetes mellitus, hypertension, dyslipidemia, and transient ischaemic attacks. Due to the past medical history of repetitive transient ischaemic attacks, a brain MRI was done, anterior communicating artery saccular aneurysm was incidentally discovered. So, the decision was made to proceed with cerebral angiography which revealed a non-ruptured saccular aneurysm of the anterior communicating artery that was surgically clipped. On postoperative day 4, a gadolinium-based contrast CT angiogram revealed complete exclusion of the aneurysm. Following the CT angiogram, the patient developed confusion, disorientation, aphasia, and generalized tonic-clonic seizures. An urgent head CT was done, showing unremarkable findings. Conservative measures were used to manage CIE. The patient was discharged, and the postoperative period was uneventful.

Conclusion: The clinical features of CIE are non-specific, resulting in diagnostic challenges. Multiple risk factors predispose patients to CIE. Supportive measures are the main treatment option.

Introduction

Contrast-induced encephalopathy (CIE) is an uncommon and temporary neurological condition that is linked to intraarterial iodinated contrast injection [1, 2]. This condition is a rare type of neurotoxicity and can be a transient and life-threatening situation [3]. The incidence of CIE is usually manifested by encephalopathy, or focal neurological deficits including aphasia, seizures, and visual disturbance [2, 4, 5]. These manifestations developed within hours of contrast media exposure [6, 7]. Despite the reversibility of this condition, a fatal encephalopathy case was reported [2]. Statistically, CIE incidence ranges from 0.3% to 2% with no gender preference [3, 8]. Of these, 4% is associated with ionic and hyperosmolar contrast materials [8].

Herein we report a 53-year-old female patient who became confused, disoriented, and aphasic post-brain MRI with an IV Gadolinium-based agent. The condition was found to be representative of contrast-induced encephalopathy.

History and Case Presentation

A 53-year-old female patient who was presented with a long-standing history of headaches and left-sided weakness. The patient's history was notable for type II diabetes mellitus, hypertension, dyslipidemia, and

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transient ischaemic attacks. Due to the past medical history of repetitive transient ischaemic attacks, a brain MRI was done, anterior communicating artery saccular aneurysm was incidentally discovered. So, the decision was made to proceed with cerebral angiography that revealed a non-ruptured saccular aneurysm of the anterior communicating artery, which was clipped through the left pterional craniotomy (Figures 1 & 2). Postoperatively, the patient did well, was fully conscious, and had no neurological deficits. On postoperative day 4, a CT angiogram revealed complete exclusion of the aneurysm.

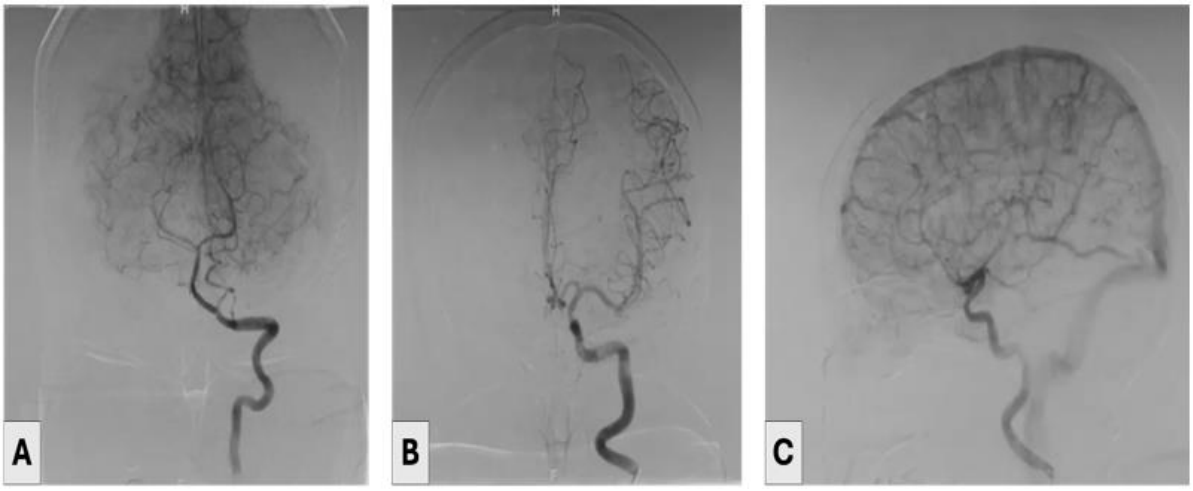

Figure 1: A-C) Illustrate the cerebral angiogram that showed an anterior communicating artery aneurysm.

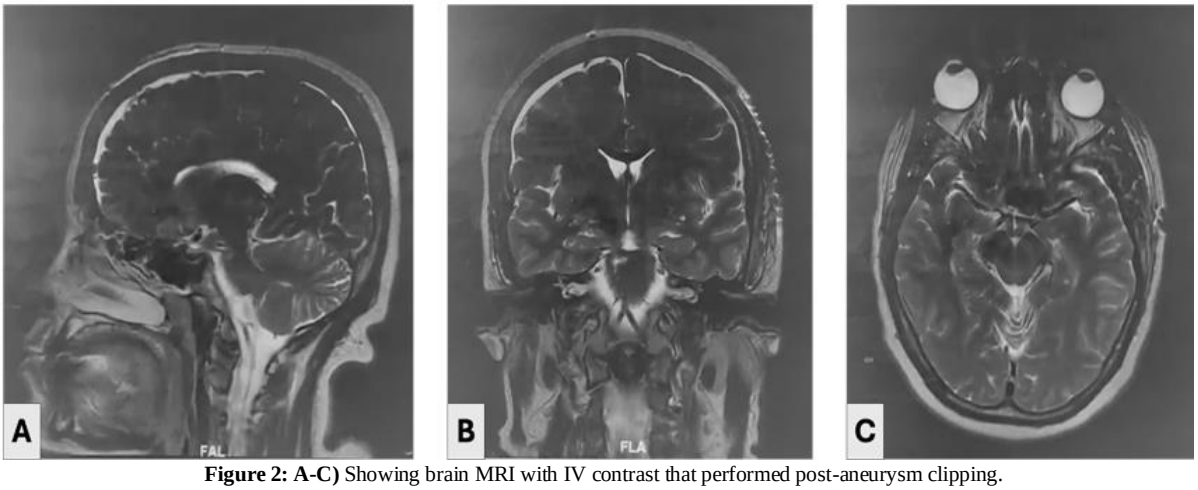

Figure 2: A-C) Showing brain MRI with IV contrast that performed post-aneurysm clipping.

Five hours later, the patient appeared confused, disoriented, and aphasic. No meningeal irritation was detected. An acute kidney injury was developed due to the contrast administration in the CT angiogram. Hydration and nephrotoxic agents should be avoided. The next morning, the patient developed generalized tonic-clonic seizures. Levacetam was administered in a loading dose and 1 gram twice daily as a maintenance dose. An urgent head CT was done, showing unremarkable findings.

The patient was discharged on the postoperative day 11 and was fully conscious and oriented. Upon follow-up, the patient showed clinical improvement.

Discussion

CIE is an unusual adverse consequence of interventional and diagnostic angiography [1]. In 1970, the first case of CIE was documented [3]. Then, Kanda *et al.* first recognized patients' brains that were exposed to linear gadolinium-based contrast media revealed patterns of high-intensity signals in the dentate nucleus and globus pallidus [9].

Patho-physiologically, the underlying mechanism is believed to occur due to blood-brain barrier disruption following the administration of iodinated, low-osmolar, and non-ionic contrast substances as gadolinium material in the case of iodine-allergy [1, 2, 8, 10]. Therefore, cerebral vasogenic edema can result from direct neurotoxicity and hyperosmolality induced by the contrast media in the extracellular region [2]. A case reported gadolinium neurotoxicity and oxidative reaction due to brain tissue deposition in gadolinium-induced encephalopathy [10]. The deposition of gadolinium-based contrast agents was mainly detected in the globus pallidus and dentate nucleus regions [9, 11]. The role of gadolinium contrast agent in blood-brain barrier malfunction is characterized by excessive production of free radicals and mitochondria dysfunction. This action is achieved through contrast material spillage and accumulation in brain tissues [10]. Additional pathological

mechanisms, such as arterial vasospasm with disruption of microcirculation, have been hypothesized [8].

Benzon *et al.* discussed 3 studies demonstrating the incidence of gadolinium-induced reactions after pain-control intervention procedures including gadolinium being documented as a hypersensitivity reaction [9]. Multiple risk factors contributing to CIE have been reported and include renal disease, acute cerebrovascular events, dyslipidemia, and hypertension. Also, CIE can be further triggered by higher concentrations or lesser temperatures of the contrast agents [1, 3, 12].

The clinical presentation of patients with CIE is non-specific. The common clinical features involve transient visual impairment, disorientation, epileptic events, speech disturbance, and hemiplegia or hemiparesis [8, 9, 11]. Our patient was confused and developed seizures and aphasia. We speculate that our patient's medical history of hypertension, acute kidney injury, and other risk factors contributed to her symptom severity, leading to neurotoxicity and CIE by increasing blood-brain barrier permeability.

Symptoms of CIE are distinguished by their hyperacute onset during cerebral angiography and by the reversibility of their clinical findings [1]. Several studies documented the onset of CIE symptoms immediately following cerebral angiography or within hours [1, 6]. Typically, the clinical condition improves in 24 to 72 hours [1, 2, 6]. Nevertheless, cases with longer duration, reversibility, and fatality have been documented [1, 3]. A study reported that the major CIE risk factors were a history of cerebrovascular events and renal disorders [9]. Others include diabetes mellitus, systemic hypertension, and utilization of high doses of contrast agents [3, 6].

Since CIE manifests similarly to hemorrhagic or embolic consequences after a cerebral angiogram, CT, or MRI, it is crucial to exclude other pathological processes [2, 4, 12]. In order to differentiate CIE from a cerebral hemorrhage or a life-threatening stroke, abnormalities on urgent CT scans that exhibit the distinctive high-intensity signals, 80 to 160 Hounsfield units, can be recognized as diagnostic hallmarks [7]. Studies reported further neuroradiological findings that can aid CIE detection [6, 8, 13]. These include contrast pigmentation of cerebral parenchyma and/or subarachnoid space and edematous cerebral regions surrounding the infarct core [6, 8, 13]. Moreover, on T2-FLAIR, gadolinium enhancement of leptomeningeal layers was documented [12]. In the presented case, other etiologies were excluded by performing ECG, EEG, and head CT.

Even though there is insufficient clinical data regarding CIE medical treatment, the most frequently identified approach involves intensive intravenous hydration with crystalloids, anticonvulsants, and steroid administration in the initial phases of supportive therapy [1, 4, 7]. Moreover, mannitol was used to manage cerebral edema in some patients [6, 12]. However, mannitol was not used in managing the presenting case. In our case, the patient was treated through a conservative approach with IV fluids and close monitoring of epileptic seizures.

Conclusion

Despite the rarity and reversibility of CIE after interventional procedures, it should be considered a complication after contrast material administration. CIE is a diagnosis of exclusion. Close observation and hydration are the basic management lines. For proper identification and management, awareness of the characteristic radiological and clinical findings of CIE is highly recommended.

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Ethical Approval

Institutional Review Board approval is not required.

Declaration of Patient Consent

The authors certify that they have obtained all appropriate patient consent.

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None.

Conflicts of Interest

None.

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