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Case Report /Vaka Raporu

An unusual complication following percutaneous coronary intervention: Contrast-induced encephalopathy

Perkütan koroner girişim sonrası sıradışı bir komplikasyon: Kontrast madde kaynaklı ensefalopati

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ABSTRACT

Contrast-induced encephalopathy (CIE) represents an infrequent adverse event linked to angiographic procedures. The clinical presentation encompasses various focal neurological impairments, notably encephalopathy, seizure activity, ophthalmoplegia, and cortical blindness. We hereby detail the clinical course of a 43-year-old man who developed CIE subsequent to a successful revascularization procedure for a Left Anterior Descending Artery Chronic Total Occlusion (LAD-CTO). The elective coronary intervention was finalized with the administration of 300 ml of iohexol. Post-procedurally, the patient suffered a convulsion and was immediately admitted to the Intensive Care Unit. Following the onset of cortical blindness, appropriate central nervous system imaging was conducted to eliminate the possibility of hemorrhagic or ischemic events, initiating hydration as the primary management strategy. The patient achieved a full recovery of his visual function within 18 hours of treatment commencement. In summary, CIE is an exceptionally rare yet recognized complication of both diagnostic angiography and percutaneous interventions. Clinicians must maintain a heightened index of suspicion for this condition during the evaluation of acute post-procedural neurological changes.

Key Words: Blindness, contrast induced encephalopathy, percutaneous coronary intervention

ÖZ

Kontrast maddeye bağlı ensefalopati (KİE), anjiyografik işlemlerle ilişkili nadir görülen bir komplikasyondur. Klinik tablo, özellikle ensefalopati, nöbet aktivitesi, oftalmopleji ve kortikal körlük olmak üzere çeşitli fokal nörolojik bozuklukları kapsar. Bu yazıda, Sol Ön İnen Arter Kronik Total Tıkanıklık (LAD-CTO) için başarılı bir revaskülarizasyon prosedürünün ardından KİE gelişen 43 yaşında bir erkeğin klinik seyrini ayrıntılı olarak açıklıyoruz. Elektif koroner girişim, 300 ml ioheksol uygulamasıyla sonlandırıldı. İşlem sonrasında hastada konvülsiyon gelişti ve hemen Yoğun Bakım Ünitesi'ne yatırıldı. Kortikal körlüğün başlamasının ardından, hemorajik veya iskemik olay olasılığını ortadan kaldırmak için uygun merkezi sinir sistemi görüntülemesi yapıldı ve birincil tedavi stratejisi olarak hidrasyon başlatıldı. Hasta, tedavinin başlamasından sonraki 18 saat içinde görme fonksiyonunda tam bir iyileşme sağladı. Özetle, CIE hem tanısal anjiyografi hem de perkütan girişimlerin son derece nadir görülen ancak bilinen bir komplikasyonudur. Klinisyenler, akut işlem sonrası nörolojik değişikliklerin değerlendirilmesi sırasında bu duruma karşı yüksek bir şüphe ile yaklaşmalıdır.

Anahtar Kelimeler: Körlük, kontrast kaynaklı ensefalopati, perkütan koroner girişim

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INTRODUCTION

Contrast-induced encephalopathy (CIE) is recognized as an uncommon complication arising from angiographic procedures. This condition was first reported in 1970, initially characterized as a transient episode of cortical blindness observed following coronary angiography (CAG) (1). The syndrome's clinical presentation encompasses a range of neurological signs, including generalized encephalopathy, seizure episodes, cortical blindness, and distinct focal deficits such as ophthalmoplegia (2). While the overall incidence of CIE typically falls between 0.3% and 1.0%, the rate can escalate to as high as 4% when hyperosmolar iodinated contrast agents are utilized (3). Our current case report details the occurrence of CIE attributable to iohexol, a non-ionic contrast agent, subsequent to a CAG procedure.

CASE REPORT

A 43-year-old male patient attended our cardiology outpatient clinic complaining of chest pain. Initial assessment revealed no chronic comorbidities, and that the patient had undergone coronary angiography only four months prior. Initial tests showed that the patient's baseline haematology and biochemistry results were normal. The patient was also found to have normal glomerular filtration values. He underwent coronary angiography four months ago, after which it was reported that his left anterior descending artery (LAD) was 100% chronic total occluded (Figure 1a). Myocardial perfusion scintigraphy revealed more than 10% ischemia despite maximal medical treatment. The patient was referred to the CAG laboratory for the elective LAD chronic total occlusion procedure. The procedure was performed with the administration of 100 U/kg unfractionated heparin and the utilisation of 300 mL iohexol for angioplasty. The LAD was successfully revascularized (Figure 1b). However, the patient subsequently experienced convulsions minutes after the conclusion of the procedure. Consequently, emergency intervention was performed, and the patient was subsequently transferred to the coronary intensive care unit. After approximately 1-2 minutes of loss of consciousness, the patient regained consciousness. However, one hour after the procedure, the patient developed visual impairment and cortical blindness within seconds.

Emergency brain computed tomography scan shows intravascular contrast material from recent coronary angiography. The presence of symmetrical hyperdensities in the posterior parts of the parietal (Figure 2a), occipital (Figure 2b) and temporal lobes, as well as the superior parts of the cerebellar hemispheres (Figure 2c), is indicative of contrast agent escape into the subarachnoid spaces. The involvement of the bilateral occipital lobes is particularly noteworthy, as it is a contributing factor to the onset of cortical blindness. Subsequent diffusion MRI scans revealed no acute ischemic pathology (Figure 3). A consultation with the ophthalmology department was also requested. The ophthalmologist conducted a thorough ophthalmological examination, which revealed that the anterior segments of both eyes were normal, the intraocular pressure was 12/13 mmHg (within the normal range), and the fundus and optic disc were unremarkable on both sides. Following a comprehensive evaluation of all findings, the patient was administered 100 ml of saline per hour. Neurological symptoms improved 18 hours after the procedure following fluid therapy. The patient was diagnosed with CIE, a diagnosis that was made on the basis of the patient's clinical findings and brain imaging.

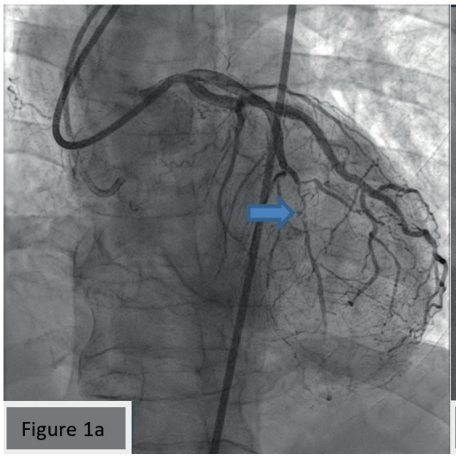

Figure 1a

DIAGNOSTIC IMAGE – LAD CTO

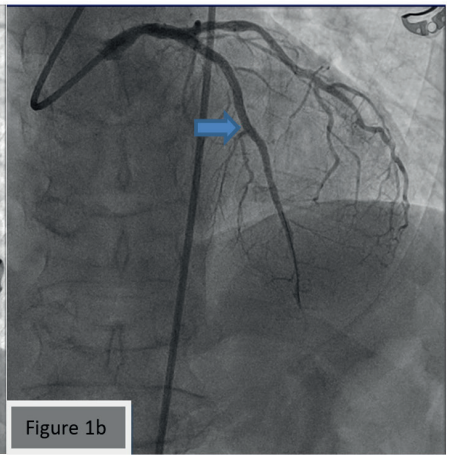

Figure 1b

LAD AFTER REVASCULARIZATION

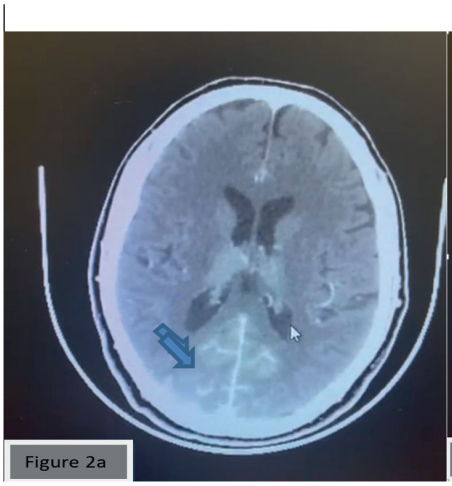

Figure 2a

HYPERDENSE AREAS IN THE PARIETAL LOBE

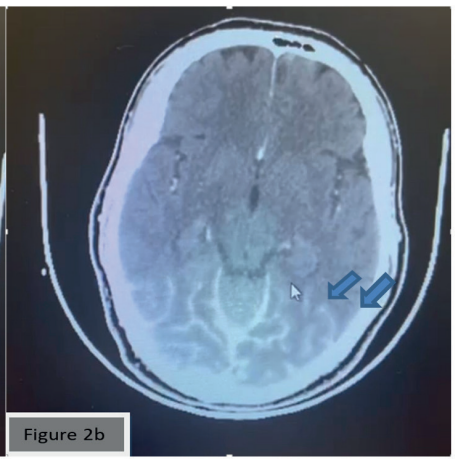

Figure 2b

HYPERDENSE AREAS IN THE OCCIPITAL LOBE

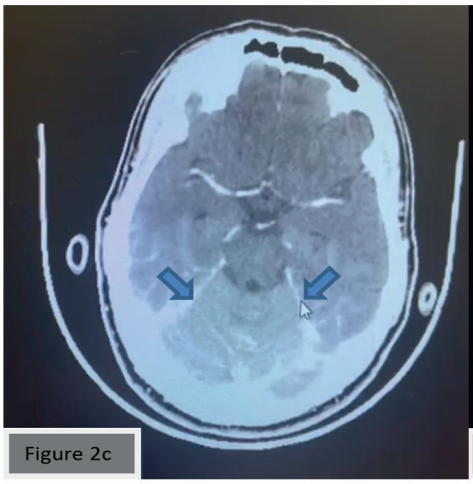

Figure 2c

HYPERDENSE AREAS IN THE TEMPORAL LOBE

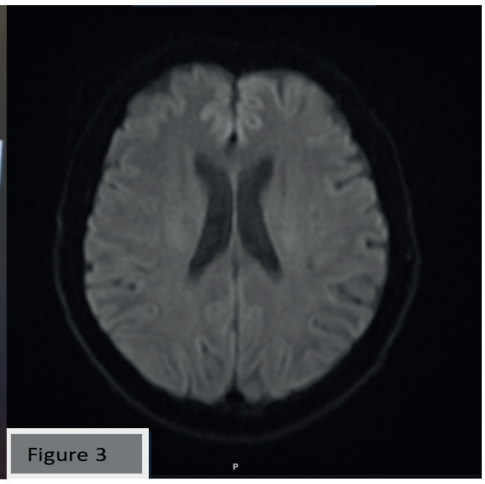

Figure 3

DIFFUSION MRI

DISCUSSION

CIE is a notably rare complication of both diagnostic angiography and percutaneous interventions (4). Although its detection is most frequent after cerebral angiography, CIE has also been documented following cardiac or peripheral angiography, as well as contrast-enhanced CT scans (5). M. Fischer-Williams et al. first documented CIE in 1970, yet the underlying pathomechanisms remain incompletely elucidated to date. One defining study characterized contrast-induced neurological dysfunction by its presentation minutes to hours post-injection of iodinated contrast media; its transient nature, with symptoms typically resolving within 48 to 72 hours; and the exclusion of other pathological processes like cerebral ischemia, hemorrhage, epilepsy, arterial dissection, or air embolism as causal factors (6).

A wide array of contrast agents, including ionic, non-ionic, hyperosmolar, and isosmolar types, have been implicated in inducing CIE (7). While the typical incidence of CIE spans between 0.3% and 1.0%, this rate has been observed to increase to as much as 4% when hyperosmolar iodinated contrast materials are employed (3). Although the exact mechanism is not fully understood, previous case reports and studies suggest that direct neurotoxicity and a compromised blood-brain barrier are both implicated. (2,8). This disturbance is commonly attributed to the hyperosmolality and chemotoxicity inherent to the contrast medium. The occipital cortex is recognized as a region displaying higher permeability of the blood-brain barrier, which is theorized to explain the increased prevalence of neurological deficits, including cortical blindness and ophthalmoplegia, in this condition. Predisposing factors include chronic hypertension, episodes of transient ischemia, compromised cerebral autoregulation, exposure to large contrast volumes, impaired renal function, male sex, and selective vertebrobasilar arteriography (10).

Given the symptomatic overlap, distinguishing CIE from thromboembolic and haemorrhagic complications post-angiography is essential. Imaging is vital for confirming the diagnosis and ruling out thromboembolic and hemorrhagic events (11). Non-contrast brain CT and magnetic resonance imaging (MRI) metrics, particularly the apparent diffusion coefficient (ADC) values, are useful for differentiating CIE from cerebral ischemia and subarachnoid haemorrhage. Establishing an accurate CIE diagnosis is critical to preventing the risks associated with inappropriate treatments, such as administering thrombolytics for suspected acute cerebrovascular ischemia or proceeding with surgery for subarachnoid hemorrhage (10).

Although no dedicated definitive treatment exists for this condition, the recommended management involves initiating hydration and ensuring close patient observation during the immediate postoperative period (11). For seizure management, symptomatic treatment with anticonvulsant therapy is usually sufficient. In isolated reports, patients have been successfully managed with steroids and mannitol, with no resultant adverse outcomes documented (2).

In summary, CIE is a highly infrequent complication of diagnostic angiography and percutaneous intervention. Clinicians should maintain awareness of this syndrome and consider it within the differential diagnosis.

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