Posterior reversible encephalopathy syndrome caused by contrast media after percutaneous coronary intervention

İbrahim Saraç[a], Gökhan Tonkaz[b], Faruk Aydınyılmaz[a], Emrah Aksakal[a]

[a] *Department of Cardiology, University of Health Sciences, Erzurum Education and Research Hospital, Erzurum, Turkey*
[b] *Department of Radiology, University of Health Sciences, Erzurum Education and Research Hospital, Erzurum, Turkey*

ARTICLE INFO

Article history:
Submitted: 2. 12. 2022
Revised: 4. 1. 2023
Accepted: 22. 1. 2023
Available online: 9. 10. 2023

Klíčová slova:
Encefalopatie
Kontrastní látka
Perkutánní koronární intervence

SOUHRN

Používání kontrastních látek je nedílnou součástí invazivních výkonů, hlavně radiodiagnostických, které se provádějí pro stanovení diagnózy a při léčbě na katetrizačním sále. Aplikace kontrastních látek po perkutánní koronární intervenci (PCI) je spojena s řadou komplikací. Vzácně popisovanou komplikací, která může vést k rozvoji encefalopatie, je kontrastní látkou indukovaná neurotoxicita. Tento článek se věnuje syndromu posteriorní reverzibilní encefalopatie (posterior reversible encephalopathy syndrome, PRES) jako vzácnému důsledku neurotoxicity vyvolané jódovou kontrastní látkou s akutním nástupem, která je – při časném stanovení diagnózy a zahájení léčby – reverzibilní; přičemž diagnózu lze stanovit neurologickým vyšetřením pomocí radiologických a zobrazovacích metod.

Keywords:
Contrast agent
Encephalopathy
Percutaneous coronary intervention

ABSTRACT

Contrast media use is an integral part of invasive procedures, mainly radiodiagnostic procedures, performed for diagnosis and treatment in the cardiac catheterization laboratory. There are many complications related to contrast agents after percutaneous coronary intervention (PCI). Contrast-induced neurotoxicity is a rarely reported complication that may result with encephalopathy. In this report, posterior reversible encephalopathy syndrome (PRES), a rare consequence of neurotoxicity of iodinated contrast agents with acute onset, reversible with early diagnosis and treatment, and diagnosed by neurologic examination radiological imaging methods, will be mentioned.

Introduction

Many complications occur due to the use of contrast material. One of these complications is neurotoxicity associated with the use of contrast agents. This situation, mainly reported after neurovascular interventions, has also been rarely seen in patients who underwent coronary angiography and may result in encephalopathy.[1] Encephalopathy can also be seen as posterior reversible encephalopathy syndrome (PRES), usually accompanied by rapidly progressive acute onset mental status changes, visual disturbances, paresis, and widespread seizures, often having a transient clinic and regressing within days. Cranial imaging in this syndrome is usually characterized by predominantly subcortical, parieto-occipital reversible vasogenic brain edema.[2] PRES is due to heterogeneous etiologies, triggering factors including preeclampsia/eclampsia, sudden rising, uncontrollable blood pressure, renal failure, immunosuppressive drugs, chemotherapeutic agents, cytotoxic drugs, hyponatremia, and hypercalcemia.[3] Despite its benign and clinically and radiologically reversible course, cases resulting in permanent brain damage, severe functional impairment, and mortality have also been reported.[4,5] In this report, PRES syndrome developing after PCI will be discussed.

Case report

Seventy-one-year old male patient, with a history of coronary artery bypass graft (CABG) surgery ten years ago, stage 3 (Glomerular Filtration Rate [GFR]: 40–60 mL/min/1,73 m^2), chronic renal failure (CRF), type 2 diabetes mellitus (DM) and hypertension (HT), presented to the emergency department with typical chest pain that started about 3 hours ago. At admission, arterial blood pressure (TA): 130/80 mmHg, oxygen saturation (SO_2): 93, cardiac troponin I (cTnI): 789 ng/mL (0–45 normal range), serum creatinine 1.32 mg/dl (GFR: 56 mL/min/1,73 m^2) and other examinations were within the normal range. There was no ST-segment elevation in electrocardiography (ECG). Transthoracic echocardiography (TTE) showed the ejection fraction (EF):

Address: İbrahim Saraç, MD, Department of Cardiology, University of Health Sciences, Erzurum Education and Research Hospital, Erzurum, Turkey, e-mail: drsaracc@gmail.com
DOI: 10.33678/cor.2023.009

Please cite this article as: Saraç İ, Tonkaz G, Aydınyılmaz F, Aksakal E. Posterior reversible encephalopathy syndrome caused by contrast media after percutaneous coronary intervention. Cor Vasa 2023;65:779–782.

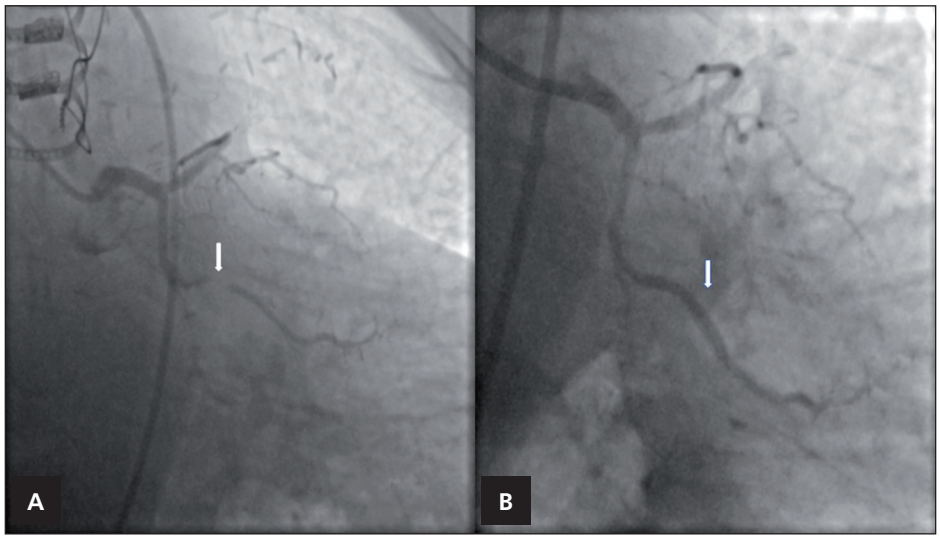

Fig. 1 – CAG images of the patient before and after PCI. (A) Culprit lesion in OM-1 before the procedure (white arrow), (B) OM-1 (white arrow) after DES implantation.

45%, and the left ventricular (LV) inferolateral wall was observed hypokinetic. Coronary angiography (CAG) was planned for the patient because of severe angina. A 99% acute thrombosed atherosclerotic lesion was observed in the CX- obtuse margin-1 (OM-1) native vessel. No acute significant stenotic lesions were detected in other coronary vessels and grafts. After pre-dilatation, a 2.5 * 20 mm drug--eluting stent (DES) was implanted into the responsible lesion, and optimal patency was achieved (Fig. 1).

No complications developed during the procedure. One hour after he was admitted to the coronary intensive care unit, headaches, changes in consciousness, confusion, difficulty in speaking, and vision problems began to appear. The patient did not have respiratory distress, and his rhythm was sinus 79/min, TA: 135/85 mmHg. Serum electrolytes, sedimentation, serum reactive protein (CRP), and white blood cell (WBC) were within the normal range. The patient was consulted at the neurology clinic because of the progress in loss of consciousness and vision and the development of generalized tonic-clonic seizures. No hemorrhagic and ischemic cerebrovascular events were observed in the brain computed tomography (CT), magnetic resonance (MR), MR angiography, and cerebral venography imaging. An appearance consistent with increased vaso-

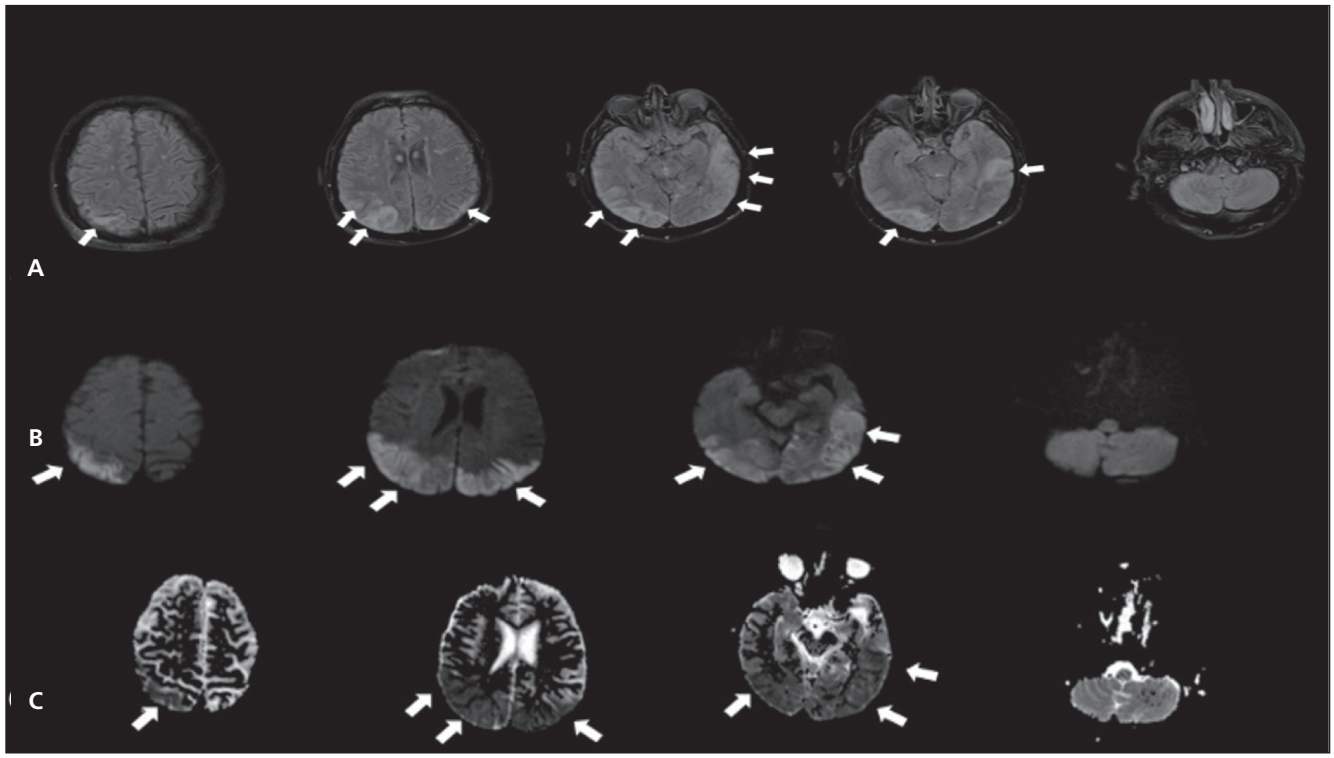

Fig. 2 – Images on cranial MRI of a 71-year-old man with PRES at diagnosis. (A) Axial T2-weighted FLAIR images showed hyperintensity signals reflecting vasogenic brain edema in the cortical and subcortical areas of the bilateral parietal, occipital, and left temporal lobes. (B, C) Diffusion-weighted images and ADC maps at 1000 s/mm^2 show hyperintense areas in bilateral parietal, occipital, and left temporal lobes.

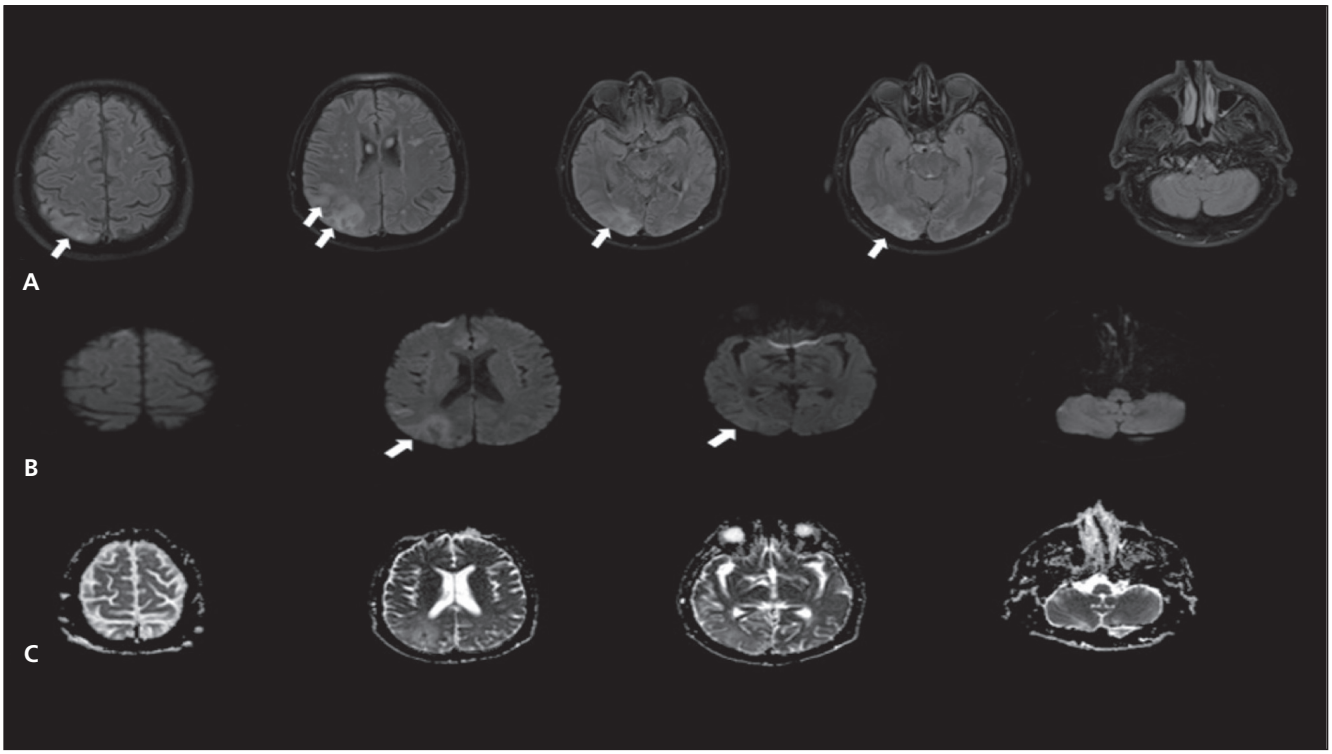

Fig. 3 – Images on cranial MRI of a 71-year-old man with PRES at follow-up. (A) Axial T2-weighted FLAIR images after three weeks showed a significant regression in the hyperintensity signal areas reflecting vasogenic brain edema. (B, C) Control images after three weeks show a substantial reduction in vasogenic edema.

genic edema in the occipitoparietal and temporal areas was observed in the brain MR images (Fig. 2).

The patient was diagnosed with PRES syndrome, and medical treatment was started in the presence of existing clinical and radiological findings. The cardiac and respiratory examination of the patient was normal. On the 2nd day of follow-up, he had spontaneous breathing and was completely unconscious. Corticosteroids and supportive treatment was administered to the patient intravenously. We used diazepam for the prevention of epileptic seizures. It was observed that there was a regression in the current findings in the follow-up MRI images of the patient (Fig. 3).

The patient, who was followed up in our clinic for about one month, had bilateral mild central vision loss but was fully conscious. The patient was discharged without any additional neurodeficiency.

Discussion

Contrast encephalopathy was reported at a frequency of 0.06% in patients who underwent CAG, while this rate was higher in patients who underwent carotid-vertebral angiography.[6] The mechanism of encephalopathy due to iodine-containing contrast agents has not yet been fully elucidated. Compared with low-osmolality or iso-osmolar contrast agents, hypertonic contrast agents have a more significant toxic effect on the blood-brain barrier. The basic pathophysiology has been attributed to edema and neuronal toxicity due to increased permeability in the blood-brain barrier.[7] In addition, the endothelin (ET) peptide family has been shown to play a significant role in the pathogenesis of contrast encephalopathy. In many studies, endothelin-1 (ET-1) levels after contrast agent administration in patients undergoing PCI have shown a significant correlation with the amount of contrast agent.[8] It is known that the release of potent vasoconstrictor mediators such as ET-1 in normotensive patients with PRES causes vasospasm, ischemia, and brain edema.[9] Although symptoms related to encephalopathy may occur immediately after contrast agent administration, they usually occur within the first 72 hours in most patients.[3,10] Neurological signs and findings that emerged approximately 1 hour after PCI was performed in the patient who applied with acute coronary syndrome (ACS) resulted in the patient's complete loss of vision and consciousness. In this process, laboratory tests were in the normal range, and hemodynamics was stable. No finding compatible with ischemic and hemorrhagic infarct was found in the cranial imaging performed on the patient. Contrast encephalopathy was considered because the typical imaging findings were detected on CT and MRI, and acute encephalitis and other neurological pathologies were excluded in the patient.[3] Although the contrast agent we use during the procedure is non-ionic, has a low osmolar structure, and is much less neurotoxic, encephalopathy has also been reported due to the use of these agents.[10] In a literature review investigating the relationship between the volume of contrast material applied and encephalopathy, it was seen that an average of 169 ml of contrast material was used (minimum 75 ml – maximum 500 ml).[10] Again, in many studies, the relationship between contrast nephropathy and the patient's creatinine clearance (ClCr) was investigated. A significant increase in the frequency

of contrast nephropathy and other complications was observed in patients with increased contrast volume / ClCr ratio.[11] In this case, 178 ml of contrast material was used. The ClCr of our patient was 57, and the contrast volume / ClCr ratio was measured as 3.4. Although it is below the risk ratio defined in many studies, it was observed that contract-related nephropathy and encephalopathy developed in many patients below this ratio and volume.[11,12] In addition to the use of increasing volumes of contrast agents, male gender, hypertension, advanced age, DM, acute renal failure, CRF, previous history of neurotoxicity due to contrast media, presence of impaired cerebral autoregulation, presence of a history of transient ischemic attack are defined risk factors for the development of contrast media encephalopathy.[3] Although an excessive amount of contrast material was not used in our patient, his comorbidities may have contributed to the development of encephalopathy.[3] Eventhough most cases of contrast medium encephalopathy have a good prognosis, cases resulting in persistent neurogenic deficits, permanent central blindness, and mortality have also been reported.[5,13] In patients admitted with PRES, respiratory tract control should be ensured first. The drugs used for seizures in non-pregnant patients are diazepam, phenobarbital, and fosphenytoin. In case of recurrent seizures, administration of propofol or midazolam can be started. Dialysis should be considered for patients who develop renal insufficiency.[14] Patients with PRES should be followed up with close hemodynamic monitoring. In addition to intravenous hydration and supportive therapy, steroids and mannitol can also reduce cerebral edema and inflammatory reactions.[13] Heart failure and stroke are independent risk factors for mortality in PRES.[15] In addition to improving patients' cardiac function, heart failure-specific treatments indirectly reduce the frequency of ischemic cerebrovascular events by preventing the occurrence of atrial fibrillation (AF) or reducing the burden of paroxysmal AF.[16,17] We applied the medical treatment and additional supportive treatment to our patient, and he became fully conscious two weeks later. After one month of treatment, significant radiological and clinical improvement was observed. We evaluated this contrast-induced encephalopathy, which is generally reversible with appropriate follow-up and treatment, as PRES syndrome.

Conclusion

In conclusion, although PRES is a well-known syndrome, it is not a standard and familiar condition for interventional cardiology. PRES should be considered in the differential diagnosis of cerebrovascular events after interventional procedures using a contrast agent. Early diagnosis of PRES and the exclusion of other possible causes determine the treatment approach, which is very important in the clinical prognosis and survival.

Conflict of interest
None.

Funding
In our study, the source of funding for the project are the researchers themselves. There is no other source of financing.

Informed consent
Written informed consent was obtained from the patient in this case.

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