Contrast-induced monoplegia following coronary angioplasty with iopromide
Monoplegia wywołana kontrastem po angioplastyce wieńcowej z zastosowaniem jopromidu

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Abstract
Seizures, alterations in mental and cerebral functions, and ophthalmoplegia are known side effects of contrast agents. Here we report a case of self-limiting monoplegia in a patient after the administration of intracoronary iopromide after coronary angiography which emphasises that, although benign, contrast-induced monoplegia is a neurological disease which requires careful evaluation and accurate management.

Key words: iopromide, contrast agent, encephalopathy, monoplegia, coronary angiography

A 68 year-old male patient was admitted to our emergency department with new onset, squeezing type, chest pain. His physical examination was normal. Electrocardiography revealed ST segment depression in anterior precordial leads with T wave inversion. He had hypertension and hyperlipidaemia for seven years. Heparin 5,000 U intravenous, 300 mg acetyl salicylic acid, and 600 mg clopidogrel was administered to the patient, who was then transferred to the catheter laboratory for coronary angiography because of unstable angina pectoris. Coronary angiography revealed 90% stenosis in the mid segment of the left anterior descending artery, and a 2.75 × 25 mm sirolimus-eluting stent implantation was performed in the same session. Full coverage was achieved with TIMI-3 flow. A total dose of 250 mL was injected to the coronaries. Within four hours, the patient complained of nausea and an inability to move his left lower extremity with a loss of sensation in the lower extremity, and then he became confused. Babinski sign was present in the left lower extremity with loss of motor function and sensation. A computerised tomography (CT) scan of the brain was performed which revealed extravascular localised contrast media in the sagittal sinus and occipital lobe. After 12 hours, he recovered completely without any neurologic deficits by use of supportive medications and adequate hydration. Cranial diffusion magnetic resonance imaging (MRI) was performed after 36 hours. This revealed normal findings and the contrast agent was reabsorbed from the subarachnoid space (Fig. 1).

Non-ionic contrast agents usually have several side effects including seizure, alteration of cerebral function, confusion, short-term memory loss, mental aberrations and ophthalmoplegia [1, 2]. Also encephalopathy after administration of non-ionic contrast agents has been reported in the literature [4–7]. The blood brain barrier mostly prevents the entry of contrast agent into the central nervous system (CNS), but cerebral angiography and aortography may alter the barrier and increase the amount of contrast agent entering the CNS [3].
But encephalopathy after coronary intervention is very rare and has been reported only on a few occasions [7–10]. The clinical scenario in this patient was consistent with intracranial haemorrhage, but there must be a sign in the cranial CT. It may be postulated that early radiologic evaluation in intracranial haemorrhage is not always 100% accurate, but we performed cranial MRI to the patient 36 hours after the situation. Furthermore, the symptoms were resolved spontaneously. Another possible explanation may be transient ischaemic attack, but this was clearly exluded in the cranial MRI. The patient was free from any metabolic disease or abnormality which could be a contributory cause of monoplegia and confusion.

So the only remaining explanation in this scenario was that monoplegia was induced by the contrast agent, which was iopromide. Unfortunately, we could not perform an electroencephalography because of the short duration of symptoms.

There is a debate about the mechanism and causes of neurotoxicity. In high concentrations, hypertonic contrast agents can disrupt the blood brain barrier and facilitate their entry into the CNS. Repeated injections of contrast agents may result in neurotoxic effects within several minutes [3, 6]. The leakage of the contrast into the cerebrospinal fluid and electrolyte imbalance may cause an encephalopathy [3]. Extravasation of contrast agent usually affects the posterior circulation and causes cortical blindness. The presented case did not have any visual problems. Clearance of the contrast agent from CNS may be achieved via backward washing of this agent and sink action of cerebrospinal fluid. Sustained high concentrations of the agent in the CNS may be observed in patients with renal failure [3]. Our case is the first in the literature to describe iopromide-induced monoplegia.

Although it is rare, contrast-induced monoplegia and encephalopathy may occur during coronary intervention without a particular predisposition. Despite the benign nature of contrast encephalopathy, it may be an early warning sign of future nervous system disease. Thus, close follow up of these patients is necessary.

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflict of interest: none declared

References