Brief Communications

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Recurrent contrast-induced encephalopathy following coronary angiography

Roberto Spina,¹ Neil Simon,^{2,3} Romesh Markus,² David W. M. Muller^{1,3} and Krishna Kathir¹

Departments of ¹Cardiology, and ²Neurology, St Vincent's Hospital, and ³St Vincent's Clinical School, University of New South Wales, Sydney, New South Wales, Australia

Key words

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Correspondence

Roberto Spina, Department of Cardiology, St Vincent's Hospital, 390 Victoria Street, Darlinghurst, NSW 2010, Australia. Email: roberto.spina@hotmail.com

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Abstract

Contrast-induced encephalopathy (CIE) is an acute and reversible neurological disturbance associated with the intra-arterial administration of iodinated contrast medium during cardiac catheterisation. It may manifest with encephalopathy, motor and sensory disturbances; vision disturbances, including cortical blindness, ophthalmoplegia, aphasia; and seizures. Disruption of the blood-brain barrier and direct neuronal toxicity are believed to be implicated in the pathophysiology of the syndrome. Symptoms appear soon after contrast administration and resolve completely within 24-48 h. Risk factors may include hypertension, diabetes mellitus, renal impairment, the administration of large volumes of iodinated contrast, percutaneous coronary intervention or selective angiography of internal mammary grafts and previous adverse reaction to iodinated contrast. On cerebral imaging, CIE may mimic subarachnoid haemorrhage or cerebral ischaemia, but imaging may be normal. Prognosis is excellent with supportive management alone. CIE may recur, but re-challenge with iodinated contrast without adverse effects has been documented. CIE is a diagnosis of exclusion and is an important clinical entity to consider in the differential diagnosis of stroke following cardiac catheterisation. Physicians should be aware of it and consider it prior to initiating thrombolysis.

A 65-year-old, left-handed man presented with intermittent retrosternal chest discomfort of several hours duration. His medical background included coronary artery disease, with a stent implanted in the first obtuse marginal branch in 2001. He had hypertension and dyslipidaemia and reported a 15 pack/year of smoking. Following pre-medication with intravenous hydrocortisone 100 mg and promethazine 25 mg because of a previous

Funding: None. Conflict of interest: None. reaction to iodine contrast, the details of which the patient could not recall, and regarding which we did not have any readily available documentation, coronary angiography was performed through the right radial approach. A total of 110 mL of low-osmolar, non-ionic monomeric iodine-based contrast agent (iopromide; Ultravist 370, Bayer Healthcare, Pittsburgh, PA, USA) was used. The procedure demonstrated a patent obtuse marginal stent and minor coronary disease elsewhere. Within minutes of radial sheath removal, the patient developed mild respiratory distress, and mild erythema appeared over the upper trunk. He became rapidly drowsy, confused and disorientated. His speech was slurred, and within a further 5-10 min, he developed complete aphasia. He also complained of weakness in the legs. Neurological examination demonstrated profound global aphasia with intact comprehension. Cranial nerves were intact. He had normal tone in all limbs, normal power in the upper limbs and mildly reduced power in (4/5) in the lower extremities. Coordination was not affected. Sensory examination was normal. Computed tomography (CT) scanning of the brain without additional contrast performed within 30 min of symptom onset, and within 45 min of completion of the procedure, demonstrated no abnormality (Fig. 1). All major intracranial arterial vessels were visualised, given the recent iodine contrast administration during the coronary angiogram, and appeared unobstructed. Following the CT brain scan, and at the time the neurology service was considering intravenous thrombolysis for stroke, details became available of a previous cardiac catheterisation performed at another institution 2 years prior and complicated by a stroke-like syndrome. The latter episode is described here before returning to the index case. Two years prior, approximately 10 min after the completion of a diagnostic cardiac catheterisation through the right femoral route, during which 120 mL of iopromide were used, the patient became drowsy, confused and disorientated, with his level of consciousness fluctuating between 8 and 10 out of 15 on the Glasgow Coma Scale. He also developed limb weakness, with upper limb strength (4/5) affected less than the lower limb strength (3/5). The remainder of the neurological examination was normal, as were vital signs, serum glucose, ECG and CT brain. He was treated with intravenous hydrocortisone 100 mg three times daily and intravenous fluids. He recovered full consciousness within hours of the event, and upon return of clear mentation, he had a hazy recollection of the events. Magnetic resonance imaging (MRI) of the brain with intracranial angiography, performed approximately 24 h after the event, together with an MRI of the spinal cord with sagittal and axial sequences and MRI of the chest with aortography did not demonstrate any acute pathology to explain his symptomatology (Fig. 2). He recovered limb strength fully within 72 h.

With the information available regarding the previous possible neurotoxic contrast reaction, and given the non-



Figure 1 Non-enhanced computed tomography of the head performed shortly after the onset of contrast-induced encephalopathy demonstrating no abnormality.



Figure 2 Magnetic resonance angiography of the cerebral vessels performed 24 h after the onset of symptoms demonstrates no abnormality.

localising nature of the neurological symptoms and the lack of evidence on cerebral CT to suggest acute cerebral infarct, a decision was made not to administer thrombolysis. With supportive therapy consisting of intravenous fluids and close observation, he regained full strength in his legs within 6 h, and 12 h post-completion of the angiogram, his speech normalised completely. He did not recollect the events immediately following angiography.

Our patient presented on two separate occasions with a constellation of non-lateralising neurological symptoms and altered mentation of rapid onset following coronary angiography. His symptoms resolved within 24-48 h. Neurological imaging (CT and MRI/A in the first episode and CT in the second episode) of the brain and of the extra-cranial and intra-cranial vessels was normal on both occasions, ruling out cerebral infarction or obstructive cerebrovascular disease. Air embolism was ruled out on both occasions by careful review of the angiographic images obtained at cardiac catheterisation. Transient cerebral ischaemia appeared unlikely given the nonlocalising neurological symptom complex; it is not anatomically possible to attribute his presenting symptoms of altered mentation, profound global aphasia and upper limb weakness to a single cerebral artery territory, although it is conceivable that multiple emboli caused symptoms and signs in different vascular territories. However, if the phenomenon was cardioembolic in nature, one could reasonably expect some abnormal findings even on initial imaging studies given his

profound speech disturbance. It is also statistically improbable that he would experience two transient ischaemic attacks following two successive cardiac catheterisations and not at any other point in time. Finally, the non-specific nature of his neurological symptomatology, coupled with an altered level of consciousness and retrograde amnesia, points rather to an encephalopathic process temporally related to the coronary angiogram.

Discussion

The incidence of stroke following cardiac catheterisation has been estimated from registry studies to range between 0.05 and 0.10% for diagnostic coronary angiography^{1,2} and between 0.12 and 0.40% for percutaneous coronary intervention (PCI).²⁻⁴ Haemorrhagic stroke accounts for about 16-45% of all strokes following PCI.^{3,4} Neurological adverse effects of contrast media are a recognised, albeit uncommon, complication of invasive cerebral angiography, with an estimated prevalence rate of 1–2%.⁵ CIE following cardiac catheterisation procedures, on the other hand, is rare. It may manifest with encephalopathy, motor and sensory disturbances; vision disturbances, including cortical blindness, ophthalmoplegia, aphasia; and seizures.⁶ Disruption of the blood-brain barrier and direct neuronal toxicity are believed to be implicated in the pathophysiology of the syndrome. Symptoms typically appear soon after contrast administration and resolve completely within 24-48 h. Risk factors may include hypertension, diabetes mellitus, renal impairment, the administration of large volumes of iodinated contrast, percutaneous coronary intervention or selective angiography of internal mammary grafts and previous adverse reaction to iodinated contrast.⁶ On cerebral imaging, the syndrome of CIE may mimic subarachnoid haemorrhage or cerebral ischaemia. Typical findings on non-enhanced CT study performed shortly after the onset of CIE include hemispheric contrast staining of the cortical sulci and hyperdensity of the subarachnoid space attributable to iodinated contrast extravasation, followed by disappearance of the abnormalities on follow-up imaging.⁷ The density of the lesion measured on the Hounsfield scale on CT is useful in distinguishing haemorrhage from contrast extravasation: whereas blood has a density ranging between 40 and 60 Hounsfield units (HU), contrast media have higher attenuation values, usually between 100 and 300 HU.8

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The absence of xantochromia or red blood cells on examination of the cerebrospinal fluid argues against the diagnosis of subarachnoid haemorrhage. MRIderived apparent diffusion coefficient (ADC) is a reliable imaging modality that differentiates contrast extravasation from cerebral ischaemia.9 In acute ischaemic stroke, decreased water diffusion in infarcted tissue causes a decreased ADC. In CIE, the apparent diffusion coefficient is normal. In some cases, brain imaging is normal. Management is typically supportive, and prognosis is excellent. CIE tends to recur, although rechallenge with iodinated contrast without adverse effects has been documented.¹⁰ CIE is a diagnosis of exclusion and is an important clinical entity to consider in the differential diagnosis of stroke following cardiac catheterisation. Physicians should be aware of it and consider it prior to initiating thrombolysis.

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