

Contrast-Induced Encephalopathy Post-Angiography

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Citation

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Abstract

Contrast agents are an important diagnostic tool used in many imaging modalities but carry risk. Of these risks, contrast-induced encephalopathy is a rare adverse event reaction to contrast media, but carries significant morbidity. While this reaction is most common in cerebral diagnostic and therapeutic intervention, it can also be found post-coronary angiography. In addition to the mental state disturbance associated with this condition, derangements of other neurological functions have also been associated with contrast agents. While the underlying mechanism of contrast-induced encephalopathy remains unclear, there have been many postulated theories and contributory factors outlined in the literature. Despite the infrequency of this complication, due to the associated morbidity and neurological sequelae associated with contrast-induced encephalopathy, better vigilance amongst medical staff is required in a post-diagnostic or –interventional period in procedures utilising contrast media.

CONTRAST-INDUCED ENCEPHALOPATHY POST-ANGIOGRAPHY

A 49 year old male underwent a coronary angiogram at a tertiary hospital for investigation of a three-week history of intermittent chest pain with a positive exercise electrocardiogram and dyspnoea on exertion.

There was no relevant past medical history, in particular, no history of renal impairment, and the patient's baseline estimated glomerular filtration rate was 83 mL/min/BSA.

An investigative angiogram was performed, utilising 130mL of Iopromide contrast solution (Ultravist-370). Angiography revealed mild non-obstructive coronary artery disease. Post-angiography, Computed Tomography (CT) of the abdomen was performed to investigate post-operative hypotension after removal of the femoral sheath. This required an additional 75mL of Iopromide solution (Ultravist-370), and revealed no obvious retroperitoneal or other bleeding.

Four hours later, the patient complained of a severe bitemporal headache, however there was no associated confusion. This ceased with pharmacological intervention (Oxycodone 5mg). An hour following, the patient was visibly confused, drowsy, emetic and clinically obtunded. The patient was unable to consistently follow verbal commands; however vital signs were unremarkable at this time. The patient had a Glasgow Coma Scale (GCS) of 10,

and on thorough neurological examination had no focal signs.

A non-contrast CT scan of the brain was subsequently performed to exclude intracerebral haemorrhage and revealed no obvious abnormalities, however the GCS decreased to 8 and the patient developed neurological findings, including brisk lower limb reflexes and positive Babinski sign bilaterally. The patient was subsequently intubated. Magnetic Resonance Angiography, looking for an occlusive event in the posterior circulation, was performed but revealed no obvious abnormalities, thereby excluding a structural cause for acute encephalopathy. Over the next day, neurological symptomatology and signs resolved and the patient was extubated. Electroencephalography was performed and revealed no epileptiform activity.

A preliminary diagnosis of contrast-induced encephalopathy with complete neurological recovery was made. The patient was discharged, and informed that any future procedure involving contrast should be avoided.

DISCUSSION

Non-ionic contrast agents are an important and useful tool in diagnostic enhancement of imaging modalities, however are not without risk. Most notable of these risks include non-specific adverse drug reactions (including injection site warmth, urticaria, erythema, nausea and/or vomiting)¹ and contrast nephropathy in those with impaired renal

function.² A lesser known, but just as important side effect is contrast-induced encephalopathy.

Contrast-induced encephalopathies are a rare adverse effect of contrast media, but there are known cases reported in the literature, predominantly associated with endovascular intervention or diagnostic imaging directly involving the cerebral circulation, whereby the cause may be multifactorial.^{3,4} A relatively less common causation of this entity is contrast use during coronary angiography, with only a few reported cases in the literature.^{5,6} Furthermore, derangement of other neurological function has been associated with contrast agents, including seizures, ophthalmoplegia, upper motor neuron dysfunction, and transient cortical blindness.^{7,8}

The novel features of this case include a delayed onset of encephalopathy post-contrast infusion, as well as the concept of a “second-hit” dose relationship. The reasoning for these events is unclear, but it does bare thought that perhaps there is a threshold for clinical effects with contrast dose, which is influenced by individual susceptibility.

The exact underlying mechanism of contrast-induced

encephalopathy is unclear, however, neurotoxicity due to contrast media is a well-recognised phenomenon. Possible contributing factors include disruption of the blood-brain barrier, direct and indirect neurotoxicity to neurons, arterial vasospasm and microvascular sludging in the cerebral vasculature.⁹⁻¹¹ These effects are seen at higher concentrations and are more frequently associated with ionic and high-osmolar agents, however, non-ionic, low-osmolar contrast media can still be associated with adverse reactions involving the neural circuitry, as was seen in this case.¹² Fortunately, the transition to these newer contrast media solutions has resulted in a decline in the incidence of this complication.

Despite the infrequency of this complication, medical staff should be vigilant in the post-angiographic procedure period for signs and symptoms of neurological dysfunction. This would translate to better outcomes for the patient in the short- and long-terms, and possibly deter further utility of unnecessary contrast to a patient at-risk of developing or already displaying features of this condition.

References

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