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The possible role of iodinated contrast in enhancing acute kidney injury in a critically ill neonate – a case report

Wevelin Santos de Matos[1], Fernando Silva Ramalho[2], Inalda Facincani[1], Paulo Henrique Manso[1]

A multi-modality approach is necessary for accurate diagnosis in case of congenital heart diseases present in the neonatal period. Advances in medical imaging have increased dependence on the information provided by radiological studies. However, several of these studies require the use of contrast, which may enhance the risk of contrast-induced acute kidney injury. We report on a case of a neonate with transposition of great arteries who was submitted to iodinated contrast during angiotomography. Soon after the procedure, he presented anuria and increased urea and creatinine levels, and was diagnosed with a contrast-induced acute kidney injury. Despite the use of peritoneal dialysis, he died soon thereafter. Post mortem analysis showed extensive coagulative necrosis in the renal cortical and medullary layers.

Key words: ACUTE KIDNEY INJURY; HEART DEFECTS, CONGENITAL; CONTRAST MEDIA

INTRODUCTION

Severe congenital heart diseases present in the neonatal period require a multi-modality approach for accurate diagnosis (1). Advances in medical imaging have increased dependence on the information provided by radiological studies. However, several studies require the use of contrast, which may increase the risk of contrast-induced acute kidney injury (CI-AKI) (2).

Herein, we report a case of a neonate with transposition of the great arteries in whom iodinated contrast induced AKI.

CASE REPORT

The report was approved by the local Ethics Committee (CAE: 31564820.7.0000.5440), and an informed consent of the next of kin was obtained. A full-term male neonate weighing 2.9 kg was delivered at home and presented cyanosis and dyspnoea a few hours after birth. He was brought to the hospital on the first day of life, and inhaled oxygen and antibiotics (ampicillin + gentamicin) were started. On the third day of life, transthoracic echocardiogram showed transposition of the great arteries, a three-mm ductus arteriosus, and a small foramen ovale (two mm in diameter). The baby was immediately submitted to balloon atrioseptostomy in the catheterisation laboratory. Coronary angiography showed normal coronary arteries. However, coronary angiogram raised suspicion of aortic coarctation. After the procedure, the patient presented mild arterial hypotension and reduced urinary output. Dopamine, adrenaline, and furosemide were started on the fifth day of life. Because the patient presented signals of infection and low cardiac output syndrome, vancomycin was started on the 11th day of life, and vasoactive drugs had their dosages increased. The surgical team requested aortic computed tomography (angio CT) to rule out aortic coarctation before the arterial switch operation. Angio CT was performed on the 11th day of life, and a bolus of iodinated contrast (300 mg of iodine

[1] Department of Pediatrics, Ribeirao Preto Medical School, University of Sao Paulo
[2] Department of Pathology, Ribeirao Preto Medical School, University of Sao Paulo

Correspondence to:
Paulo Henrique Manso, PhD, MD, Avenida Bandeirantes, 3900. Ribeirão Preto-SP. Brazil. 14049-900, Department of Pediatrics. Ribeirao Preto Medical School. University of Sao Paulo, e-mail: phmanso@fmrp.usp.br

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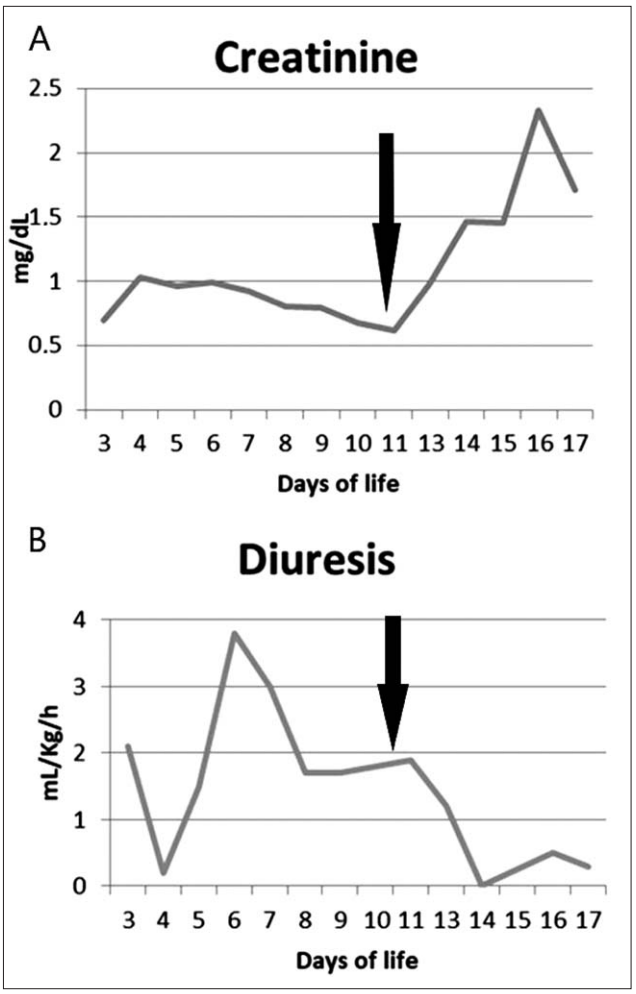

FIGURE 1. Serum creatinine levels (A) and diuresis volume (B). Arrows indicate the day when iodinated contrast was applied.

per mL) at a dose of 2 mL/kg was used, followed by saline injection. Twenty-four hours after angio CT, the patient presented low urine output; anuria; oedema; increased plasma creatinine, potassium, and urea; and decreased estimated glomerular filtration rate (eGFR) (Figure 1 and 2), with a diagnosis of CI-AKI. Although peritoneal dialysis was started on the next day, the patient died five days after angio CT.

Autopsy ruled out aortic coarctation, but it showed severe pallor of the cortical layer of both kidneys. Histopathological analysis revealed extensive coagulative necrosis in the renal cortical and medullary layers. Vascular congestion and haemorrhagic foci were also observed in both kidneys (Figure 3). In addition, large areas of coagulative necrosis and sinusoidal congestion appeared in the liver parenchyma.

DISCUSSION

The growing use of contrast for diagnostic radiological methods has made CI-AKI the third cause of hospital-acquired AKI in adults (3). Medullar kidney inflammation, hypoxia, and oxidative stress are the main factors underlying CI-AKI (4), which is defined as an increase in creatinine level above 25% of the baseline or 0.5 mg/dL on three days fol-

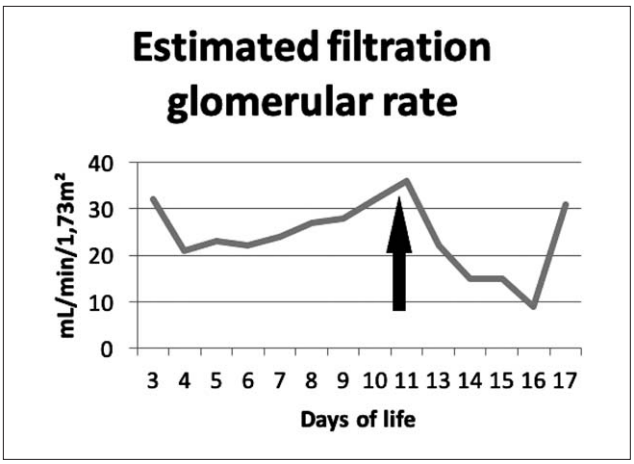

FIGURE 2. Schwartz-estimated glomerular filtration rate. Arrow indicates the day when iodinated contrast was applied.

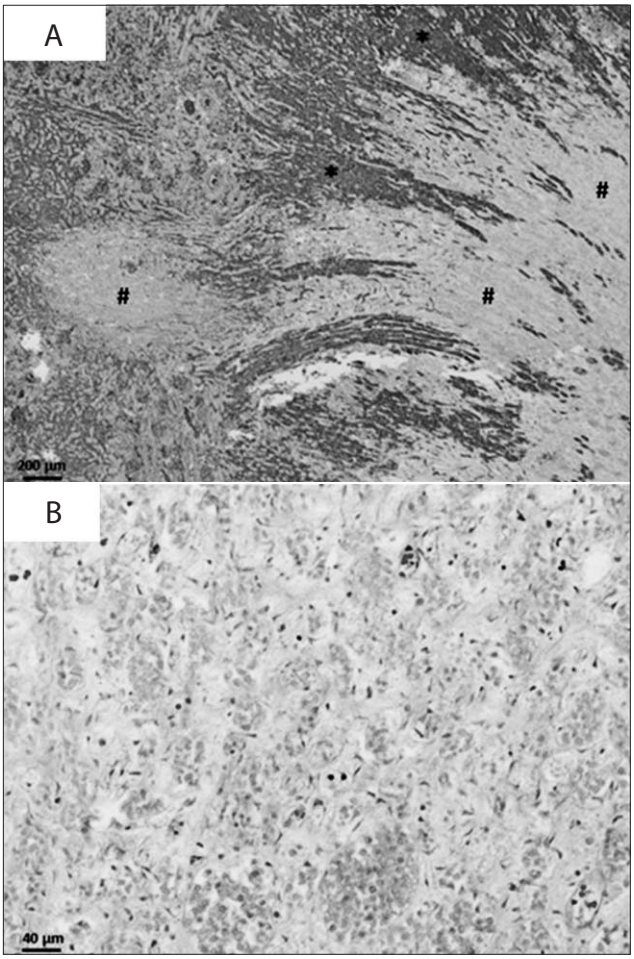

FIGURE 3. Representative photomicrographs of kidney sections: extensive coagulative necrosis (#) affects the renal cortex and medulla. Foci of haemorrhage are frequent in the renal cortical-medullary transition zone (*) (A); tubular epithelium in the renal cortical layer is totally necrotic (B).

lowing contrast medium administration (5). According to these criteria, already validated in a paediatric critical care population (6), our patient presented CI-AKI.

The incidence of CI-AKI in adults varies from 2% to 3.9%, but it may be as high as 50% in patients with associated risk factors such as diabetes, arterial hypertension, and pre-existing renal dysfunction (7).

In paediatric population, CI-AKI incidence ranges from 3.3% to 10%, depending on the patient cohort (3, 8). Risk factors including hypotension, congenital heart disease, cyanosis, use of nephrotoxic antibiotics, and vasoactive agents may have a role, but such factors have not been well described as risk factors in paediatric population (3).

Although the patient's renal function worsened after iodinated contrast use during angio CT, the real role of iodinated contrast is unclear in this scenario because the patient was also receiving furosemide, vancomycin, and vasoconstrictor agents such as dopamine and adrenaline. The patient also had a cyanotic heart disease. Deteriorated kidney function after iodinated contrast led us to suspect CI-AKI, but a cumulative effect of multiple insults to the kidney may have also contributed to the fatal outcome (9, 10).

Contrast-induced acute kidney injury is thought to develop secondary to vasoconstriction in the outer kidney medulla, which may lead to acute tubular necrosis (5). Coagulative necrosis is a typical early response to hypoxia, ischemia, or toxic injury. In this patient, all the associated factors may have contributed to kidney injury. The fact that the liver also presented coagulative necrosis indicates that not only iodinated contrast, but also ischaemia and hypoxia were related to the injury.

Intravenous hydration is the only intervention that benefits CI-AKI patients. The use of antioxidant agents such as N-acetylcysteine or theophylline is controversial (11, 12). Our patient was receiving furosemide, and therefore robust intravenous hydration was not performed. Even the beginning of peritoneal dialysis did not reverse the scenario, and the patient died.

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SAŽETAK

Moguća uloga iodiniranog kontrasta u pogoršanju akutnog oštećenja bubrega kod kritično bolesnog novorođenčeta – prikaz bolesnika

Wevelin Santos de Matos, Fernando Silva Ramalho, Inalda Facincani, Paulo Henrique Manso

Više modalitetni pristup neophodan je za postizanje točne dijagnoze u slučaju prirođenih srčanih grešaka prisutnih kod novorođenčeta. Napredak u medicinskim slikovnim metodama povećao je ovisnost o podacima dobivenim radiološkim pretragama. Međutim, neke od tih pretraga zahtijevaju primjenu kontrasta, što može povećati rizik od kontrastom izazvanog akutnog oštećenja bubrega. Opisujemo slučaj novorođenčeta s transpozicijom velikih arterija, koje je izloženo iodiniranom kontrastu tijekom angiotomografije. Nedugo nakon ovoga postupka kod novorođenčeta je nastupila anurija uz povišene razine ureje i kreatinina pa je dijagnosticirano akutno oštećenje bubrega izazvano kontrastom. Ubrzo potom dijete je umrlo usprkos primjeni peritonejske dijalize. Analiza provedena post mortem pokazala je proširenu koagulativnu nekrozu kortikalnih i medularnih slojeva bubrega.

Ključne riječi: AKUTNO OŠTEĆENJE BUBREGA; SRČANE GREŠKE, PRIROĐENE; KONTRASTNA SREDSTVA