Editorial

Thirsty Kidneys: Case Report

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Abstract

Contrast induced nephropathy is a reversible form of acute kidney injury occurs following administration of radio-contrast agents. It is associated with serious adverse short and long-term outcomes. The risk is negligible in children with normal renal function but increased in children with underlying kidney disease, dehydration and on nephrotoxic drugs. Contrast induced nephropathy is defined as increase in serum creatinine by >0.5 mg/dL or 25% increase from baseline within 48 to 72 hours of contrast administration. It is one of the commonest cause of hospital acquired acute kidney injury. We present a case of 10 years old boy with normal kidney function who developed acute kidney injury following intravenous contrast administration.

Keywords: Acute kidney injury, Contrast nephropathy, Hemodialysis

Introduction

Acute kidney injury (AKI) is one of the independent risk factor for mortality and morbidity in critically ill hospitalized children [1,2]. Nephrotoxic medications are the frequently attributed cause of AKI in these children. Nonsteroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting enzyme inhibitors, antibiotics and radio contrast agents are the frequently encountered drugs as a cause for AKI in this group. Contrast induced AKI (CI AKI) is one of the leading causes of hospital acquired AKI. It is also known to increase the duration of hospital stay significantly.

Case History

A previously well 10 years old boy presented with fever, abdominal pain and vomiting of 3 days duration. History of blunt abdominal injury a month ago while he was riding a bicycle. His urine output was good. He was evaluated with ultrasound abdomen elsewhere revealed splenic cyst with hemorrhage, hence started on antibiotics (Inj. Amikacin) and referred to our centre for further management. On arrival he was afebrile with the heart rate of 100/min, respiratory rate of 24/min, BP of 100/70mmHg. He had tenderness over epigastrium, umbilical and left iliac region. Other system examinations were not contributory.

His investigations showed hemoglobin of 10.8g/dL, total white blood cell counts of 20,300/ cumm with 72% polymorphic predominance, sterile blood culture, normal blood urea nitrogen (6mg/dL), serum creatinine (0.4mg/dL), serum electrolytes and urine routine. Serum amylase was mildly elevated, 125u/L and serum lipase was normal. Contrast enhanced computed tomography of abdomen showed laceration at the tail of pancreas with well-defined cyst in peri pancreatic region anterior to tail of pancreas suggestive of pancreatic pseudo cyst. There was no pathology in other organs. He underwent laparoscopic cysto gastrostomy after approximately 40 hours of contrast study. His urine output was nil intra as well as post operatively. He did not respond to fluid challenge. Other wise he was hemodynamically stable. Repeat blood urea nitrogen was 36mg/dL, serum creatinine was 4.1mg/dL and electrolytes were within normal limits.

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In view of anuric AKI with significant elevated creatinine, hemodialysis (HD) was initiated. Using 10 Fr double lumen catheter, right femoral vein was accessed, HD was done with F4 dialyser, with the blood flow of 150ml/min, dialysate flow of 300ml/min, concurrent dialysis for the initial session and subsequent countercurrent dialysis with a total of 4 HD sessions. Repeat CT abdomen on 3rd post-operative day showed bilateral renomegaly and mild dilatation of proximal ureter with distal tapering on both sides [Figure 1& 2]. Hence viscous contrast material blocking both side ureter causing oliguric AKI was considered as the cause. He started making urine on 4th post-operative day and gradually urine output improved to 2ml/kg/day. He was discharged on 12th postoperative day with the creatinine of 0.3mg/dL.

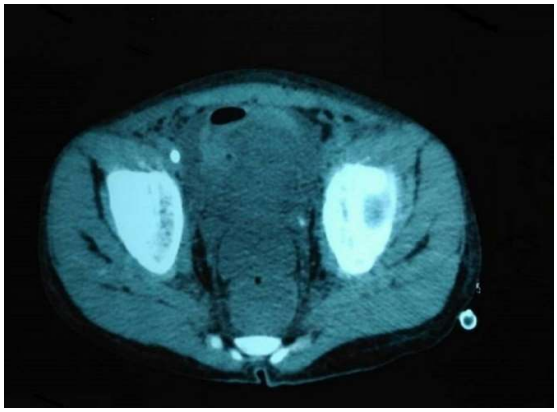

Figure-1: Bilateral enlarged kidneys

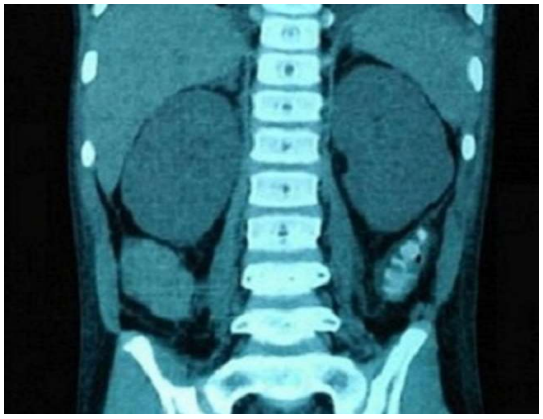

Figure-2: Bilateral enlarged kidneys

Discussion

The prevalence of AKI in children who need intensive care varies between 5% to over 80% depending upon the underlying causes and co morbidities [3,4]. More than 80% of hospitalized children are exposed to at least one nephro toxic medication during their hospital stay. Contrast induced acute kidney injury is a reversible form of AKI. It is defined as rise in serum creatinine of more than 0.5 mg/dL or a 25% increase from baseline value assessed at 48 hours after a radiological procedure [5]. Other possible causes of AKI should be ruled out before making the diagnosis of CIAKI. Our patient also diagnosed as CI AKI after ruling out other possible causes of AKI like sepsis, dehydration. He received 2 doses of aminoglycoside prior to admission which might be the additive factor for the development of AKI. The incidence of CI AKI in adult is 18% and is one of the major cause of hospital acquired kidney injury [5]. A study conducted by Ajami G et al showed the incidence of CI AKI as 18.75% in 80 children who had undergone cardiac angiography [6].

Serum creatinine usually peaks at 3 to 5 days after contrast administration and returns to baseline or near baseline within a couple of weeks. Common presentation of contrast induced AKI is non-oliguric renal failure and does not need renal replacement therapy. Since this boy presented with anuric AKI, he had been initiated on hemodialysis. Risk factors can be divided as patient related and procedure related. Pre-existing renal insufficiency, volume depletion like diarrhoea, vomiting, poor oral intake and long term nephrotoxic drug intake are patient related risk factors whereas procedure related factors include dose of contrast, osmolality as well as viscosity and route of administration. Iso/low osmololnon-ionic second and third generation contrast agents are preferred in view of lesser adverse effects [7]. Our child received second generation contrast agent for his study. The pathogenesis of contrast induced AKI is not completely understood.

The accepted couple of theories are focusing on renal tubular injury. Intravenous contrast ingestion causes renal vasoconstriction results in medullary hypoxia which is probably mediated by alterations in nitric oxide, endothelin, and adenosine. Second possible theory is that acute tubular injury is a direct cytotoxic effect of the contrast agents [8]. As the serum creatinine rises only after 48 hours of renal injury, other sensitive early biomarkers such as plasma neutrophil gelatinase-associated lipocalin (NGAL), plasma cystatin C, urinary interleukin 18 and urinary kidney injury molecule-1 (KIM-1) may be helpful to find out the injury earlier [9].

Non-pharmacological measures like maintaining good hydration and monitoring urine output are the mainstay of prevention of contrast induced AKI. Normal saline is

preferred rather than other intravenous fluids as it maintains good intravascular volume and prevents renin angiotensin axis activation [10,11]. It is ideal to maintain urine output of > 1 to 1.5ml/kg/hour 3 to 12 hours pre-contrast period and for 6 to 12 hours following contrast administration[5]. Always use lower possible dose of contrast medium. Avoid repetitive studies closely spaced within 48 to 72 hrs.

Pharmaco-logical prevention strategies are discontinuation of nephrotoxic drugs at least 24 to 48 hours prior to contrast administration. Sodium bicarbonate and antioxidant N-acetylcysteine are being tried though there is no clear evidence-based guidelines [12]. Always repeat the renal function test at 24 and 48-72hrs after the procedure to find out any alteration in renal function and to decide about management.

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