Bilateral ureteral obstruction from papillary necrosis secondary to household cleaner ingestion

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We report a case of a patient who developed bilateral hydroureteronephrosis from papillary necrosis secondary to ingestion of commercial toilet bowl cleaner. Eight days after her ingestion, acute renal failure prompted a renal ultrasound that showed bilateral hydroureteronephrosis. Emergent bilateral percutaneous nephrostomy tubes were placed and subsequent ureteroscopy revealed a large amount of obstructing necrotic material consistent with papillary necrosis. Ureteroscopic removal of the material and bilateral ureteral stents improved renal function. The etiology of this patient's papillary necrosis was likely due to a combination of hypovolemia, systemic acidosis from the ingestion, and direct toxicity of the substance on the renal vasculature. This case demonstrates the importance of early recognition of renal insults and the extra intestinal manifestations of toxic household ingestions.

Key Words: renal papillary necrosis, ureteral obstruction

Introduction

Poisoning from occupational, accidental and intentional exposure is a major developing world public health problem. However, deliberate self poisoning causes a great majority of deaths and puts an immense strain on hospital services. In 1990, Jeyaratnam estimated that self harm resulted in 2 million cases of poisoning each year with 200,000 deaths.[1] The renal effects of poisoning are well described and involve known nephrotoxic responses including interstitial nephritis, acute tubular necrosis and systemic acid base disturbances.[2] However, few reports have described renal papillary necrosis as a response to acute ingestion of toxic substances. To our knowledge, ingestion of a household cleaning substance leading to papillary necrosis, bilateral hydroureteronephrosis and subsequent renal failure has never been previously reported. We present the first case thereof.

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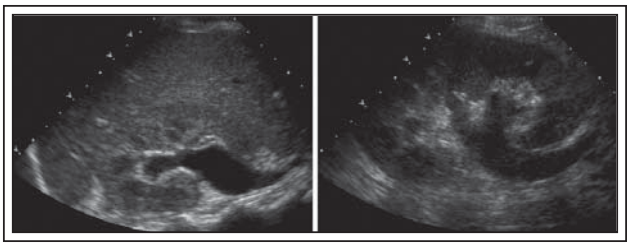

Figure 1. Renal ultrasound scans showing bilateral hydroureteronephrosis.

Case report

An 18-year-old female with a past medical history significant for severe depression presented to the emergency department conscious, alert and oriented after ingestion of commercial toilet bowl cleaner (The Works: Active ingredients - Hydrochloric acid 20%, Rodine 50.1%, Perfumes 0.1%) during a suicidal attempt. Shortly after admission, she was intubated and sedated for impending respiratory failure secondary to aspiration. She remained in the intensive care unit for 8 days. Hematuria, leukocyturia and low urine output were noted on day two, but creatinine levels remained stable. Given the multitude and complexity of her other problems, which included esophageal and gastric necrosis, hypoxemia secondary to airway edema, and respiratory failure secondary to chemical pneumonitis, these findings were not immediately worked up by the intensive care team. However, on admit day number six, her creatinine levels rose acutely from 1.1 to 4.4 and she became anuric. Ultrasound demonstrated severe bilateral hydroureteronephrosis, Figure 1. Percutaneous nephrostomy tubes were emergently placed and subsequent antegrade nephrostograms showed bilateral ureteral obstruction, Figure 2, of unknown etiology. Urine output from the nephrostomy tubes was brisk and her creatinine level improved rapidly.

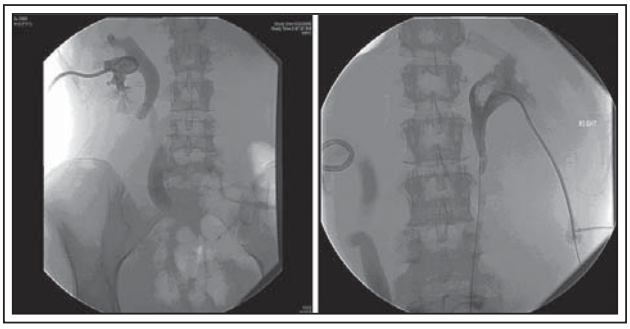

Figure 2. Antegrade nephrostograms showing bilateral ureteral obstruction.

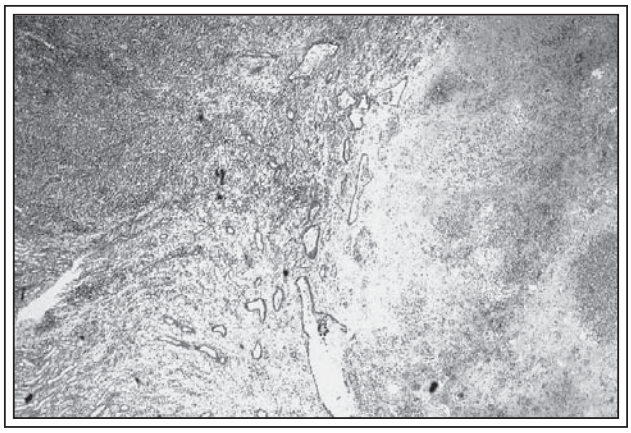

Figure 3. Low power light micrograph demonstrating sloughed renal tissue consistent with renal papillary necrosis.

Two days after percutaneous tubes were placed, an attempt was made to internalize the stents in an antegrade fashion but was unsuccessful secondary to large amounts of lucent obstruction of both ureters. Due to problems with nephrostomy tube drainage (likely from necrotic debris clogging the 10 F tubes), further attempts were made to place stents in a retrograde fashion. Ureteroscopic examination during stent placement revealed a large amount of necrotic material in both ureters. The material was removed ureteroscopically and stents were successfully placed. The remaining hospital course was unremarkable from a urologic standpoint and the stents were removed in 10 days after complete recovery of renal function. Pathologic examination of the material demonstrated necrosis of the entire papillae with demarcation and sequestration, consistent with papillary necrosis, Figure 3.

Comment

Renal papillary necrosis is a rare condition characterized by disruption of the renal papillae and consequently, decreased renal function. Usually, sloughed papillae will pass spontaneously through the ureter, but occasionally necrotic buildup may lead to acute ureteral obstruction.[3,4] The etiological factors causing papillary necrosis are well described and include diabetes mellitus, sickle cell anemia, pyelonephritis and analgesic abuse. There have been few cases reported of papillary necrosis requiring surgical intervention for complete ureteral obstruction. In Flaster et al's series, 5 of 56 patients with papillary necrosis developed significant ureteral obstruction requiring nephrostomy tube

insertion or ureteral catheter placement.[3] Additionally, analgesic nephropathy with both phenylbutazone and phenacetin abuse has been reported in cases of acute ureteral obstruction ultimately requiring surgical treatment.[4,5] To our knowledge, this is the first reported case of toxic ingestion of household material leading to papillary necrosis, bilateral hydroureteronephrosis and subsequent renal failure.

The pathogenesis of papillary necrosis is believed to be a sequela of ischemia occurring in the renal papillae and the medulla. The vasa recta supplying the papillae are thin walled and can easily become compressed from edema, inflammation and fibrosis secondary to diabetes mellitus or urinary obstruction.[6] Analgesics inhibit the vasodilatory effects of prostaglandin, thus predisposing the papillae to ischemia.[7] Additionally, renal papillary circulation can become sluggish secondary to increased blood viscosity as in sickle hemoglobinopathy.[6] These changes could sufficiently strangle the vascular supply to the medulla, causing ischemia and eventual necrosis.

Although the pathogenesis of papillary necrosis can be well delineated in some instances, the etiology of the majority of cases, including our own, is usually multifactorial.[6] Our patient's vomiting and subsequent dehydration status may have been the initial hypovolemic insult to her renal medulla. This state, coupled with considerable third spacing from gastrointestinal toxicity, could have led to a state of severely low intravascular volume and medullary ischemia. Additionally, our patient's systemic acidosis may also have contributed to the papillary and medullary insult as there have been reports of organ ischemia due to adverse metabolic consequences secondary to states of systemic acidosis.[8] However, these clinical situations alone rarely lead to papillary necrosis, especially in an otherwise healthy female. This is why we hypothesize that the direct effects the ingested toxic substance had on the kidneys was the most important contributing for its development.

Of the major constituents in the solution ingested by our patient, Rodine (50.1 wt%) is the substance known to be most toxic in humans. Rodine (*a.k.a.* Red Squill) is the liquid extract of the bulb of *Urginea maritima*, a perennial growing in the Mediterranean area. In addition to having cardiac glycoside properties, Rodine is a known rodenticide. In large doses it is a dangerous irritant poison, producing inflammation and vasculitis of the alimentary canal and urinary organs, and proving fatal in doses of only 24 grains of the powder.[9] The Rodine induced vasculitis likely contributed to the papillary necrosis by propagating the ischemic insults to the vasa recta.

This case demonstrates the importance of early recognition of the extra intestinal manifestations of toxic household ingestions. This patient's clinical scenario was presumptively attributed to the known sequelae of concentrated acidic ingestion; namely severe dehydration, aspiration pneumonitis and gastrointestinal hemorrhage. Consequently, her urologic pathophysiology was overlooked and there was a delay in the diagnosis of ureteral obstruction which was the ultimate cause of her renal failure.

Conclusion

Bilateral hydroureteronephrosis can be detected on ultrasound with high sensitivity and specificity. Although papillary necrosis secondary to toxic ingestion is rare, a careful evaluation of such patients is warranted with imaging, including renal ultrasound. Early recognition can be essential in these cases as acute papillary necrosis is usually fully recoverable if recognized in a timely fashion. In our case, early ultrasound could have prompted earlier intervention with ureteral stents and may have avoided transient consequences of renal failure as well as the percutaneous nephrostomy tube placement. □

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