CASE REPORT

Retention of iodinated contrast material within renal cysts in a patient with autosomal dominant polycystic kidney disease

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ABSTRACT. Hyperdense renal cysts, a common condition in autosomal dominant polycystic kidney disease, may be induced by haemorrhage into the cysts. However, hyperdense renal cysts resulting from retention of contrast material after intravenous injection is extremely uncommon because the intravenous administration of contrast material does not induce an increase in the attenuation of renal cysts. We report a case of retention of iodinated contrast material within renal cysts in a patient with autosomal dominant polycystic kidney disease.

Received 14 March 2011

Revised 7 June 2011

Accepted 9 June 2011

DOI: 10.1259/bjr/24588919

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Renal cysts are sharply margined round smooth homogeneous structures with attenuation values similar to that of water (–10 HU to +20 HU). They consist of a thin wall that is often imperceptible on a CT scan. No immediate or delayed increase in the attenuation of a renal cyst is observed after intravenous administration of contrast material [1, 2]. Hyperdense renal cysts are defined as having attenuation values higher than that of water on a CT scan and attenuation values higher than 20 HU; they are also a well-known occurrence in autosomal dominant polycystic kidney disease [1]. They are considered to be indeterminate cysts; thus, the differential diagnoses include benign entities, such as complicated cysts, as well as primary and secondary solid malignant neoplasms. To our knowledge, this is the first report in the English language literature of hyperdense renal cysts resulting from the retention of contrast material after an intravenous injection in a patient with autosomal dominant polycystic kidney disease.

Case report

A 38-year-old male with autosomal dominant polycystic kidney disease was referred for a CT evaluation owing to left flank pain. The patient underwent a three-phase CT scan using a routine kidney CT protocol consisting of pre-contrast, nephrographic and urographic phases. The pre-contrast CT scan showed opacification of the left renal pelvis because the patient had undergone an intravenous urography 3 days earlier. The pre-contrast scan revealed no significant interval change in the presence of innumerable renal cysts, including a few hyperdense cysts on both kidneys (Figure 1a), compared

with the kidney CT scan obtained 1 year earlier to evaluate autosomal dominant polycystic kidney disease. Post-contrast CT scans were obtained after the intravenous injection of 140 ml of the low-osmolality contrast agent ioxaglate (Hexabrix 320, Guerbet, Villepinte, France). Post-contrast CT scans revealed innumerable renal cysts and ureterolithiasis at the left ureteropelvic junction with hydronephrosis and delayed perfusion (Figure 1b). Follow-up pre-contrast CT scans obtained 14 days after the administration of contrast medium for CT showed persistent retention of contrast medium within innumerable renal cysts of the left kidney, appearing with an extreme increase in attenuation of the internal contents of renal cysts of the left kidney (Figure 1c); fewer than 50% of the cysts were involved. A plain radiograph obtained 21 days after the administration of contrast medium for CT still showed innumerable radio-opaque round lesions in the left renal shadow (Figure 1d). After 2 months, follow-up pre-contrast CT scans revealed the disappearance of left-sided urolithiasis and persistent retention of contrast medium within innumerable renal cysts on the left kidney, appearing with extremely increased attenuation of the internal contents of the renal cysts on the left kidney. Renal cysts that retained the contrast medium reached 400 HU, compared with CT scans obtained 1 year before when the hyperdense cysts in the left kidney were less than 70 HU. After 17 months, a follow-up pre-contrast CT scan showed persistent retention of contrast medium within renal cysts in the left kidney (Figure 1e).

Discussion

Simple renal cysts, the most commonly found mass in the adult kidney, are fluid-filled non-neoplastic lesions. Autosomal dominant polycystic kidney disease is a common inherited disorder in which the renal parenchyma is replaced by multiple cysts of varying size. Cysts with haemorrhage in patients with autosomal dominant

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Figure 1. A 38-year-old male with autosomal dominant polycystic kidney disease. (a) Pre-contrast scan of the kidney hilar level shows diffuse enlargement of both kidneys with innumerable renal cysts, including a few hyperdense cysts within both kidneys, affected by autosomal dominant polycystic kidney disease. (b) The coronal plane of a post-contrast CT scan shows ureterolithiasis at the left ureteropelvic junction (arrow) with hydronephrosis and delayed perfusion. (c) After 14 days, the follow-up pre-contrast CT scan shows persistent retention of contrast medium within an innumerable number of renal cysts on the left kidney, with extremely increased attenuation of the internal contents of renal cysts of the left kidney (attenuation reached 400 HU). (d) Plain radiograph obtained 21 days after the CT scan shows new evidence of multiple radio-opaque round lesions in the left renal shadow. (e) After 17 months, a follow-up pre-contrast CT scan shows persistent retention of contrast medium within renal cysts of the left kidney.

polycystic kidney disease present as hyperdense on CT, which is a common CT finding among these patients [3]. In autosomal dominant polycystic kidney disease, 70% of patients exhibit one or more hyperdense cysts (58–84 HU) [1]. Several mechanisms exist by which masses may become hyperdense. Cysts may have an elevated CT attenuation because of haemorrhage with clot retraction, concentration of the protein components of blood, elevation of iron content, colloid formation, infection or secondary to transient iodine accumulation within a simple cyst [1]. However, intravenous injection of contrast medium does not directly induce the development of hyperdense cysts [2] because renal cysts are not communicated to the nephron or renal tubular structures. Although they may originate in a few nephrons and collecting ducts, in autosomal dominant polycystic

kidney disease, cysts originate as mural diverticula of nephrons and collecting ducts. In the early stages of cyst development, the enlarging cyst communicates with a proximal and distal tubular segment. As each cyst enlarges, bending and twisting forces ultimately break the connection with the parent tubule, resulting in isolated cysts [4]. Our case involved persistent hyperdensity of renal cysts by the retention of contrast medium, the mechanism of which was unclear. However, a possible mechanism of this inflow of contrast medium into the cyst is reflux of contrast medium from tubules into the cyst through retained communication between the cyst and tubules by high pressure from ureteral obstruction by urolithiasis. This may explain why a proportion of cysts in the present case (<50%) were opacified by contrast medium. Although inflow and accumulation

of contrast medium after intravenous injection within multiple renal cysts are extremely uncommon conditions, obstructive uropathy can induce this phenomenon.

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