Simple Renal Cysts: Are they Really Benign?

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Benign renal neoplasms constitute a large and heterogeneous group of lesions which include the simple renal cysts (SRCs), selected complex renal cysts, cortical and metanephric angiomyolipoma, oncocytoma, the rarer cystic nephroma, mixed epithelial/stromal tumor and leiomyoma, as well as other, even more esoteric tumor types. SRCs are discrete lesions within the kidney that are typically cortical, extending outside the parenchyma and distorting the renal contour. They are oval or circular in shape and have a distinct, sharply defined outline. On an average, simple cysts measure about 0.5 to 1.0 cm in diameter, but 3 to 4 cm cysts are not uncommon. The cyst wall is characteristically smooth, transparent, avascular, yellowish or bluish white in color, and formed by a thin layer of fibrous tissue lined by a single layer of flattened or cuboidal epithelia. They are filled with a homogeneous transudate like, clear or straw-colored fluid of low viscosity. SRCs may be solitary or multiple and bilateral. SRCs remain the most common of benign renal lesions, representing more than 70% of asymptomatic renal masses. Rarely, however, they can be associated with rupture (hemorrhage), hematuria, abdominal or back pain due to bleeding, palpable abdominal mass, evidence of infection, and/or hypertension. Clinical symptoms are more common with neoplasms than with simple cysts.¹ The Bosniak classification for renal cystic lesions, as reviewed² ³ in Table 1, is the most useful and widely employed method for characterizing renal cystic lesions and for assessing the likelihood of the presence of a concomitant malignancy within the cyst. Imaging modalities such as ultrasound and magnetic resonance imaging (MRI), CT scan (with and without contrast enhancement) are frequently used in the evaluation of renal masses, and decide its intervention.

SRCs usually originate from the distal convoluted tubule or collecting ducts and may arise from renal tubular diverticula. Focal tubular obstruction and renal parenchymal ischemia have been suggested as possible pathogenic mechanism. The prevalence of SRCs depends on the population studied, age and imaging modality used for diagnosis. Risk factors for the development of sporadic renal cysts are increasing age,¹ smoking,⁴ male gender,¹ presence of hypertension¹ and presence of renal insufficiency. Hypertension has also been discussed as a consequence of renal cysts.⁶

Karoli et al⁷ now report in this journal the association between SRCs and hypertension. In their study a total of 6230 patients were included and SRCs were present in greater numbers in patients with hypertension and author found significant association between hypertension and SRCs. They concluded that routinely close

<table>
<thead>
<tr>
<th>Class</th>
<th>Imaging characteristics</th>
<th>Incidence of malignancy</th>
<th>Therapy</th>
</tr>
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<tbody>
<tr>
<td>I</td>
<td>Simple cyst with a hairline thin wall that does not contain septa, calcifications, or solid components. It measures water density in Hounsfield units and does not enhance with intravenous administration of a contrast agent.</td>
<td>1.7%</td>
<td>No therapy or follow-up required.</td>
</tr>
<tr>
<td>II</td>
<td>Cyst may contain few hairline thin septa and fine calcification, or short segments of slightly thickened calcification may be present in the wall or septa. Uniformly high attenuation lesions &lt;3 cm (so called high density cysts) are well arginated and don’t enhance with IV administration of a contrast agent.</td>
<td>18.5%</td>
<td>No therapy or follow-up required.</td>
</tr>
<tr>
<td>IIIF</td>
<td>Cyst may contain multiple hairline thin septa or minimal smooth thickening of their wall or septa contain calcifications that may be thick and nodular but no measurable contrast enhancement is present. Totally intrarenal nonenhancing high attenuation renal lesions ≥3 cm are also included in this category.</td>
<td>18.5%</td>
<td>Repeat imaging to assess stability of size and radiographic characteristics</td>
</tr>
<tr>
<td>III</td>
<td>“Indeterminate” cystic masses have thickened irregular or smooth walls or septa in which measurable contrast enhancement is present.</td>
<td>33%</td>
<td>Excision or ablation</td>
</tr>
<tr>
<td>IV</td>
<td>Clearly malignant cystic masses can have all the criteria of category III but also contain enhancing soft tissue components.</td>
<td>92.5%</td>
<td>Excision or ablation</td>
</tr>
</tbody>
</table>

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monitoring of blood pressure among subjects with SRCs is warranted and the presence of SRCs should not be overlooked.

The mechanism by which the presence of SRCs is related to hypertension is still not clear. Some authors consider that perihilar cysts cause increased renin release and stimulates RAAS (renin–angiotensin–aldosterone system). \(^8,9\)

The other explanation why SRCs was related to hypertension might be that the loss of nephrons along with aging was involved in both development of hypertension and formation of peripheral renal cyst. The evidence that simple renal cysts are an important cause of hypertension remains controversial. Several authors reported that simple renal cyst was related to high blood pressure (BP) in cross-sectional studies\(^{10,11}\) and the reduction in BP after removal of large cysts, was attributed to the inactivation of the RAAS. Recently, Al-Saïd et al\(^{12}\) reported that the presence of renal cysts, even a single cyst, was associated with reduced renal function in hospitalized patients younger than 60 years but not with hypertension.

The chance of renal cyst to activate the RAAS by compressing renal artery may be related to location as well as the size and the number of cysts. In present study by Karoli et al\(^7\) results showed that SRCs ≥2 in number or ≥2 cm in size were important determinants of hypertension. Author could not measure plasma renin activity or serum renin levels to support the hypothesis of role of increased renin levels in patients with SRCs who had pre-hypertension or hypertension. This is one of the limitations of the study.

Karoli et al\(^7\) did not examine the effect of removal of large cysts on hypertension. A study done by Terada et al\(^{13}\) reported that hypertension may induce kidney dysfunction, leading to renal cyst formation. Some authors have reported\(^{14,15}\) about normalization of blood pressure after renal cystectomy and percutaneous cyst decompensation, indicating that simple renal cyst may be a preceding condition to hypertension.

Simple renal cysts did not gain much attention in the past. Although the study by Karoli et al\(^7\) found a certain relationship between SRCs and hypertension, but this may not be enough to explain the accurate order of relation. Further studies with long term follow-up and large sample size are needed to address this issue in future.

References