Laparoscopic management of renal cystic disease

Abstract Renal cystic disease, ranging from simple cysts to autosomal dominant polycystic kidney disease (ADPKD), can lead to significant complications such as pain, hypertension, infection, upper urinary tract obstruction, and even renal failure. For patients with preserved renal function, laparoscopic ablation of renal cysts is a recent, though safe and effective alternative to open surgery in patients who have failed conservative measures. Likewise, for symptomatic patients with renal failure, laparoscopic nephrectomy offers a less invasive option to open nephrectomy. Both laparoscopic cyst decortication and laparoscopic nephrectomy offer patients the benefits of minimal intraoperative blood loss, minimal postoperative pain, brief hospital stay, and rapid convalescence while offering a short-term outcome equivalent to open surgery. Further study and follow-up are indicated to assess the long-term effect of these procedures on the outcome of ADPKD with regard to durability of pain relief, preservation of renal function, and impact on renal hypertension.

Renal cystic disease of various etiologies can lead to significant morbidity such as pain, infection, renal obstruction, and even renal failure. At one end of this spectrum are simple renal cysts which are usually incidentally found and remain asymptomatic for the majority of patients. In sharp contrast, other cystic diseases are progressive and associated with a myriad of other systemic effects. One such disease is autosomal dominant polycystic kidney disease (ADPKD), which represents the most common form of inherited renal cystic disease, affecting approximately 1 in 400–1000 individuals in the United States [15,16,26]. It is the fourth leading cause of chronic renal failure worldwide, accounting for approximately 10% of all patients on chronic renal dialysis [15,16]. Despite being referred to as adult onset, the disease can be identified in the pediatric age group by radiographic imaging and has even been documented in-utero by prenatal ultrasound [28,34].

The etiology for simple cyst development in the majority of patients is essentially unknown. However, cyst development in ADPKD has been attributed to genetic mutations in three PDK genes. The most common form is due to a mutation in the PKD-1 gene located on the short arm of chromosome 16 and accounts for 85% cases. Mutations in PKD-2, on chromosome 4, comprise the majority of the remaining 15%, with rare cases attributed to mutations of PKD-3, whose location is unknown [17,18,27]. Defects in these genes lead to deregulation of normal tubular development, resulting in tubular epithelial hyperplasia and cyst development [19]. The sporadic nature of cyst development in ADPKD suggests that a 2-hit mechanism is required for cyst growth. It is possible that a somatic mutation in one PKD allele is later joined by an acquired mutation in the normal allele, thus leading to cystogenesis [17–19,27]. Cysts can originate from renal tubules anywhere along the nephron. As the affected epithelial cells proliferate, the tubule bulges out like a diverticulum, eventually losing its connection to the rest of the nephron. Transepithelial transport of solutes and fluid into the cyst is then required for it to further expand [37]. Cysts also secrete an unidentified secretagogue that has been demonstrated to stimulate cyst fluid secretion via activation of cyclic AMP, potentially explaining the progressive increasing cyst size [37].
Clinical presentation

For the majority of patients, simple cysts are found incidentally on imaging studies during investigation for other problems. The incidence of benign simple cysts increases with age, with roughly 50% of patients having them after age 50 [16, 26]. In ADPKD, patients may be asymptomatic until the third or fourth decades of life although the different genetic forms vary in presentation. Mutations in PKD-1 lead to a more severe form of cystic disease that usually presents earlier than defects in PKD-2 [18]. The most common symptoms from expanding renal cysts are pain in the abdomen or flank region, hematuria, and gastrointestinal complaints, such as nausea and early satiety [15,16,26]. In ADPKD, hypertension is the most common presenting sign and plays a major role in the progression to renal failure and patient mortality [6]. The pathogenesis of hypertension in renal cystic disease results from stimulation of the renin-angiotensin-aldosterone system by vascular compression that occurs from cyst development [1,6]. Overall, urologic complications have been reported in 40% of patients with ADPKD; this includes calculus, infection, hemorrhage, and upper tract compression [36].

Management

For patients suffering from the complications of renal cystic disease, such as severe pain, hemorrhage, recurrent infections, and hypertension refractory to medical control, invasive management may be required in the form of cyst aspiration, marsupialization, or, in the patient with renal failure, nephrectomy.

In the case of infected renal cysts, intravenous antibiotic therapy often fails due to poor penetration through the cyst wall. As such, percutaneous cyst aspiration and laparoscopic cyst decortication and drainage have both been reported as successful treatment modalities in patients with simple and ADPKD renal cysts [7,20]. In the latter condition, infected cysts may be difficult to identify based on ultrasound or CT scan, due to the large number and heterogeneous appearance of the renal cysts. The laparoscopic approach, either through a retroperitoneal or transperitoneal approach, has the advantage of accessing a greater number of cysts.

For patients suffering from chronic pain, refractory hypertension and renal obstruction, the minimally invasive approach via percutaneous needle aspiration, sclerotherapy, or percutaneous cyst obliteration, has been used as well to decompress and/or resect suspected symptomatic renal cysts. While these methods are effective in patients with a single simple symptomatic cyst (i.e., 60–90% success rate), the results in ADPKD patients are variable at best [23,31,35]. Simple needle cyst aspiration invariably leads to reaccumulation of cyst fluid and recurrence of symptoms. As a result, sclerosing agents (e.g., 95% ethanol, tetracycline based solutions, and bismuth sulfate) have been introduced to prevent recurrence. Success rates utilizing ethanol sclerotherapy have been reported to be approximately 90% [31]. Due to the progressive nature of ADPKD, this usually provides only transient relief, with recurrence of pain usually within 3–6 months [31]. Likewise the morbidity of the procedure is related to the number of cysts aspirated. Thus, for patients with ADPKD, percutaneous cyst aspiration, sclerotherapy, or resection is an impractical approach with poor long-term results.

Open surgical cyst decompression was introduced in 1911 by Rosving, but later abandoned in the 1960s after being associated with postoperative worsening of renal function [4,31]. Closer evaluation revealed that these procedures were complicated by infection and often performed in the face of preoperative renal insufficiency. In 1980, Shangzhi and associates reported prolonged pain relief (90% at 6 months and 77% at 5 years) after open cyst marsupialization in 52 patients; no impairment of renal function was noted from the procedure itself [32]. Subsequent reports of surgical cyst decompression have been shown to be beneficial in ADPKD patients with chronic abdominal pain refractory to oral analgesics. Elzinga and colleagues noted pain relief in 80% of 26 previous narcotic-dependent ADPKD patients at 1 year and in 62% at 2 years [14]. In 1997, Ye and co-authors reported pain relief at 1 and 5 years in 92% and 81% of 260 ADPKD patients, respectively, who underwent open surgical cyst decompression [38]. The data available support the hypothesis that a more durable response may result if a more aggressive decortication is performed. This procedure has also been associated with a reduction in hypertension and stabilization of renal function [32,38].

Open cyst marsupialization clearly provides better pain relief than percutaneous aspiration. Bennett and associates reported an 81% pain free rate at 18 months after open cyst decortication compared to 33% pain free rate after percutaneous aspiration [2]. However, the complication rate of the open approach is not surprisingly higher. Kropp and associates reported a 33% perioperative complication rate, including wound infection, prolonged immobilization, urinary retention, venous thrombosis, and pulmonary complications in 126 ADPKD patients who underwent open surgical cyst decompression [24]. In addition, a 37% complication rate has also been reported, which included urine leakage, infection, bleeding, wound hernia, and a bowel obstruction [2].

Laparoscopic cyst decortication/marsupialization

The recent introduction of laparoscopy into urology has altered the approach to renal cystic disease by providing a less morbid option of management that potentially simulates an open procedure. Laparoscopic decortication of simple renal cysts was first reported by Hulbert and colleagues in 1992 and later applied to patients with