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Review Article

Xanthogranulomatous pyelonephritis: a review

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ABSTRACT

Xanthogranulomatous pyelonephritis (XP), first described in 1916, is a rare form of chronic granulomatous inflammation. The etiology is still unclear; however, the development of the disease is associated with chronic urinary obstruction secondary to lithiasis, tumors and urological malformations, among others. This leads to the destruction of the renal parenchyma and its replacement by solid sheets of lipid-laden macrophages. Female gender, diabetes and obesity are attributed as predisposing factors to the development of XP. It is estimated that the incidence presents a maximum peak between 50 and 70 years, with a ratio of 2:1 women-men respectively. Computed tomography (CT) is described as the mainstay in the evaluation. However, the definitive diagnosis is made by histopathological study, where a mixture of lipid-laden foamy macrophages, lymphocytes, neutrophils, giant cells, and plasma cells can be seen. Nephrectomy (open or laparoscopic) continues to be the first-line treatment. The laparoscopic approach is associated with an increase in operating time; however, the recovery time is shorter compared to the open approach. Given the natural history of the disease and the associated complications, this makes it a challenging approach for the surgeon. Therefore, a surgeon experienced in laparoscopic skills is necessary. This review seeks to synthesize existing information regarding the appropriate surgical approach in conjunction with the clinical context.

Keywords: XP, Nephrectomy, Minimally invasive surgery, Inflammation, Laparoscopic

INTRODUCTION

Xanthogranulomatous pyelonephritis (XP) first described in 1916, is a rare form of chronic granulomatous inflammation, which causes damage to renal parenchyma and adjacent tissues. It manifests pathologically with the appearance of xanthomatous cells; They are macrophages with a large granular and eosinophilic cytoplasm. It is known to mimic almost any other inflammatory kidney disease. Most cases are unilateral, where there may be infection, obstructive renal stones, and granulomatous inflammation, resulting in hydronephrotic kidney with diffuse parenchymal destruction.[1,2] Etiology is still unclear, however the development of disease is associated with chronic urinary obstruction by stones (where approximately 30-50% is secondary to staghorn stones), tumors, urological malformations, renal ischemia, ineffective treatment of infections urinary tract, alterations in lipid metabolism, alterations in immune

response, malnutrition, lymphatic obstruction, venous occlusion and arterial failure; this leads to destruction of renal parenchyma and its replacement by solid sheets of lipid-laden macrophages.[3-5]

EPIDEMIOLOGY

It is estimated that the incidence has a maximum peak between 50 and 70 years, with a ratio of 2:1 woman, men respectively, being predominant in Caucasians. Children and adolescents under 17 years of age account for about 10% of all patients and there is no gender preference in this age group.[6] It has an incidence of 1.4 cases per 100,000 inhabitants per year. In a systematic review conducted by Harley et al they identified 40 studies including 1,139 patients with XP where 18 deaths were reported, with a weighted pooled perioperative mortality rate of 1,436 per 100,000 patients.[2] In adults, it is more prevalent in the sixth decade of life, in females, with the right kidney being the most affected. In pediatric patients there is a higher prevalence in males, from 60% to 75% of cases occur in children under 6 years of age, and the left kidney is the most frequently affected.[5]

RISK FACTOR

Female gender, diabetes and obesity are attributed as predisposing factors to the development of XP.[7] In a study carried out by Avilés-Ibarra et al. In a Mexican population where type 2 diabetes is a prevalent pathology, they found said disease as a demographic characteristic in 31.9%, considering type 2 diabetes as a fundamental factor in the development of XP, although with inconclusive results, since it is necessary to have larger series to establish a probable significant association. It was reported that in more than 50% of the positive urine cultures an extended-spectrum beta-lactamase-producing bacterium developed.[8] In some case reports, *Escherichia coli* and *Proteus mirabilis* have been isolated from approximately 90% of positive urine cultures from patients with XP.[9] Harley et al conducted a systematic review, where they reported that 69% of patients diagnosed with XP had upper urinary tract lithiasis, of which 48% were staghorn stones.[2]

PATHOPHYSIOLOGY

The pathophysiology of XP is not completely accurate. The combination of obstruction and infection are the primary inducers, triggering interstitial pyelonephritis, followed by a chronic granulomatous immune response.[10] A common obstructive pathway results in stone formation, secondary to superinfection of the urine. Suggesting a limited/incomplete immune response of the host, and a chronic inflammatory response due to failure of complete degradation of bacterial products. Since there is a presence of bacteria within the granulomas, both intracellular and extracellular, the hypothesis that granuloma formation is mainly induced by bacteria is supported.[11,12] In the affected renal unit, destruction of the renal parenchyma secondary to chronic inflammation will be evidenced microscopically, which conditions the presence of xanthoma cells (foamy macrophages with the presence of lipids). Some research has suggested that abnormal lipid metabolism might play a role in the development of XP; however, there is no experimental evidence behind this theory.[13,14] Nephrolithiasis, more frequently associated with staghorn stones, is not a prerequisite; however, it has been described in different series as a well-established predisposition for XP. Predisposing factors include urinary tract infection, especially with *P. mirabilis* and *E. coli*, urinary malformations such as ureteral duplication, ureteropelvic junction obstruction, vesicoureteral reflux, chronic interstitial nephritis, type 2 diabetes, metabolic syndrome, and immunocompromised. A relationship has also been described with renal cell carcinoma, squamous cell carcinoma, and transitional cell carcinoma of the renal pelvis.[13]

MOLECULAR BASES AND GENETIC ANALYSIS

In a study conducted by Bartoli et al showed that the expression of tissue MCP-1 was higher in patients diagnosed with XP than in controls, and hospitalized patients with ureteropelvic junction obstruction without complications. The local release of chemokines, such as MCP-1, may play an important role in the development of tubulointerstitial lesions mediated by the recruitment of circulating monocytes. This is also confirmed by the finding that MCP-1 expression was directly correlated with the extent of monocyte infiltrates.[15] Monocyte chemoattractant protein-1 (MCP-1/CCL2) is a member of the C-C chemokine family and a potent chemotactic factor for monocytes. MCP-1 is believed to be identical to JE, a gene whose expression is induced in mouse fibroblasts by platelet-derived growth factor. The human homologue that has been best characterized as CCL2 was first purified from human cell lines on the basis of its monocyte chemoattractant properties. CCL2 is the first discovered human CC chemokine. Located on chromosome 17 (chr.17, q11.2), human MCP-1 consists of 76 amino acids and is 13 kDa in size.[16]

CLASSIFICATION

In 1978, Malek and Elder proposed a histopathological staging (Table 1) made up of three stages, depending on their extension.[1,17] However, currently the most widely used classification considers the diffuse or focal presentation of XP. The diffuse form, which represents the most frequent form; it affects both renal poles, characterized by increased renal volume, hydronephrosis, replacement of the cortico-medullary junction by xanthochromic content, and is frequently associated with nephrolithiasis. The focal form, which represents less than 15%, has a pseudotumoral presentation, confined to a renal segment or pole, being more frequent in the lower pole. The differential diagnosis includes renal tumors and

infectious processes (such as tuberculosis and renal abscess).[5]

Table 1: Malek and elder staging according to extension.

Stages	Level of affectation	Clinic	Treatment
Stage I	Exclusive involvement of the renal parenchyma.	Focal	Partial nephrectomy / nephron salvage
Stage II	Renal involvement and perirenal fat.	Diffuse	Radical nephrectomy
Stage III	Renal, perirenal and pararenal involvement.	Extra-renal	Radical nephrectomy + management of abscesses and extrarenal fistulas

Table 1 classification stages according to Malek and Elder correlated with their presentation and treatment.

DIAGNOSIS

XP is an atypical clinical entity associated with late identification. It has been reported that up to 42% of patients present with a prolonged clinical picture, up to 6 months. 12 The clinical manifestations are non-specific; related to renal lithiasis and recurrent urinary tract infections, such as lower back and abdominal pain. Weight loss, fever and palpable mass have also been reported, in 29.8%, 43.9%, 24.6% respectively.[18] Sometimes the only symptoms are the manifestations of its extrarenal complications, these include the liver, spleen, diaphragm, pleural space, chest wall, abdominal wall, buttocks, and skin. These late, nonspecific signs and symptoms have the potential to divert clinical attention away from possible XGP until further studies are obtained.[17] In a study conducted in 2023 by Sanyaolu et al. It was evidenced that 13.4% of the patients with XP presented urosepsis, and 22.7% reported recurrent urinary tract infection.[19] In immunocompromised patients, XPG should be taken into account as a diagnostic possibility when there is an atypical clinical picture and poor response to antimicrobial therapy with imaging studies and doubtful interpretation, since they may have a high rate of morbidity and mortality.[20]

The diagnostic process prior to the histopathology study is complicated because the clinical and radiological findings of XP are nonspecific, as well as its radiological similarity with tuberculosis, pyelonephritis, perirenal abscess, and malignant tumors.[14] Laboratory studies are nonspecific, being common to find hematological alterations. In a retrospective study carried out by Pais et al they included 57 patients diagnosed with XP from 2005 to 2019. Laboratory studies at diagnosis were analyzed, reporting alterations in blood counts, such as anemia, leukocytosis, neutrophilia, lymphocytosis, in a 78.9%, 43.6%, 41.50% and 13.20% respectively. General urinalysis showed red blood cells, leukocytes, and bacteria. The cultures showed a predominance of *E. coli* and *P. mirabilis*. Alterations in liver function tests have been observed in a minimal percentage of patients diagnosed with XP, which normalize after treatment.[18] Radiological findings are usually non-specific, however, some associated radiological characteristics have been highlighted, suggestive of XP, including renal hyperplasia, urinary tract stones, and urinary tract obstruction.[14] As an initial study, abdominal radiography is a cheap and rapid study. Radiopaque stones can be identified, as well as an initial evaluation of the widened renal contour and enhancement of the ipsilateral psoas margin that is present in advanced disease.[10] Visualization of a perirenal fluid collection in association with diffuse or segmental renal abnormalities by ultrasound suggests XGP. Other findings are thinning of the renal parenchyma, hydronephrosis, and ultrasonographic signs of obstructive uropathy. Ultrasound demonstration of a renal sinus stone or calcified nodule distant from a renal mass may help differentiate XP from a calcified renal tumor such as renal cell carcinoma.[21]

CT is described as the mainstay in the evaluation of XP, where dilated renal calyces, changes in renal size and shape, as well as the presence of stones in the urinary tract and associated complications can be observed (Figure 1). The bear paw sign is a characteristic imaging feature, but not pathognomonic; describes the appearance of multiple rounded regions of low attenuation (-10 to +30 Hounsfield Units) radiating into the renal cortex and centering on a contracted renal pelvis. It represents the replacement of renal parenchyma by necrotic areas or xanthomatous collections, where each finger pad represents necrotic areas. Although this appearance mimics hydronephrosis, hypoattenuation represents infiltrating inflammation rather than calyceal distension in most cases. In the presence of extrarenal extension, it allows defining if it is limited to the perirenal and pararenal spaces and if it infiltrates adjacent organs and tissues.[12,22] If a dimercaptosuccinic acid (DMSA) scintigraphy is performed, areas of cortical scarring will be seen as photopenic foci. If there is any doubt about the diagnosis, follow-up DMSA can be performed, where we can visualize persistent photopenic foci, confirming the presence of established scars.[12] Since the PET/CT findings are not specific; It is not used routinely due to limited availability, high cost, and possible overlap with malignancy.[23] On magnetic resonance imaging (MRI), the loculated component corresponding to cavities and abscesses are usually hypointense on T1 and hyperintense on T2. On T1, hyperintensity is observed in the solid component of XP, however, authors have reported isointensity. This is probably secondary to the dependence on the number of xanthogranulomatous cells involved in the granulomatous process, for the intensity

of the solid component on T1, the findings on T2 are isointensity compared to the contralateral renal parenchyma. Enhancement of the thickened perirenal fascia has been described, reflecting the level of inflammation; This finding is highly relevant for dictating the surgical plan.[24] Definitive diagnosis is made pathologically, where a mixture of lipid-laden foamy macrophages, lymphocytes, neutrophils, giant cells, and plasma cells can be visualized. Varying degrees of renal tubular atrophy, cholesterol crystals, tubular dilation, fibrosis, focal squamous metaplasia of the urothelium, microabscesses, spindle cell proliferation, and lymphoid aggregates with germinal center formation may be seen.[13,14] The differential diagnosis of XP includes renal carcinoma, abscesses, tuberculosis, lymphoma, angiolipoma, leiomyosarcoma, interstitial megalocytic nephritis, malacoplakia, or Wilms tumor. XP can be differentiated from renal cell carcinoma (RCC) histologically, by the presence of granulomatous cavities, and by immunohistochemical analysis. XP is usually positive for CD68, lysozyme, and vimentin, while CRC is positive for CD10 and epithelial membrane antigen.[17]

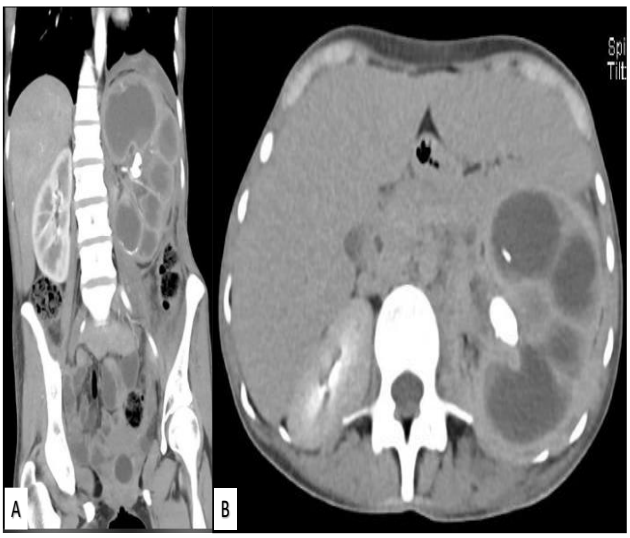

Figure 1 (A and B): XP. Coronal section of CT. Left kidney with increased dimensions, altered morphology and loss of sinus-parenchyma relationship. Parenchyma with homogeneous enhancement of the cortex. In the renal pelvis, an image is observed that adopts its morphology, hyperdense, corresponding to renal lithiasis. Axial section of CT, where data of XP can be observed in the left kidney, as well as the presence of a stone in the renal pelvis.

TREATMENT

The treatment is controversial, cases have been reported in which antibiotic treatment without surgery is the first choice, particularly in pediatric patients. However, nephrectomy (open or laparoscopic) remains the first-line treatment. The choice of the type of nephrectomy will depend on the extent of the lesion; whether focal or diffuse. Total nephrectomy is indicated in the diffuse variety, while in the focal variety partial nephrectomy with preservation of nephrons is chosen. The surgical procedure is a therapeutic challenge since in most cases there will be the presence of adhesions secondary to perirenal inflammation.[12] Currently, the open approach has been replaced by the laparoscopic one, since the first successful laparoscopic nephrectomy was performed in 1991 by Clayman et al. performed a retrospective study where they report their experience with laparoscopic and retroperitoneoscopic nephrectomy for XP. reporting an operating time of 193.6 minutes (range 123-340). Estimated bleeding of 223.5 ml (range 30-1000). Median hospital stays 4.8 days (range 3-12). Transfusion rate 29.6%. Major complications were reported in 11.1%.[25] Kapoor et al carried out a retrospective analysis, where they compared transoperative and postoperative data from the open vs. laparoscopic approach, with the result that the mean hospital stay for the laparoscopic group was 3.8 days, compared with 8.2 days for the open group. The average operating time in the laparoscopic group was 3.8 hours compared to 2.5 hours in the open group. The incidence of major complications was 20% and 10% in the open and laparoscopic groups, respectively. Concluding that the laparoscopic approach is associated with an increase in operating time, however, the recovery time is shorter compared to the open approach. Barboza et al carried out a comparative study of the open and minimally invasive approach. In the group treated by the open approach, greater blood loss, an increase in the number of transfusions, longer hospitalization time, as well as a greater risk of requiring intensive care were reported. The minimally invasive approach showed less blood loss, fewer transfusions, shorter hospital stay, and less risk of requiring intensive care compared to the open approach.[26]

Regarding the preferred type of open approach, and in accordance with the results obtained from a study carried out by Robles-Torres et al it was found that the transperitoneal open approach in the midline offers a larger work space, as well as a clearer vision. of kidney anatomy. For its part, the open retroperitoneal approach offers the advantage of direct access to the renal artery and reduces the risk of vascular complications. Therefore, according to this study, the open transperitoneal approach is the best option when the laparoscopic approach is not feasible or cannot be carried out by experienced surgeons.[27] In a retrospective analysis carried out by Francesco Chiancone et al which included 67 patients with a histopathological diagnosis of XP, it was concluded that open nephrectomy is indicated in cases in which XP is associated with urinary lithiasis, while the laparoscopic approach it is preferred in cases with previously established stenosis. These findings corroborate that severe renal inflammation is more frequently associated with older urinary stones. Unfortunately, the possibility of a transoperative complication is not ruled out, thus, no independent factors for postoperative complications were found.

Danilovic et al demonstrated that a higher score from the American Society of Anesthesiology, septic shock, a kidney size greater than 12 cm, and a renal abscess were associated with a Clavien-Dindo score >1.[28] Coinciding with other studies that confirm that the use of minimally invasive surgery as an approach to XP plays an important role. [29] Laparoscopic nephrectomy is a feasible procedure, however, given the natural history of the disease and associated complications, it makes it a challenging approach for the surgeon. Therefore, a surgeon experienced in laparoscopic skills is necessary.[30]

In a review by Gravestock et al in which 52 studies were included, it was observed that only 3 reported antimicrobial management; in which intravenous schemes with Piperacillin/Tazobactam were used together with Ceftriaxone, ampicillin/gentamicin and cefazolin were the most used drugs. Regarding the oral route of administration, ciprofloxacin, nitrofurantoin and trimethoprim with sulfamethoxazole were used. Fosfomycin with clotrimazole and ceftriaxone with piperacillin/tazobactam demonstrated moderate levels of antimicrobial resistance 16.6% and 14.3% respectively. No evidence of antimicrobial resistance against carbapenems in this study. Concluding that a specific antibiotic therapy is not necessary for the treatment of this entity.[14]

COMPLICATIONS

Complications are described in the literature, from 11.1% to 92%, this difference can be attributed to the prevalence of comorbidities in different populations. Surgical wound infections, skin fistulas, retroperitoneal collections, septic shock, and incisional hernia stand out. Avilés-Ibarra OJ et al carried out a retrospective analysis in Mexico, where clinical records of 72 patients with a diagnosis of XP were taken into account, in which a considerable percentage of major complications required admission to the ICU. The most common admission criteria were; single organ failure and septic shock. This suggests that delaying surgical intervention until clinical parameters are optimized (anemia, resolution of urinary tract infection, renal function), as well as performing a preoperative evaluation by the ICU, may be feasible behaviors to improve prognosis.[8]

DISCUSSION

XP is a rare entity. In recent years, its diagnosis has increased, probably due to increased knowledge of the pathology, and advances in imaging studies. Since the first report by Malek et al observed a percentage of 0.6% of XP among patients with chronic pyelonephritis, to date, where several cohorts, in which kidneys of patients who underwent simple nephrectomy for diagnosis of inflammatory kidney disease, stone disease, and atrophic kidneys reported XP in 12%, 10.8%, and 9.4% of cases, respectively.[31] The most frequent comorbidities reported in the series are type 2 diabetes, chronic kidney disease, and hypertension. Gauhar et al performed a retrospective study where a total of 365 patients met the inclusion criteria for the analysis. There were 228 (62.5%) women. The mean age was 45±14.4 years. Chronic kidney disease (CKD) was the most common comorbidity (71%), followed by hypertension (29%) and diabetes mellitus (28.5%). Almost 23% of the patients had a history of recurrent urinary tract infections. Renal colic was the most frequent presenting symptom (39.7% of patients). Fever was present in 142 (38.9%) patients, while sepsis and septic shock were present in 49 (13.4%) and 24 (6.6%) patients, respectively. Bladder urine culture was positive in 185 (53.2%) patients, while kidney urine culture was positive in 118 (81.9%) cases.[1] One of the main diagnostic challenges of XP is the non-specificity of the symptoms, which leads to long periods of time before the definitive diagnosis. Country et al reported a mean time to presumptive diagnosis of 365.1 days, which tells us about the lack of knowledge about XP and reinforces the need to improve the dissemination of diagnostic criteria.[18] Imaging studies are essential for diagnosis; Despite the fact that there are no specific data of the XP, it will be possible to classify, observe the extension, and laterality, data that are pillars for the surgical plan. The XP study should include a CT of the abdomen. The classic radiologic presentation is the “bear claw sign,” or kidney enlargement with several discrete hypodense concentric cavities enhancing the border. Focal XGP is characterized by partial involvement of the kidney by hypodense cyst-like masses. There may be thickening of Gerota's fascia but without perirenal involvement. Diffuse XGP, on the other hand, is characterized by multiple hypodense areas with thinning of the renal parenchyma, inconspicuous excretion of contrast material (indicating poor renal function), and perirenal fat enhancement. [17]

Weber et al. when mentioning nuclear magnetic resonance as the diagnostic method of choice, since the contrast medium (gadolinium) is less nephrotoxic than the contrast medium used in CT; however, a potential complication of gadolinium is nephrogenic systemic fibrosis.[6] The treatment controversy lies in partial versus total nephrectomy, and the laparoscopic approach versus the classical open method. Asali et al. performed an analysis of the laparoscopic approach to PGX. Laparoscopic nephrectomy was successful in 96% of patients, conversion to open surgery was required, mean operating time was 193.6 minutes (range 123-340). The estimated mean blood loss was 223.5 mL (range 30-1000). The mean hospital stay was 4.8 days (range 3-12). The transfusion rate was 29.6%. Serum creatinine was 1.3 mg/dl the day before and the day after the operation. Major complications occurred in 11.1%.[25] Antibiotics are an adjunct to the treatment of XP. The choice of antibiotic depends on the organism and its susceptibility to the antimicrobials reported in the urine culture. In a study carried out by Artiles-Medina et al they included 26 case series, reporting 693 total cases of XP; the most frequently prescribed antibiotics for treatment were

piperacillin/tazobactam and ceftriaxone. The reported resistance rates to piperacillin/tazobactam and ceftriaxone were 14.3% and 16.6%, respectively.[32] The prevalence of complications varies according to the literature, population, diagnostic approach, and surgical experience. In a study carried out by Avilés-Ibarra et al the overall prevalence of complications, both intraoperative and postoperative, was 92%. According to the Clavien-Dindo classification, the incidence of minor and major complications was 53% and 35%, respectively. The most frequent complication was anemia that required blood transfusion (40%), followed by fever (13%), organic dysfunction (10%), septic shock (6%), surgical site abscess (6%), retroperitoneal hematoma (4%) septic shock with multiple organ failure (6%) retroperitoneal hematoma (4%) acute myocardial infarction (4%) pneumothorax (1%) and enterocutaneous fistula (1%). Concluding that Malek's stage (Malek II-III) and elevated creatinine values (>2.5mg/dL) increase the risk of developing major complications in patients with XP undergoing nephrectomy, by 40.8% and 58.4% respectively.[8] However, Pais et al reported surgical or postoperative complications in 13 (22.8%) patients, which included 3 surgical wound infections, 3 cutaneous fistulas, 3 retroperitoneal collections, 2 septic shock, 2 splenic lesions that required splenectomy, 1 incisional hernia, 1 duodenal perforation, and 1 necrotizing pancreatitis. [18]

CONCLUSION

It is important to take into account the diagnosis of XP, when we are faced with a non-specific clinical picture, characterized by urinary symptoms, a history of pyelonephritis that does not respond to medical treatment, urinary malformations, renal lithiasis, or obstruction data, considering the main differential diagnoses. how neoplasms, renal inflammatory diseases how malacoplakia, interstitial nephritis, infections, renal abscesses and tuberculosis. Imaging studies are fundamental in the diagnosis, since they will give us important data for the decision of surgical treatment, being able to assess the location and extension of the disease, crucial to define the type of surgery. Treatment includes antibiotics and abscess drainage prior to nephrectomy; with the aim of reducing the risk of trans and post-surgical complications. The type of nephrectomy and the decision to perform open or laparoscopic nephrectomy will depend on the extent of the disease, initially assessed with imaging studies and the experience of the center where it is performed.

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