Scientific Research Publishing

Open Journal of Nephrology, 2025, 15(2), 219-227
https://www.scirp.org/journal/ojneph
ISSN Online: 2164-2869
ISSN Print: 2164-2842

Retroperitoneal Fibrosis Secondary to Tuberculosis and Sigmoid Colon Cancer: Two Case Reports Presenting with Acute Obstructive Renal Failure and a Review of the Literature

Fall Serigne[1], Mbengue Mansour[1], Sall Idrissa[1], Sow Ibrahima[1], Diouf Mohamed[1], Tshabayembi Jatt[1], Ouahbi Nada[1], Mbodj Ismaïla[2], Sow Ahmadou[2], Diallo Abass[2], Ndiaye Modou[3], Niakhaleen Keita[1], Niang Abdou[1]

[1]Department of Nephrology, National Hospital of Dalal Jamm, Dakar, Senegal
[2]Department of Radiology and Medical Imaging, National Hospital of Dalal Jamm, Dakar, Senegal
[3]Department of Urology, National Hospital of Dalal Jamm, Dakar, Senegal
Email: fallserigne498@gmail.com

How to cite this paper: Serigne, F., Mansour, M., Idrissa, S., Ibrahima, S., Mohamed, D., Jatt, T., Nada, O., Ismaïla, M., Ahmadou, S., Abass, D., Modou, N., Keita, N. and Abdou, N. (2025) Retroperitoneal Fibrosis Secondary to Tuberculosis and Sigmoid Colon Cancer: Two Case Reports Presenting with Acute Obstructive Renal Failure and a Review of the Literature. *Open Journal of Nephrology*, **15**, 219-227.
https://doi.org/10.4236/ojneph.2025.152022

Received: April 28, 2025
Accepted: June 7, 2025
Published: June 10, 2025

Abstract

Retroperitoneal fibrosis is a rare condition, with an estimated incidence between 1 in 200,000 and 1 in 500,000 per year, characterized by the proliferation of fibrous tissue in the retroperitoneal space, which can compress the ureters and cause acute obstructive renal failure. It may be idiopathic or secondary to various causes, including infections such as tuberculosis or malignancies such as sigmoid colon cancer. We report two distinct cases: a 42-year-old woman with tuberculous retroperitoneal fibrosis presenting with low back pain and anuria, successfully treated with corticosteroids and antituberculosis therapy; and a 25-year-old man with post-surgical retroperitoneal fibrosis secondary to sigmoid adenocarcinoma, who progressed to chronic renal failure despite oncologic management. A review of the literature indicates that these secondary forms, although rare, involve inflammatory (tuberculosis) or desmoplastic (cancer) mechanisms. Diagnosis is based on imaging (ultrasound, CT scan, magnetic resonance imaging), and treatment is guided by the underlying etiology. These cases highlight the importance of a multidisciplinary approach to determine the cause and optimize outcomes.

Keywords

Retroperitoneal Fibrosis, Cancer, Tuberculosis, Acute Renal Failure,

Corticosteroid Therapy

1. Introduction

Retroperitoneal fibrosis (RPF) is a rare pathology, with an estimated overall incidence between 1 in 200,000 and 1 in 500,000 per year in the general population [1]. It is characterized by the proliferation of fibrous tissue in the retroperitoneal space, leading to compression of adjacent structures, notably the ureters, with serious functional consequences such as obstructive renal failure due to bilateral ureterohydronephrosis. It may be idiopathic (known as Ormond's disease), or secondary to various etiologies, including infections such as tuberculosis or malignancies such as sigmoid colon cancer.

Tuberculosis-induced RPF is believed to result from chronic granulomatous inflammation in the retroperitoneum, where Mycobacterium tuberculosis triggers immune activation and fibroblast stimulation through cytokines such as TNF-α and TGF-β. In cancer-associated RPF, tumor cells may induce desmoplastic reactions by releasing profibrotic mediators like TGF-β and PDGF, leading to fibroblast proliferation and collagen deposition.

The diagnosis of RPF is often challenging because of its nonspecific clinical presentation and its radiological similarity to other retroperitoneal pathologies such as lymphoma, abscesses, or retroperitoneal sarcoma. Delays in diagnosis are common, and histological confirmation is not always feasible due to the proximity of the fibrotic tissue to major vascular structures.

We report two cases of retroperitoneal fibrosis secondary to tuberculosis and adenocarcinoma of the sigmoid colon, followed by a detailed review of the literature.

2. Observation

2.1. Case 1

A 42-year-old female patient was admitted to our department with a two-week history of right-sided lower back pain associated with symptoms suggestive of tuberculosis. The patient had no prior urological or nephrological history. The initial clinical examination revealed anuria lasting more than 72 hours, a pulmonary condensation syndrome, and a well-tolerated anemic syndrome.

Laboratory tests showed impaired renal function, with a creatinine level of 120.83 mg/L, blood urea of 1.14 g/L, sodium at 136 mmol/L, hyperkalemia (6.1 mmol/L), chloride at 108 mmol/L, hypocalcemia (81.8 mmol/L), and phosphatemia at 75.4 mmol/L. A nonspecific inflammatory syndrome was present, with C-reactive protein at 161.3 mg/L, negative procalcitonin, microcytic hypochromic anemia (7.7 g/dL), thrombocytosis (640 G/µL), and lymphopenia (1600/µL).

A contrast-enhanced CT scan showed bilateral ureteropelvic dilatation upstream of a retroperitoneal tissue mass, extending to the iliac bifurcation and in

close contact with the ureters, aorta, and non-displaced inferior vena cava (Figure 1). Magnetic resonance imaging was not performed. Cystoscopy showed a normal bladder and visible ureteral orifices.

The tissue mass was not biopsied due to its proximity to major vessels. The diagnosis of obstructive renal failure secondary to retroperitoneal fibrosis was retained. Etiological investigations revealed a positive tuberculin skin test (16 mm), aseptic leukocyturia, and positive antinuclear antibodies at a titer of 1:320 with a mottled pattern. Autoimmune causes were deemed unlikely: anti-ENA antibodies were negative, as were IgG4 antibodies. A chest X-ray revealed a bilateral diffuse interstitial syndrome. Tuberculous RPF was ultimately diagnosed.

The patient underwent ureteral stenting and was treated with corticosteroids at 1 mg/kg/day and a standard antituberculosis regimen including rifampicin, isoniazid, pyrazinamide, and ethambutol. The outcome was favorable, with progressive normalization of renal function (creatinine levels returned to normal after 2 months).

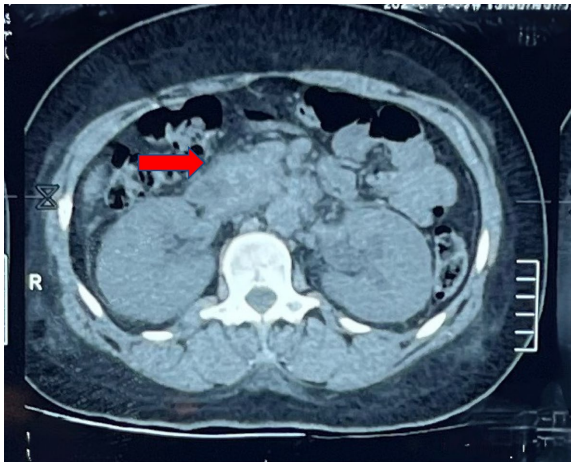

Figure 1. Axial contrast-enhanced abdominal CT showing a central retroperitoneal tissue mass displacing surrounding structures.

2.2. Case 2

A 25-year-old male patient with a history of well-differentiated grade 1 sigmoid colon adenocarcinoma (pT3N1aM0), treated with chemotherapy (oxaliplatin, capecitabine, and calcium folinate), was admitted to our department three years after sigmoidectomy, presenting with anuria, renal edema, late postprandial vomiting, and diffuse abdominal pain, predominantly in the left flank.

Laboratory findings showed impaired renal function: blood urea 3.23 g/L, creatinine 370 mg/L, sodium 129.4 mmol/L, potassium 8.29 mmol/L, chloride 97.9 mmol/L, and calcium 85.4 mmol/L. A nonspecific inflammatory syndrome was present, with C-reactive protein at 17 mg/L, negative procalcitonin, normochromic microcytic anemia (12.3 g/dL), lymphopenia (1400/µL), and a normal platelet count.

Abdominal ultrasound showed normal-sized kidneys with good corticomedullary differentiation, but bilateral grade III ureterohydronephrosis. Contrast-enhanced CT revealed a retroperitoneal tissue mass located below the aortic bifurcation at the L5 level, invading the right ureter and contacting the left ureter and right iliac arteries (Figure 2 and Figure 3). MRI showed a retroperitoneal mass, posterior to the mesentery, intensely and progressively enhancing, with irregular contours, retractile, and enveloping the right lumbar ureter (measuring 30 × 25 mm), consistent with retroperitoneal fibrosis. Cystoscopy showed a permeable left ureteral orifice but not the right one. A biopsy was not performed due to the mass's proximity to the iliac arteries.

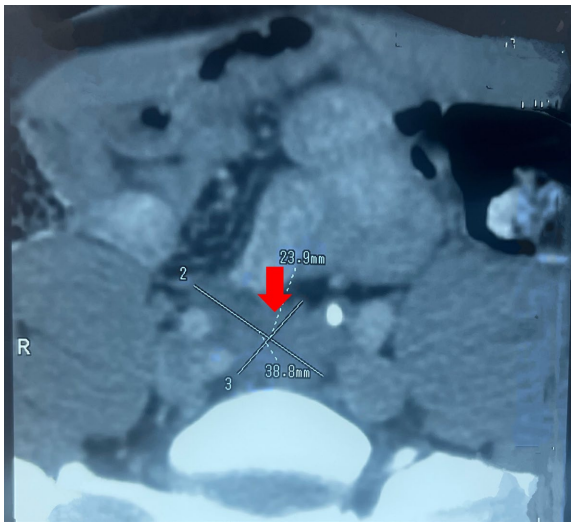

Figure 2. Axial abdominal CT (L4 level) showing a retroperitoneal mass, below the aortic bifurcation, posterior to the mesentery, with irregular contours.

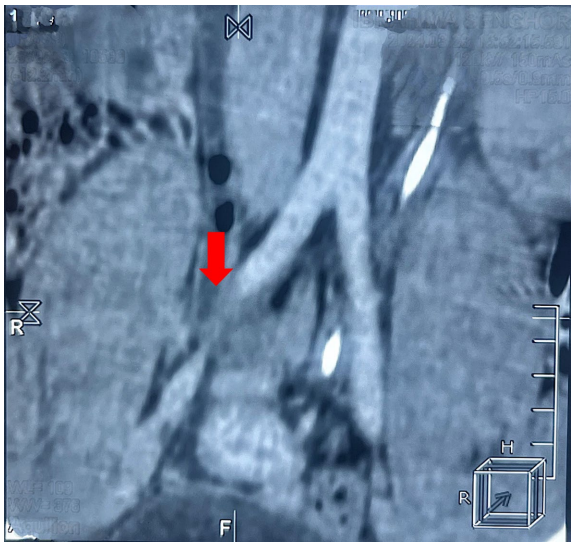

Figure 3. Coronal CT image showing ureteral encasement by retroperitoneal fibrosis.

A diagnosis of obstructive renal failure due to retroperitoneal fibrosis was retained. There was no history of radiotherapy or systemic inflammatory disease. Immunological tests were negative, the tuberculin skin test was anergic, and IgG4 levels were not measured. Retroperitoneal fibrosis secondary to sigmoid colon cancer was considered the most likely diagnosis.

The outcome was unfavorable despite attempted urinary diversion, with irreversible loss of right kidney function and progression to end-stage chronic renal failure.

3. Discussion

3.1. Prevalence

RPF is a rare disease, with an estimated incidence between 1 in 200,000 and 1 in 500,000 per year in the general population [1]. Secondary forms account for approximately 30% - 40% of cases and may include infections and malignancies. Extrapulmonary tuberculosis (15% - 20% of global TB cases [2]) is an exceptional but likely underdiagnosed cause of RPF, especially in endemic regions such as Africa and India, due to limited access to advanced imaging [3]. A Moroccan series of 11 cases of RPF (2005-2017) identified two related to tuberculosis (18% of secondary forms) [4]. Malignancies account for 8% - 10% of secondary RPFs [5], with colorectal cancer implicated in 1% - 2% of cases [6]. Sigmoid colon cancer (20% - 25% of colorectal cancers [7]) may cause RPF via direct extension or metastatic spread, though exact prevalence remains uncertain due to the lack of systematic histological confirmation [8].

3.2. Mécanisme Etiopathogénique

The primary mechanism behind tuberculosis-related RPF is chronic granulomatous inflammation induced by Mycobacterium tuberculosis. Abdominal tuberculosis, often lymphatic or peritoneal, can spread locally to the retroperitoneum and lead to extensive fibrosis. The characteristic granulomatous inflammation involves the release of pro-inflammatory cytokines (TNF-α, IL-1, and TGF-β), which activate fibroblasts and stimulate collagen synthesis [9].

In contrast, sigmoid colon cancer-associated RPF arises from tumor invasion or a desmoplastic reaction. Cancer cells may infiltrate the retroperitoneum via direct serosal extension or lymphatic spread, leading to fibroblast proliferation mediated by tumor-derived factors like TGF-β, PDGF, and VEGF [10] [11]. This results in dense fibrotic stroma. Retroperitoneal metastases can also simulate idiopathic RPF and be difficult to differentiate radiologically without histology [8]. Recent molecular studies have highlighted the role of matrix metalloproteinases (MMPs) and integrin signaling in facilitating fibrosis in malignancy, contributing to tissue remodeling and immune evasion [11].

3.3. Clinical, Biological, Imaging and Histological Manifestations

Table 1 details the characteristics of tubercular and sigmoid colon cancer-related RPFs.

Table 1. Comparative features of retroperitoneal fibrosis secondary to tuberculosis and sigmoid colon cancer.

Aspect	Tuberculous RPF	RPF in sigmoid colon cancer
Clinical symptoms	Low back pain (60% - 80% of FRPs [1]), moderate fever, night sweats, weight loss, hydronephrosis (flank pain, anuria) [3] [12].	Localized pelvic pain, altered bowel movements (constipation/subocclusion), weight loss, fatigue, hydronephrosis (30% - 50% of cases [6]) [7].
Biology	Elevated CRP (>80% of cases [1]), accelerated VS, chronic normocytic anemia, positive tuberculosis tests (IDRT, IGRA), elevated creatinine if obstruction [3] [12].	Moderate CRP, elevated CEA (60% - 70% of advanced stages [7]), hypochromic microcytic anemia (bleeding), hypoalbuminemia (metastases), elevated creatinine if obstruction [13].
Imaging	• Ultrasound: Hypoechoic mass, bilateral hydronephrosis • CT: Asymmetrical periaortic fibrous plaque, possible calcifications (60% - 70% [5]) • MRI: Hypersignal T2 (active inflammation), hyposignal T1/T2 (fibrosis) [1].	• Ultrasound: Hydronephrosis, normal kidneys. • CT: Heterogeneous mass under aortic bifurcation, irregular contrast [5]. • MRI: Heterogeneous T2 hypersignal (necrosis), adjacent infiltration [14].
Histology	an inflammatory granulomatous reaction typical of *Mycobacterium tuberculosis* infection. Biopsies reveal caseous granulomas with central necrosis, surrounded by lymphocytes, macrophages and multinucleated Langhans-type giant cells [9] [15]. Fibrous tissue is dense, composed of abundant collagen and fibroblasts, reflecting fibrosis secondary to chronic inflammation [1]. Microcalcifications may be present in granulomatous areas, and Ziehl-Neelsen staining can identify acid-fast bacilli (AFB), although their detection is inconsistent due to the low bacterial load in extrapulmonary forms [3].	malignant component infiltrating abundant fibrous stroma. Biopsies show adenocarcinomatous cells typical of colorectal cancer, with irregular glands, nuclear atypism and a marked desmoplastic reaction (tumor cell-induced proliferation of fibroblasts and collagen) [10] [11]. Lymph node metastases or tumour deposits in the retroperitoneum may be observed, confirming the malignant origin [13]. One case series noted that malignant RPF can be difficult to differentiate from benign fibrosis without immunostaining (e.g. positivity for cytokeratin in tumor cells) [6].

Imaging and histology play a fundamental role in the diagnosis of retroperitoneal fibrosis. Imaging, in particular CT and MRI, not only allows us to visualize the location and extent of fibrosis, but also to differentiate between benign and malignant forms. MRI, in particular, offers a better characterization of the soft tissues and can point to inflammatory activity or tumor infiltration. Histology, although sometimes difficult to perform because of its proximity to major vascular structures, remains the method of choice for etiological confirmation, particularly for distinguishing a granulomatous inflammatory reaction from neoplastic involvement. It is therefore of major diagnostic interest when it can be obtained in complete safety.

The decision not to perform biopsies in both patients was based on the high risk of complications, as the masses were closely adherent to the aorta and iliac arteries. Moreover, the clinical context, radiological appearance, and biological findings were sufficiently suggestive to guide management without histological proof. This conservative approach is supported in the literature when biopsy is

unsafe or non-contributory [5].

Differential diagnoses were carefully considered in both cases. Alternative possibilities such as Erdheim-Chester disease, IgG4-related disease, retroperitoneal sarcomas, and chronic infections including actinomycosis or histoplasmosis were evaluated. However, the clinical presentation, imaging findings, and biological context were not consistent with these entities, and specific features such as bone involvement (Erdheim-Chester), elevated serum IgG4, or necrotic abscess-like features were absent.

3.4. Traitement

Successful treatment of RPF depends on accurate identification of the underlying cause.

In tuberculous RPF, the treatment is dual: eradicating the infection and managing the mechanical complications. The standard anti-tuberculosis regimen includes an initial 2-month intensive phase with isoniazid, rifampicin, pyrazinamide, and ethambutol, followed by a continuation phase of isoniazid and rifampicin for an additional 4 - 7 months (total duration 6 - 9 months, as per WHO recommendations) [2]. This regimen targets the infection, although the fibrotic component may persist [3].

Adjunctive corticosteroids (e.g., prednisone 0.5 - 1 mg/kg/day) are often prescribed for 1 - 3 months in tapering doses to limit inflammation and fibrosis [1] [16]. In cases of obstructive uropathy, urinary diversion via double-J stenting or percutaneous nephrostomy may be necessary. One study reported a clinical response in 80% of patients with tuberculous RPF on this regimen, though renal sequelae may persist [4]. Surgical ureterolysis is reserved for refractory cases where obstruction does not resolve with medical therapy [5].

In cancer-associated RPF, treatment is primarily oncological, focused on tumor control. Localized sigmoid colon cancer is treated with surgical resection (sigmoidectomy) and adjuvant chemotherapy (e.g., FOLFOX: 5-FU, leucovorin, oxaliplatin) [7]. In metastatic or locally advanced stages, systemic chemotherapy and targeted therapies (e.g., bevacizumab, anti-VEGF) may help reduce tumor burden and desmoplastic fibrosis [11]. Immunosuppressive agents and corticosteroids, although effective in idiopathic RPF, are contraindicated here due to the risk of promoting tumor progression [1].

Obstructive complications may require urinary drainage, but ureterolysis is rarely curative in malignant RPF because of the infiltrative nature of the fibrosis [6]. Prognosis is poor, with median survival ranging from 6 to 12 months in advanced malignant RPF [13]. A palliative approach may be considered in end-stage disease.

Long-term follow-up is essential in both etiologies. It includes regular imaging (CT or MRI) to monitor fibrosis progression or recurrence, renal function assessment, and clinical surveillance for relapse or oncologic progression. Multidisciplinary collaboration (nephrology, infectious disease, oncology, urology, radiology)

is crucial for optimizing management and outcomes.

4. Conclusions

RPF secondary to tuberculosis and sigmoid colon cancer is a rare but clinically significant condition. Accurate diagnosis requires a high index of suspicion and a multimodal diagnostic approach, especially in patients with nonspecific symptoms and known risk factors. The etiopathogenic mechanisms involve chronic inflammation in tuberculosis and tumor-induced desmoplastic fibrosis in malignancy.

Our two cases illustrate the diagnostic complexity of secondary RPF and highlight the importance of considering infectious and neoplastic differentials, especially in endemic or oncologic contexts. Treatment must be etiology-specific, combining anti-infectious, oncologic, and supportive urological strategies.

Further research into the molecular pathways of fibrosis may lead to targeted antifibrotic therapies in the future. In the meantime, early recognition, individualized management, and long-term follow-up remain key to improving patient outcomes.

Informed Consent

Informed consent was obtained from both patients for the publication of their medical data.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Vaglio, A., Salvarani, C. and Buzio, C. (2006) Retroperitoneal Fibrosis. *The Lancet*, **367**, 241-251. https://doi.org/10.1016/s0140-6736(06)68035-5

[2] WHO (2022) Global Tuberculosis Report. https://www.who.int/teams/global-programme-on-tuberculosis-and-lung-health/tb-reports/global-tuberculosis-report-2022

[3] Sharma, S.K. and Mohan, A. (2004) Extrapulmonary Tuberculosis. *Indian Journal of Medical Research*, **120**, 316-353.

[4] Maghraoui, A., Ammouri, W., Bouiga, N., Bourkia, M., Khibri, H., Mouatassim, N., *et al.* (2018) Profil clinique, thérapeutique et évolutif des fibroses rétropéritonéales de l'adulte. *La Revue de Médecine Interne*, **39**, A147-A148. https://doi.org/10.1016/j.revmed.2018.10.093

[5] Jakhlal, N., Elghazoui, A., Jabbour, Y., Karmouni, T., Elkhader, K., Koutani, A., *et al.* (2017) Fibrose rétropéritonéale: Revue de littérature. *Canadian Urological Association Journal*, **11**, 26-31. https://doi.org/10.5489/cuaj.4122

[6] van Bommel, E.F.H., Jansen, I., Hendriksz, T.R. and Aarnoudse, A.L.H.J. (2009) Idiopathic Retroperitoneal Fibrosis. *Medicine*, **88**, 193-201. https://doi.org/10.1097/md.0b013e3181afc420

[7] Siegel, R.L., Miller, K.D. and Jemal, A. (2020) Cancer Statistics, 2020. *CA*: *A Cancer Journal for Clinicians*, **70**, 7-30. https://doi.org/10.3322/caac.21590

[8] Mitchinson, M.J. (1970) The Pathology of Idiopathic Retroperitoneal Fibrosis. *Journal of Clinical Pathology*, **23**, 681-689. https://doi.org/10.1136/jcp.23.8.681

[9] Wynn, T.A. and Ramalingam, T.R. (2012) Mechanisms of Fibrosis: Therapeutic Translation for Fibrotic Disease. *Nature Medicine*, **18**, 1028-1040. https://doi.org/10.1038/nm.2807

[10] De Wever, O. and Mareel, M. (2003) Role of Tissue Stroma in Cancer Cell Invasion. *The Journal of Pathology*, **200**, 429-447. https://doi.org/10.1002/path.1398

[11] Levental, K.R., Yu, H., Kass, L., Lakins, J.N., Egeblad, M., Erler, J.T., *et al.* (2009) Matrix Crosslinking Forces Tumor Progression by Enhancing Integrin Signaling. *Cell*, **139**, 891-906. https://doi.org/10.1016/j.cell.2009.10.027

[12] El Mansouri, Y., Benchekroun, A., Nouini, Y., *et al.* (2002) Fibrose rétropéritonéale et tuberculose ganglionnaire abdominale. Discussion étiopathogénique. *Annales d'Urologie*, **36**, 325-328.

[13] Zhou, L., Liu, S., Han, M., *et al.* (2019) Retroperitoneal Fibrosis Associated with Malignancy: A Case Report and Review of the Literature. *Journal of International Medical Research*, **47**, 3998-4004.

[14] Treglia, G., Mattoli, M.V., Leccisotti, L., *et al.* (2014) Usefulness of 18F-FDG PET/CT in Retroperitoneal Fibrosis: A Case Series and Literature Review. *Clinical Nuclear Medicine*, **39**, e383-e388.

[15] Golden, M.P. and Vikram, H.R. (2005) Extrapulmonary Tuberculosis: An Overview. *American Family Physician*, **72**, 1761-1768.

[16] Vaglio, A., Palmisano, A., Alberici, F., Maggiore, U., Ferretti, S., Cobelli, R., *et al.* (2011) Prednisone versus Tamoxifen in Patients with Idiopathic Retroperitoneal Fibrosis: An Open-Label Randomised Controlled Trial. *The Lancet*, **378**, 338-346. https://doi.org/10.1016/s0140-6736(11)60934-3