ORIGINAL ARTICLE

Renal Disease in Cystic Fibrosis: A Study at a Tertiary Care Center in Saudi Arabia.

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ABSTRACT

Background

Cystic fibrosis (CF) is a multisystem autosomal recessive disorder with increasing life expectancy, leading to the emergence of non-pulmonary complications, including renal involvement. Although renal disease has been reported in approximately 5.1% of CF patients globally, evidence from Arab populations remains scarce.

Materials and Methods

A retrospective observational study with an embedded case series was conducted at a tertiary care center in Saudi Arabia. Medical records of 430 patients diagnosed with CF between 1984 and 2018 were reviewed. Clinical and laboratory data were collected across four follow-up time points, from initial presentation to the most recent clinic visit. The primary outcomes assessed included the presence and types of renal complications, such as electrolyte disturbances, acute kidney injury, and chronic kidney disease.

Results

Out of 430 CF patients, three cases of renal disease were identified. All affected patients exhibited proteinuria, hematuria, and characteristic ultrasound findings, including increased renal echogenicity and loss of corticomedullary differentiation. Electrolyte abnormalities were also observed in all cases. Additionally, these patients demonstrated pulmonary involvement, including bronchiectasis and interstitial lung changes. A shared CFTR mutation (c.1418delG, exon 11) was identified in at least one allele in all three patients.

Conclusion

Renal involvement, although uncommon, represents a clinically relevant complication in patients with CF. Early and routine renal monitoring is essential for timely detection and management, which may help prevent progression to chronic kidney disease and reduce the need for renal replacement therapies.

Keyword

Cystic fibrosis, renal, proteinuria, chronic kidney disease

INTRODUCTION

Cystic fibrosis (CF) is a chronic, progressive disorder that affects multiple organ systems, including the respiratory, gastrointestinal, urogenital systems, as well as the sweat glands **[1,2]**. It is inherited as an autosomal recessive condition caused by mutations in the cystic fibrosis transmembrane conductance regulator (CFTR) gene **[1]**. CF represents the most common life-limiting autosomal recessive disease among Caucasian populations, with an estimated incidence of 1 in 2,000 to 3,000 live births **[2]**. The ΔF508 mutation on chromosome 7 is the most frequently identified variant in North America and Western Europe **[3]**. However, in Saudi Arabia, different mutations such as 1548delG and I1234V are reported to be more predominant **[4–8]**.

Over the past two decades, advances in the management of CF have led to a marked improvement in patient survival **[2]**. However, the increasing longevity of this population has been accompanied by the emergence of

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previously underrecognized complications, thereby shifting clinical focus from predominantly pulmonary care to the management of complex, multisystem chronic sequelae. Renal involvement has gained attention as one such complication. The cystic fibrosis transmembrane conductance regulator (CFTR) protein is expressed in the kidneys, particularly within the proximal and distal tubular epithelium, and its dysfunction has been associated with abnormalities such as low-molecular-weight proteinuria. Despite this, the precise pathophysiological role of CFTR in the development of CF-related renal disease remains incompletely understood. While primary renal pathology is relatively uncommon in CF, secondary renal impairment is being increasingly reported, likely reflecting the cumulative effects of chronic disease burden and therapeutic exposures **[9,10]**.

Individuals with CF are increasingly recognized to be vulnerable to both acute kidney injury (AKI) and chronic kidney disease (CKD). This susceptibility is multifactorial, with a significant contribution from repeated or prolonged exposure to nephrotoxic medications, including aminoglycoside antibiotics, non-steroidal anti-inflammatory drugs (NSAIDs), and immunosuppressive agents. In addition to drug-related toxicity, intrinsic disease mechanisms may also play a role, such as alterations in renal salt handling associated with CFTR dysfunction. Furthermore, chronic colonization with *Pseudomonas aeruginosa* and the development of cystic fibrosis-related diabetes (CFRD), often necessitating insulin therapy, may further exacerbate renal impairment through inflammatory, metabolic, and vascular pathways **[11–13]**.

Clinically significant kidney involvement in CF is rare but has been documented by several studies **[14–19]**. A study by Wilcock et al., which examined 1,532 CF patients, found a 5.1% prevalence of renal disease. Of the 80 renal disease episodes identified, the most common issue was renal stones (38% of episodes), followed by acute kidney injury (AKI) in 21%, CKD in 16%, isolated hematuria in 10%, isolated proteinuria in 2.5%, and nephrotic syndrome in 2.5%. The prevalence of renal stones in adult CF patients (2%) was comparable to that of the general population, with drug-induced AKI being the second most common renal complication **[14]**.

A separate study analyzed 24-hour urine samples from 63 patients, revealing hyperuricosuria in 16 patients and hypocitraturia in 14. Elevated calcium oxalate (CaOx) saturation was found in 26 patients, with hyperoxaluria present in 19. Increased calcium phosphate ($CaHPO_4$) saturation and uric acid (UA) saturation were noted in 19 and 11 patients, respectively. Urolithiasis was diagnosed in one patient, while nephrocalcinosis was identified in four patients **[15]**.

This study aimed to report cases of renal disease in cystic fibrosis (CF) patients at a tertiary care center, contributing to a deeper understanding of renal involvement in CF through data from a specialized care setting. Additionally, it compared the spectrum of renal diseases observed in these patients with previously published literature **[14–19]**. This comparison is intended to identify trends and guide clinical management. Ultimately, the study sought to enhance the knowledge of renal health in CF patients.

METHODOLOGY

This study employed a retrospective observational design involving a cohort of 430 patients diagnosed with CF. Within this population, a nested retrospective case series was undertaken to provide a detailed description of three patients who developed renal complications. Medical records spanning a period of 34 years (1984-2018) were systematically reviewed. All cases of CF patients who exhibited evidence of renal involvement during this period were identified and included. The study was conducted at a tertiary care center in Saudi Arabia, which serves as a referral center for complex CF cases.

Eligibility for inclusion was based on established diagnostic criteria for cystic fibrosis. Patients were included if they fulfilled at least one of the following: (1) presence of characteristic pulmonary and/or gastrointestinal manifestations, or a positive family history of CF, in conjunction with a sweat chloride level exceeding 60 mmol/L; (2) identification of pathogenic CFTR mutations on both alleles; or (3) presence of typical clinical manifestations with borderline or normal sweat chloride levels (30–60 mmol/L), supported by confirmed CFTR gene mutations. This comprehensive approach ensured inclusion of both classical and atypical CF phenotypes.

Renal involvement was evaluated through a combination of biochemical, clinical, and radiological parameters. Electrolyte disturbances were defined using standard laboratory reference ranges, including hyponatremia (serum sodium <135 mmol/L), hypokalemia (potassium <3.5 mmol/L), hypochloremia (chloride <98 mmol/L),

hypomagnesemia (magnesium <0.70 mmol/L), and hypophosphatemia (phosphate <1 mmol/L). Additional indicators of renal dysfunction included proteinuria (≥+1 on urine dipstick), hematuria, elevated serum urea (>6.2 mmol/L), and increased serum creatinine (>115 µmol/L), with particular attention to critically elevated levels. Furthermore, specific renal pathologies such as pseudo-Bartter syndrome, nephrolithiasis, nephrocalcinosis, acute kidney injury, chronic kidney disease, nephrotic syndrome, and diabetic nephropathy were assessed where applicable.

Genotypic characterization of CFTR mutations was performed as per previously validated methodologies described in earlier publications. Clinical and laboratory data were extracted across four predefined time points to allow longitudinal assessment. These included the initial presentation (Period 1), follow-up at 3–5 years or at the time of transplantation if applicable (Period 2), subsequent follow-up visits with or without transplantation (Period 3), and the most recent clinic evaluation (Period 4). This temporal stratification enabled evaluation of disease progression and the emergence of renal complications over time.

The study adhered to ethical standards outlined in the Declaration of Helsinki and complied with Good Clinical Practice (GCP) guidelines. Ethical handling of patient data was maintained throughout the study, with secure storage within the pediatrics research unit. Statistical analyses were conducted by an independent team from the Department of Biostatistics, Epidemiology, and Scientific Computing (BESC), ensuring methodological rigor and unbiased interpretation of findings.

Table 1. Patient demographics and renal disease overview

Characterestics	Number (%)
Gender Male Female	 213 (49.54) 217 (50.46)
Region: Eastern Central others	 156 (36.3) 100 (23.3) 174 (40.4)
First degree cousins: Yes NO	 194 (45.1) 236 (54.9)
Family history of CF: Positive Negative	 193 (44.9) 237 (55.1)

Statistical Analysis

All collected data were entered into a structured database using Microsoft Excel, following strict anonymization procedures. Each patient was assigned a unique study identifier, and all personal identifiers were removed to maintain confidentiality. Data accuracy and consistency were ensured through supervision by the principal investigator, with access limited to authorized research personnel only. Data analysis was performed using IBM SPSS Statistics for Mac, Version 26.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize the baseline characteristics and clinical findings of the study population. Categorical variables were presented as frequencies and percentages, while continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR), depending on data distribution.

RESULTS

Patient Demographics and Renal Disease Overview

Out of 430 confirmed cystic fibrosis (CF) patients, 213 (49.54%) were male and 217 (50.46%) were female. The majority of patients, 156 (36.53%), were from the Eastern region, followed by 100 (23.42%) from the Central region. A significant proportion, 194 (45.04%), were first-degree cousins, and 193 (44.96%) had a positive family history of CF. Additionally, 136 males (63.79%) and 150 females (69.13%) had between 1 to 3 siblings diagnosed with CF.

Renal Disease in CF Patients

Three patients developed renal disease, with the age of CF diagnosis ranging from 2 to 9 years.

- Patient #1 had recurrent vomiting and electrolyte disturbances from 1 month of age, and was diagnosed with interstitial nephritis at age 2. At age 9, she presented with acute pancreatitis, and diagnosed as CF. The patient developed renal disease and had elevated urea and creatinine at the time of CF diagnosis which continued to rise. Initially the patient refused dialysis, which was eventually started at age 12. At age of 13 years, renal transplant was done and renal function normalized post-transplant, but the patient later developed Cystic Fibrosis-Related Diabetes (CFRD) at age 16.
- Patient # 2 had history of recurrent vomiting and electrolyte disturbance from age of 5 months,

diagnosed as CF at age of 2 years, and developed renal failure at age of 9 years with elevated urea and creatinine which continued to rise. The patient refused dialysis and was not compliant with follow up.

- Patient # 3 diagnosed at age of 2 years with CF; he had frequent exacerbation and underwent lung transplant at age of 12 years. At age of 14 years, she had elevated urea and creatinine, which normalized with follow-up.

Clinical Findings in the 3 renal patients

- Proteinuria: Present in all three patients but at different stages—before renal transplant in Patient #1, upon renal failure diagnosis in Patient #2, and before lung transplant which continued to show thereafter in Patient #3.
- Hematuria: Noted in Patient #1 both before and after the renal transplant at age of 14 years.
- Glycosuria: Detected in Patient #1 before renal transplant, with CFRD developing at age 13. Patient #3 also developed glycosuria and CFRD before their lung transplant at age 12.
- Ketonuria: Both Patient #1 and Patient #3 developed trace ketonuria (at ages 16 and 15, respectively).
- Electrolyte Disturbances and Renal Function: All three patients had recurring episodes of hyponatremia, hypokalemia, and hypochloremia. Hypomagnesemia developed in Patient #1 post-renal transplant and in Patient #3 post-lung transplant. Hypophosphatemia was present in Patient #2 at age 17. High urea and creatinine were seen in all three patients at ages 9, 9, and 14, respectively.
- Elevated liver enzymes were noted in Patient #2 and Patient #3.

Table 2. Laboratory parameters across study periods for three patients

	Patient 1 (M.A.)				Patient 2 (M.S.)				Patient 3 (A.D.)			
Variable	**Period 1**	**Period 2**	**Period 3**	**Period 4**	**Period 1**	**Period 2**	**Period 3**	**Period 4**	**Period 1**	**Period 2**	**Period 3**	**Period 4**
Hb (g/L)	**129**	**97 (L)**	**120**	**100 (L)**	**130**	**143 (H)**	**155**	**130**	**143**	**100 (L)**	**123**	**165**
MCV (fL)	76.4 (L)	84.2	85.8	71.7 (L)	77.1 (L)	77.1	83.2	87.7	81.1	78.6	86.3	84.7
MCH (pg)	25.5 (L)	28.4	27.4	20.3 (L)	25.7 (L)	25.9 (L)	28.9	29.4	27.9	24	27.6	28.7
RDW (%)	16.7 (H)	16.1 (H)	13	16.7 (H)	16	12.8	12.6	12.3	12.4	16.9 (H)	13.2	12.6
Urea (mmol/L)	5.6	17.5 (H)	3.6	6.2	5	7.8 (H)	7.3 (H)	18.9 (H)	3.4	4	20.5 (H)	5.6
Creatinine (µmol/L)	23	633 (H)	48	56	66	152 (H)	147 (H)	435 (H)	33	64 (H)	173 (H)	81
Mg (mmol/L)	0.74	0.52 (L)	0.72	0.55 (L)	0.78	0.86	0.83	0.90	0.80	0.97	0.69 (L)	0.75
PO□ (mmol/L)	1.32	1.82 (H)	1.55 (H)	1.46	1.44	1.84 (H)	0.83 (L)	1.77 (H)	1.35	1.14	1.71 (H)	1.62 (H)
Urinalysis (UA)	T.P	+2 P, T.G	+3 B	+1 P	T.K	NA	NA	NA	NA	+1 P, P.B	−ve	T.K

L = Low ; H = High ; T.P = Trace protein , T.G = Trace glucose , P = Protein , B = Blood ; P.B = Protein + Blood ; T.K = Trace ketones ; NA = Not available

Genetic Findings and Pulmonary Function

All three patients shared a common CFTR mutation: c.1418 Del G Exon 11.

Pulmonary function tests (PFTs) showed that patient #1 had normal parameters at the time of renal failure but showed mild deterioration post-transplant. Patient #2 maintained normal PFTs up until age 22. Patient #3 had moderate combined obstructive and restrictive lung disease before the lung transplant, with deterioration afterward.

Table 3. Pulmonary function test (PFT) parameters across study periods for three patients

	Patient 1 (M.A.)				Patient 2 (M.S.)				Patient 3 (A.D.)			
Variable	Period 1	Period 2	Period 3	Period 4	Period 1	Period 2	Period 3	Period 4	Period 1	Period 2	Period 3	Period 4
Resp. C/S	–ve	–ve	–ve	–ve	–ve	–ve	–ve	–ve	–ve	M. PS	M. PS	–ve
FVC (%)	82	74	77	68	75.4	45.9	92.5	69	59.1	21	62	35
FEV1 (%)	85	73	72	70	88.8	53	99.1	67	61.4	24	69	30
FEV1/FVC (%)	106	100	82	91	100	99.3	89.4	88	87.98	100	98	77
MMEF 25–75 (%)	87	59	64	84	149	67.7	87.4	61	53.3	71	98	19
RV (%)	125	114	NA	NA	NA	97.8	135	NA	NA	713	NA	NA
TLC (%)	82	82	NA	NA	NA	57.7	81	NA	NA	187	NA	NA
RV/TLC (%)	39	36	NA	NA	NA	172	150	NA	NA	79	NA	NA

L = Low ; H = High ; T.P = Trace protein , T.G = Trace glucose , P = Protein , B = Blood ; P.B = Protein + Blood ; T.K = Trace ketones ; NA = Not available

Imaging and Other Findings

- Renal Ultrasound in all three patients showed echogenic kidneys with poor cortical-medullary differentiation.
- All three patients had Bronchiectasis and interstitial lung pattern.
- No renal biopsies were performed on any of the patients.
- Patient #1 had an echogenic fatty liver, while Patient #3 had an echogenic liver with mild enlargement.
- Both Patient #1 and Patient #3 had echogenic pancreas, with mild to moderate dilatation of the main pancreatic duct in Patient #1.

DISCUSSION

This study identified three CF patients with renal disease in a tertiary care centre, emphasizing the necessity of regular renal monitoring in CF patients. Several studies have explored the relationship between cystic fibrosis (CF) and renal disease. Hoppe B. et al. reported that people with CF are more prone to nephrocalcinosis and urolithiasis due to the increased excretion of lithogenic substances and urinary supersaturation. Hyperoxaluria was observed in 25 out of 63 CF patients in their study, with urinary calcium levels elevated in 13 patients (4.1–8.22 mg/kg per 24 hours) **[15]**.

Katz et al. also examined 38 kidney tissue specimens stained for calcium deposits. Furthermore, 24-hour urinary calcium excretion was measured in 14 patients and 15 control subjects. Microscopic nephrocalcinosis was found in 92% of the specimens (35 out of 38), while hypercalciuria (above 182 mg per gram of creatinine) was observed in 36% of patients (5 out of 14). Their findings suggest that renal calcium deposits may be

associated with the underlying genetic defect of CF rather than factors like pulmonary dysfunction, chronic infection, or treatment **[16]**.

Similarly, Yahiaoui et al. identified 13 CF patients with renal disease, 11 of whom had proteinuria. This was associated with progressive renal impairment in four patients, with a median serum creatinine level of 85 µmol/L. Various nephropathies were found through renal biopsies, including amyloidosis, diabetic glomerulopathy, and focal segmental glomerulosclerosis. The authors recommended that kidney biopsy be considered in CF patients with significant renal disease, particularly those requiring organ transplantation **[17]**.

In our series, renal disease presented between ages 9 and 14, with proteinuria, hematuria, and electrolyte disturbances being consistent features in all patients. Proteinuria was observed at different stages of renal disease progression, and hematuria was persistent in one patient even after renal transplantation. Glycosuria and eventual cystic fibrosis-related diabetes (CFRD) also developed in two patients, highlighting the metabolic challenges that often accompany CF, particularly in those with pancreatic involvement. These observations align with other reports suggesting that renal complications in CF are multifactorial, often linked to chronic infections, nephrotoxic medications, and electrolyte imbalances **[18]**.

Previous research has shown that circulating immune complexes from infection may contribute to kidney disease in CF patients, with methicillin-resistant *Staphylococcusaureus*(MRSA)beingimplicatedinsome cases of membranoproliferative glomerulonephritis. Furthermore, the nephrotoxic effects of aminoglycoside antibiotics are well documented **[19,20]**, and diabetic nephropathy has been reported in some CF patients, a pattern also observed in one patient in our study **[20]**.

Organ transplantation, including renal and lung transplants, significantly impacted the renal disease course in two of our patients. Patient #1 experienced normalization of renal function following a transplant but later developed CFRD, a common complication due to immunosuppressive therapy **[18]**. Patient #3, who underwent a lung transplant, experienced temporary normalization of renal function post-transplant, but glycosuria and other renal issues persisted. This association between organ transplantation and renal dysfunction in CF is well established, particularly in lung transplant recipients who are at higher risk for chronic kidney disease due to nephrotoxic immunosuppressants like calcineurin inhibitors **[19]**.

All three patients exhibited varying degrees of pulmonary impairment, with bronchiectasis and interstitial lung patterns noted on imaging. Interestingly, pulmonary function tests (PFTs) remained relatively stable in two patients, while one showed deterioration post-transplant. Previous studies have shown that the severity of pulmonary disease in CF can correlate with the degree of renal impairment, particularly in patients with advanced lung disease or those requiring lung transplantation **[18]**. This relationship may be attributed to the increased use of nephrotoxic medications in severe cases.

Researchers at Sapienza University in Rome, Italy, assessed renal function in 226 CF patients and found that 65 patients (28.8%) had an estimated glomerular filtration rate (eGFR) below 90 mL/min/1.73 m^2, indicating chronic kidney disease (CKD). The majority of these patients had the class II CFTR mutation, which is known to be associated with more severe CF phenotypes. Additionally, lung transplant recipients had lower eGFR values compared to CF patients without transplants, suggesting a direct impact of transplantation on renal function. Lipid profile abnormalities, including elevated triglycerides and low-density lipoproteins (LDL), were also associated with more severe kidney damage **[20]**, though lipid tests were not routinely performed in our patient cohort.

In our study, all three patients had a common CFTR mutation in at least one allele (c.1418 Del G Exon 11). Pulmonary function tests (PFTs) showed varying degrees of lung function impairment, with deterioration noted in some patients. Renal ultrasound findings in all three patients consistently showed echogenic kidneys with poor corticomedullary differentiation, a hallmark feature of nephrocalcinosis and interstitial nephritis, conditions commonly seen in CF patients with renal involvement **[17]**. Although no renal biopsies were performed, the imaging findings align with typical CF-related renal pathology reported in the literature **[18]**.

CONCLUSION

This case series highlights the diverse clinical presentation of renal disease in CF patients, showing that while rare, renal complications can significantly impact patient outcomes, particularly in those requiring organ

transplants. Early recognition of renal dysfunction, close monitoring of electrolyte imbalances, and careful management of nephrotoxic therapies are critical to improving outcomes in CF patients. The presence of common CFTR mutations such as c.1418 Del G Exon 11 may offer insight into which patients are at higher risk of developing renal disease, warranting further genetic research. As CF patients continue to live longer, renal disease may become an increasingly relevant aspect of CF care, necessitating proactive screening and intervention strategies. Renal disease is a common but often underdiagnosed complication of cystic fibrosis. Routine evaluation of renal function, including urinalysis and electrolyte monitoring, should be implemented to prevent chronic kidney disease and minimize the need for renal transplantation.

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