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Polycystic Kidney Disease: Epidemioclinic, Evolutionary and Prognostic Profile in the Nephrology Department of the Point G Hospital, Mali

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Abstract

Introduction: Autosomal dominant polycystic kidney disease (ADPKD) is a dominantly inherited genetic disorder. The affected parent has a 50% chance of transmitting the mutated gene to his or her offspring. The aim of this study was to evaluate the epidemioclinic, evolutionary and prognostic profile of ADPKD cases in the nephrology department of the Point G University Hospital. **Materials and Methods:** We conducted a descriptive study with retrospective data collection on 36 medical records of ultrasound-confirmed polycystic kidney disease in the Nephrology Department at Point G University Hospital during the period from January 1, 2018 to December 31, 2021. **Results:** The overall prevalence of ADPKD in the study was 1.08%, with an annual incidence of 09 cases. The mean age of patients was 49.14 ± 13.842 years, with extremes of 16 and 82 years. The 41 - 59 age group accounted for 56% of cases. The circumstances of discovery were, in order of frequency, lumbar pain and/or heaviness (39%), renal failure (36%), ultrasound (22%) and macroscopic hematuria (3). Chronic renal failure was the main complication (83% of cases). Progression was favourable in the majority of cases (75% of patients). **Conclusion**: ADPKD is probably underdiagnosed in black Africa. It is often

discovered late in life, when renal function is already impaired. Earlier family screening combined with nephroprotective measures will improve patient prognosis.

Keywords

Polycystic Kidney Disease, Nephrology, Point G University Hospital

1. Introduction

Autosomal dominant polycystic kidney disease (ADPKD) is a dominantly inherited genetic disorder. The affected parent has a 50% chance of passing on the mutated gene to his or her offspring. Two genes may be mutated in ADPKD. In 85% of cases, these are PKD1 (16p13.3), and in 15% PKD2 (4q21). These proteins form a complex at the base of the primary cilium, an organelle that acts as an antenna, informing the cell of its external environment and polarity. Mature polycystin-1 is cleaved; the level of residual mature polycystin-1 correlates with disease severity [1]. It is the most common hereditary kidney disease [2]. It occurs at a median age of 46 for PKD1 and 51 for PKD2 [3]. It occurs in 13% of children [4]. It is characterized by the progressive development of renal cysts at the expense of the various segments of the tubule, resulting in increased kidney size and progressive destruction of the renal parenchyma, leading to end-stage renal failure before the age of 65. ADPKD is often associated with extra-renal localization of cysts, particularly in the liver [5] [6].

Several complications may arise during the course of the disease, or be the cause of its discovery. Despite numerous advances in the management of the disease over the past 20 years, bacterial infection of the kidney or liver, with intracystic infection at its forefront, remains a serious and potentially severe complication, often leading to cyst rupture. It is not always easy to diagnose, and its treatment is poorly codified [1] [7].

Estimating the prevalence of ADPKD is difficult, as the condition is long asymptomatic and therefore only diagnosed late. The prevalence of PKRAD is one in 1000 in the general population. Worldwide, according to population-based studies, the prevalence of ADPKD screening is between 3.3 and 4.6/10,000 inhabitants [2], and is responsible for 8.8% of cases of chronic end-stage renal disease in France [8].

A study carried out in Senegal in 2006 by Mohamed Ould M [9] reported an incidence of 5 cases/year at the Dakar university hospital. In Mali, from January 2004 to January 2006, Atteyine F found a frequency of 0.48%, with an incidence of 16 cases/year [10]. From February 2012 to December 2013, Diarra A found sixty-three (63) cases of polycystic kidney disease, or 5.3% of all examinations performed in the radiology and nuclear medicine department of the Point G University Hospital [11].

At present, there are few studies on polycystic kidney disease in Mali. With this in mind, we felt it necessary to carry out this review, the aim of which was to assess the epidemioclinic, evolutionary and prognostic profile of polycystic kidney disease in the nephrology department of Point G University Hospital in Mali.

2. Methodology

2.1. Setting and Type of Study

Our study was carried out in the nephrology and haemodialysis department of the Point G university hospital in Bamako (administrative and economic capital of the Republic of Mali). The Point-G hospital is a third-level referral center. It is located eight kilometers from the city center, on the Point G hill. He currently comprises 22 departments. The nephrology department was created in 1981, and the hemodialysis unit opened in April 1997.

This was a descriptive study with retrospective data collection that ran from January 1, 2018 to December 31, 2021, *i.e.*, four years. It concerned the records of patients hospitalized and/or followed as outpatients in nephrology during the study period.

2.2. Study Population

The study focused on patients with uni or bilateral renal cysts.

Inclusion criteria: Patients diagnosed with polycystic kidney disease according to the Ravine criteria were included in the study.

Non-inclusion criteria: patients with renal cysts who did not meet the Ravine criteria, and patients with incomplete records, were not included.

2.3. Data Collection

A pre-established individual survey form was used to collect patient data from their hospitalization record and consultation register. The variables studied included.

- Socio-demographic variables: age, sex, origin, ethnicity, nationality and marital status.
- Clinical variables: reasons for hospitalization, medical and surgical history, cardiovascular risk factors, medications taken, general signs (fever, physical asthenia, weight loss, anorexia), functional signs (vomiting, nausea, insomnia, somnolence, diarrhea, pruritus, muscle cramps, headaches, erectile dysfunction, vertigo, tinnitus, lumbar pain, pelvic pain, dyspnea, hemoptysis, hematemesis, rectorrhagia, melena, cough, irritability, asterixis, confusion), diuresis abnormalities (anuria, oliguria, polyuria), micturition disorders (dysuria, micturition burning, pyuria, pollakiuria, macroscopic hematuria).

Elements of physical assessment were evaluated, such as eye examination (conjunctiva, icterus), World Health Organization (WHO) performance index, blood pressure, heart rate, temperature, body mass index, diuresis, cardiac examination,

pulmonary examination, abdominal examination, skin examination, neurological and joint examination.

- Biological tests included blood tests (hemogram, urea, creatinemia, uric acid, ionogram, phosphocalcic balance, martial balance, lipid balance, phosphocalcic balance, albuminemia, protidemia, transaminases and infectious diseases), urine tests (urine cytobacteriological study, 24-hour proteinuria, urine ionogram). The standards used were those of the analysis laboratories in the Kayes region.
- Imaging included reno-vesico-prostatic ultrasound and uro-scanner.
- Management was medical (conservative treatment) and hemodialysis (HD). HD was indicated as an emergency treatment for severe hyperkalemia ≥7.5 mmol/l refractory to medication as assessed by ECG, clinical metabolic acidosis with impaired ventilatory compensation with no margin for correction by bicarbonate in the event of hypervolemia, pulmonary acute edema refractory to diuretic treatment and uremic syndrome with encephalopathy (asterixis, confusion or coma) or pericardial friction [9].

The evolution of our patients included lethality, total or partial recovery of renal function in HD, and maintenance of patients in chronic HD.

- Operational definitions:

Acute kidney injury (AKI): is defined by a sudden and severe drop in glomerular filtration rate, usually reversible after treatment (**Table 1**).

Table 1. Definitions of AKI according to K-DIGO 2012 (Kidney Disease Improving Global Outcome) [12].

Stage of AKI	Creatininemia	Diuresis
1	Increase > 26 µmol/L (3 mg/L) in 48 h or >50% in 7 days	<0.5 ml/kg/h for 6 to 12 h
2	Creatinineemia × 2	<0.5 ml/kg/h ≥ 12 h
3	Creatininemia × 3 Or Creatininemia > 354 µmol/L (40 mg/L) in absence of previous value Or Need for dialysis	<0.3 ml/kg/h ≥ 24 h or Anuria ≥ 12 h

Table 2. Stages of chronic kidney disease.

Stages	Description	GFR (ml/min/1.73 m²)
1	Chronic kidney disease with normal renal function	≥90
2	Chronic kidney disease with mild renal impairment	60 - 89
3A	A Mild to moderate renal failure	45 - 59
3B	Moderate to severe renal failure	30 - 44
4	Severe renal failure	15 - 29
5	Severe renal failure	<15

Chronic kidney disease (CKD): this is defined by the existence of a functional

or structural abnormality that has been evolving for more than three months (this may be a morphological abnormality provided it is clinically significant, a histological abnormality or an abnormality in the composition of blood or urine secondary to kidney damage; and/or a glomerular filtration rate (GFR) < 60 ml/min/1.73 m2 for more than 3 months. Based on the clearance value, we defined five stages of chronic renal failure shown in **Table 2** [13].

ADPKD: The diagnosis of polycystic kidney disease was based on the arguments proposed by Ravine D *et al.* [14]. **Table 3** shows the ultrasonographic diagnostic criteria for ADPKD.

Table 3. Ultrasonographic diagnostic criteria for ADPKD by Ravine [14].

Age in year	Ultrasound diagnostic criteria
<30	At least two (2) cysts in one or both kidneys
30 - 59	At least two (2) cysts in each kidney
≥60	At least four (4) cysts in each kidney

High blood pressure: Hypertension was defined as any elevation of blood pressure ≥ 140 mmHg systolic and/or ≥ 90 mmHg diastolic in people with or without known hypertension. Hypertension was classified according to the WHO [15].

2.4. Data Entry and Analysis

Variables were initially recorded on survey forms and then entered and analyzed using an epidemiological analysis tool, SPSS version 26.0 and Excel 2018. Qualitative and quantitative variables were expressed in tabular form. The statistical test used to compare qualitative variables was Pearson's Chi-square. A threshold of $p < 0.05$ was considered statistically significant.

2.5. Ethical Considerations

The study complied with the ethical standards of our institution's research committee. Each patient and/or family was informed of the objectives of the study and the use of data for research purposes. Strict anonymity was guaranteed.

3. Results

During the 4 years, 3332 patients were seen in the department, 36 of whom were ADPKD carriers, *i.e.*, a frequency of 1.08% (36/3332) and an annual incidence of 09 cases. The mean age in our study was 49.14 ± 13.842 years, with extremes of 16 and 82 years. The 41 to 59 age group accounted for 56% of cases (**Table 4**). Females accounted for 75% of cases. The circumstances of discovery were, in order of frequency, lumbar pain and/or heaviness in 14 cases (39%), renal failure in 13 cases (36%), incidental finding on ultrasound in 8 cases (22%), macroscopic hematuria in 1 case (3) (**Table 5**). One patient in two was known to be hypertensive. General signs were mainly anorexia in 12 cases (55%) and physical asthenia in 9

cases (41%). Functional signs were dominated by headache in 25 cases (69%) and dizziness in 22 cases (61%). On physical assessment, a large palpable kidney was found in 78% of patients (28 cases) (Table 6). Among the 58% (21 cases) of hypertensive patients, hypertension was WHO grade 3 in 22% of cases. Diuresis was preserved in 71% of cases, versus 26% with oliguria and 3% with anuria. CKD was the main complication, accounting for 83% (34 cases) (Table 7). CKD was end-stage in 49% of patients (17 cases). The mean hemoglobin level was 8.54 g/dl, with extremes ranging from 8 to 10 g/dl. In 10 patients, hemoglobin was between 8 - 10 g/dl. Blood ionograms showed hypocalcemia, hyponatremia and hyperkalemia in 21 cases (58.3%), 9 cases (25%) and 3 cases (8%) respectively. Proteinuria was absent in 59% of cases, minimal and moderate in 24% and 17%. On ultrasonography, the kidneys were enlarged in 81% of cases. Cysts were located in the liver in 12 cases (33%). On urine cytobacteriological study, haematuria was found in 8 cases. It was associated with leukocyturia in 7 cases (23%). Uro-culture was positive in 10 patients (33%), with *Escherichia coli* as the most predominant germ (60%). In terms of management, hygienic and dietary measures included protein restriction, a low-potassium and low-phosphorus diet, treatment of anemia with iron supplementation in 10 cases, blood transfusion in 12 patients, and erythropoeitin administration in 5 patients.

Table 4. Patient distribution by age group.

Age group in years	Numbers	Proportion in %
16 - 40	6	16
41 - 59	20	56
≥60	10	28
Total	36	100

Table 5. Occurrence of PKRAD.

Circumstances of discovery	Numbers	Proportion in %
Lumbar pain or heaviness	14	39
Renal insufficiency	13	36
Incidental discovery on ultrasound	8	22
Macroscopic hematuria	1	3
Total	36	100

Table 6. Clinical data.

Clinical Data		Numbers	Percentage
Antecedent	Hypertension	18	50
	Gross hematuria	3	8
	Diabetes	1	3

Continued

General signs	Anorexia	12	55
	Asthenia	9	41
	Fever	7	19
Functional signs	Headache	25	69
	Vertigo	22	61
	Nausea/Vomiting	16	73
	Effort dyspnea	9	25
	Pelvic pain	3	8
Physical sign	Conjunctival pallor	20	56
	Large kidney	28	78
	Lower limb edema	11	31
	Pulmonary crackling	3	8
	Deshydration	8	22

Table 7. Distribution of patients according to complications.

Complications*	Number	Proportion in %
CKD	30	83
Abdominal pain	28	78
Hypertension	28	78
Urinary tract infection	12	33
Macroscopic hematuria	7	19
Cardiac involvement	2	6
Lithiasis	1	3

*A patient could have one or more associated complications.

Calcium supplementation was used in 21 patients. Hypertension was treated by monotherapy (5 cases), bitherapy (16 cases), tritherapy (7 cases). Molecules used were calcium antagonists, ACE inhibitors, beta-blockers and diuretics. Pain was treated with antispasmodics and/or in combination with tramadol/paracetamol. Urinary tract infections were treated with ciprofloxacin, 3rd-generation cephalosporins and imipenems. Antibiotic therapy was adapted to the antibiogram and renal function. All patients with end-stage CKD had benefited from hemodialysis, including 13 women and 4 men.

The outcome was favorable in 27 patients (75%), with a case-fatality rate of 25% (9 cases).

4. Discussion

The hospital frequency of ADPKD in the study was 1.08% (36 cases collated/3332 records reviewed) with an annual incidence of 09 cases of all patients followed.

ADPKD is the most common hereditary kidney disease [1] [2]. As the disease remains asymptomatic for a long time, its prevalence is difficult to estimate in affected subjects. Depending on the study and the population studied, it is estimated at between 1:400 and 1:1000 births [1] [16] [17]. In our context, although the frequency of ADPKD is low compared with the literature, it is clearly increasing in the nephrology department of the Point G hospital, rising from 0.48% in 2008 [10] to 1.08% in 2022. The mean age in our study was 49.14 years, with extremes of 16 and 82 years. The 41 - 59 age group accounted for 56% of cases. Atteyine F in 2008 found a mean age of 48.59 years, with extremes of 20 and 80 years, and an age range of 40 and 60 years (56.27%) in the same department [10]. In the radiology and nuclear medicine department of the Point G hospital, Diarra A found an age range between 40 and 60 years [11]. In Senegal, Mouhamed Ould M found an age range between 45 and 54 (32.7%) [9]. According to the literature, the median age at onset of PKD1 is 46, and 51 for PKD2 [3]. It is found in 13% of children [4]. The circumstances of discovery were, in order of frequency, lumbar pain and/or heaviness (39%), IR (36%), ultrasound (22%) and macroscopic hematuria (3%). Atteyine F in 2008 [10] and Diarra A in 2014 [11] found low back pain (37.5%) and (40%), hypertension (21.9%) and (20%), macroscopic hematuria (15.6%) and (15%) and renal failure (9.4%) and (7.5%). Abdominal pain is the first sign of PKRAD in 20% to 30% of cases. Their frequency increases with age and the size of the renal cysts. Pain is multifactorial in origin, and can be explained by urinary tract infections and cyst enlargement [1].

In our study, urinary tract infection was the fourth most common complication (33%), and cystic infections were not documented. Atteyine F [10] found urinary tract infection in 25% of cases.

Infections of renal or hepatic cysts are sometimes severe complications of ADPKD. Their frequency is estimated at 0.01 episodes/year/patient in hospitalized polycystic patients, representing 11% of causes of hospitalization in this population. The germs most often incriminated are of digestive origin (*Escherichia coli* in around 75% of cases), but microbiological documentation is not always available. The lack of specificity of clinical signs often delays diagnosis. The gold standard for diagnosing cyst infection is percutaneous puncture, which reveals neutrophils and microorganisms. However, this is not always feasible, either because the infected cyst is not identified, or because it is not accessible to percutaneous puncture [18].

In our sample, the incidence of hypertension was 58%. It was more frequent in subjects aged between 40 and 59 years (44.4%). In Senegal, on the other hand, Mouhamed Ould M reported a prevalence of 71% [9], and Ndongo M in 2019 found a prevalence of 46.5% [19]. Hypertension is very frequent and early in ADPKD, occurring in 50% - 70% of patients before any substantial reduction in glomerular filtration rate. It appears to result from excessive activation of the renin-angiotensin system through compression of intra-renal vessels by cysts [20].

With regard to renal involvement, 5 cases of chronic kidney disease with normal renal function, 30 cases of CKD and 1 case of ARF due to obstetric causes

(pregnancy on polycystic disease) have been reported. Atteyine F [10] in 2008 and Diarra A [11] in 2014 found CKD in 100% of cases. This difference could be explained by later consultation in their studies. The rate of progression of renal failure has been shown to correlate with the rate of growth of renal volume. Initial renal volume predicts the rate of subsequent renal growth, and a high initial renal volume (greater than 1500 mL) is associated with a greater decline in GFR (4.33 to 8 mL/min per year) [21]. In addition to renal volume growth rate, rapid progression of renal failure is also associated with truncated PKD1 mutations, male gender, the onset of early-onset hypertension, the presence of macroscopic hematuria before the age of 30, and in hypertensive women, the existence of more than 3 pregnancies [22]. In this study, 49% of patients with chronic renal failure were in the end-stage.

CKD generally occurs at a median age of 53 years for PKD1, and 69 years for PKD2 [23]. The probability of developing CKD varies in the literature. In France, it is 17% at age 50, 47% at age 60 and 70% at age 70, with no significant difference between men and women [24]. The mean hemoglobin level of our patients was 8.5 g/dl, with extremes of 2.8 and 16 g/dl. Atteyine F [10] found a mean hemoglobin level of 10.87 g/dl, with extremes ranging from 4.5 g/dl to 15 g/dl. In ADPKD, there is a tendency towards polycythemia, as the renal cysts secrete erythropoeitin. In fact, in stages 3 and 4 of chronic kidney disease, erythropoeitin production by the cysts helps maintain a high hemoglobin level. In end-stage CKD uraemia, however, erythropoeitin production is blocked by uraemic toxins [25]. This may explain the frequency of anemia in our study, since most of our patients had end-stage renal failure. In multivariate analysis, we found a statistically significant relationship between end-stage renal failure and severe anemia below 8 g/dl. P = 0.024, odd ratio = 2.036 (1.059 - 3.912). There was no correlation between age, sex, death and severity of chronic renal failure (**Table 8**).

Table 8. End stage renal disease and potential associated factors.

		End stage renal disease		*P*
		YES	**NO**	
Gender	Male	4 (44.4%)	5 (55.6%)	0.563
	Female	15 (55.6%)	12 (44.4%)	
Age	16 to 45 years	7 (50%)	7 (50%)	0.790
	46 years and over	12 (54.5%)	10 (45.5%)	
severe anemia*	Yes	12 (75%)	4 (25%)	0.024
	No	7 (36.8%)	12 (63.2%)	
Death	Yes	7 (77.8%)	2 (22.2%)	0.083
	No	12 (44.4%)	15 (55.6%)	

* Severe anemia: hemoglobin level below 8 g/dl.

Several other complications may arise during the course of ADPKD. It may be

associated with multiple and variable extra-renal involvement. In our series, we noted 12 cases (33%) of polycystic liver disease. The onset of extra-renal cysts is usually late, classically delayed by around 10 to 20 years compared with renal cysts [26].

The overall outcome was favorable in 27 patients (75%), with a case-fatality rate of 25% (9 cases). The causes of death were severe uraemia in 6 cases (67%), severe sepsis in 2 cases (22%) and 1 case (11%) of haemorrhagic syndrome.

The recommendations suggest the most conservative therapeutic attitude possible, in order to preserve as much residual renal function as possible and a good quality of life. The decision and therapeutic strategy will depend on the transplant project (living or non-living donor transplant), whether or not dialysis has been started, the mode of dialysis, the existence of complications of polycystic kidneys and the impact of liver cysts. Surgery remains the treatment of choice, particularly in cases of excessive renal volume or infectious, lithitic or neoplastic complications of polycystic kidneys [27] [28].

This study, although innovative, suffered from certain shortcomings, notably the small sample size. We used ultrasound diagnostic criteria, given the absence of genetic diagnostic confirmation. This could possibly be a factor biasing the prevalence, as ultrasound remains an operator-dependent examination. This prevalence is hospital-specific and cannot be extrapolated to the general Malian population.

5. Conclusion

ADPKD is probably underdiagnosed in black Africa. Discovery of the disease is often late, with advanced chronic kidney disease exposing patients to serious complications. Earlier family screening combined with nephroprotective measures will improve patient prognosis.

Conflicts of Interest

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

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Appendix

Inquiry form:

❖ ***Socio-demographic characteristics***:

- Full name:

- Age: /_____/

- Sex: /_____/ 1 = Male; 2 = Female

- Provenance: /_____/ BAMAKO (1) Kayes (2) Koulikoro (3) Sikasso (4) Ségou (5) Mopti (6) Gao (7) Tombouctou (8) Kidal (9) Taoudéni (10) Ménaka (11) autre pays (12) Bougouni (13) Nioro (14) Kita (15) Diola (16) San (17) Koutiala (18) Bandiagara (19) Douentza (20).

- Ethnicity: /_____/

Dogon (1) Peulh (2) Bozo (3) Malinké (4) Sonrhaï (5) Bambara (6) Senoufo (7) Tamashek (8) Arab (9) Others (10).

Nationality: /_____/ 1 = Malian; 2 = Other.

❖ ***Clinic***:

- Circumstances of discovery: /_____/ 1 = Arterial hypertension workup; 2 = Renal failure; 3 = Cyst infection; 4 = Nephritic colic; 5 = Lumbar pain or heaviness; 6 = Macroscopic hematuria; 7 = Fortuitous discovery on ultrasound; 8 = Other.

- Sites: /_____/ 1 = HTA; 2 = Diabetes; 3 = Heart failure; 4 = Arthrosis; 5 = Systemic disease; 6 = HIV.

- Past history

- Medical: /_____/ 1 = Oedematous syndrome; 2 = anuria; 3 = macroscopic hematuria; 4 = acute retention of urine; 5 = proteinuria; 6 = cancerous disease; 7 = pathological creatinemia; 8 = tuberculosis; 9 = pollakiuria; 10 = dysuria; 11 = voiding; 12 = ulcer syndrome; 13 = renal failure; 14 = atheromatous disease; 15 = multiple myeloma; 16 = stroke; 17 = others to be specified.

- Clinical evaluation symptomatology/

- Uremic syndrome: 1 = Asthenia; 2 = Daytime somnolence; 3 = Confusion; 4 = Asteric; 5 = Coma; 6 = Nausea; 7 = Vomiting; 8 = Hemorrhagic tendency; 9 = Hale; 10 = Uremic; 11 = Nycturia; 12 = Uremic pericarditis; 13 = Decreased libido/erectile dysfunction; 14 = Amenorrhea; 15 = Anorexia and weight loss; 16 = Muscle cramps; 17 = Insomnia; 18 = Pruritus; 19 = Epistaxis; 20 = Hematemesis; 22 = Other.

Urinary signs: 1 = Dysuria; 2 = Bruising; 3 = Pollakiuria; 4 = Hematuria; 5 = Lumbar pain; 6 = Pyuria; 7 = Polyuria.

- Other signs

Anuria yes/____/ no/____/; Pelvic pain yes/____/ no/____/; Oedema of lower limbs yes/____/ no/____/; Facial puffiness yes/____/ no/____/; Diarrhea yes/____/ no/____/; Constipation yes/____/ no/____/; Trembling yes/____/ no/____/; Fever yes/____/ no:/____/; Headache yes/____/ no/____/; Vertigo yes/____/ no/____/: Tinnitus yes/____/ no/____/; Dyspnea yes/____/ no/____/; Hemoptysis yes/____/ no/____/; Rectorrhagia yes/____/ no/____/; Melena

yes/____/ no/____/; Cough yes/____/ no/____/; Irritability yes/____/ no/____/; Asterixis yes/____/ no/____/; Confusion yes/____/ no/____/;

Physical examination; HR/_____/ 1 = Bradycardia <60; 2 = normal 60 - 95; 3 = tachycardia 95; BMI: /_____/ 1 = normal; 2 = overweight; 3 = obesity; 4 = weight loss; Diuresis /___/ 1 = anuria (<100 ml); 2 = oliguria (100 - 500 ml); 3 = conserved diuresis (>500 ml); DEC folds yes /____/ no /____/; Edema yes /____/ no/____/

If yes: Location; Uremic frostbite yes /____/ no /____/

Dry mouth yes /____/ no /____/; Stomatitis yes /____/ no /____/; Parotitis yes /____/ no /____/ Muscular atrophy yes /____/ no /____/; Abdominal distension yes /____/ no /____/; Operation scar yes /____/ no /____/; Abdominal mass yes /____/ no /____/; Ascites yes /____/ no /____/; Splenomegaly yes /____/ no /____/; Hepatomegaly yes /____/ no /____/; Adenopathy yes /____/ no /____/; Hepatojugular reflux yes /____/ no /____/; Jugular turgidity yes /____/ no /____/; Large kidneys yes /____/ no /____/; Bladder globe yes /____/ no /____/; Pelvic mass yes /____/ no /____/; Pericardial friction yes /____/ no /____/; Systolic murmur yes /____/ no /____/; Rhythm disorder yes /____/ no /____/; Thoracic deformity yes /____/ no /____/; Perceived vesicular murmur yes /____/ no /____/; Dullness yes /____/ no /____/; Pleural murmur yes /____/ no /____/; Crackling rales yes /____/ no /____/; Bone pain yes /____/ no /____/; Arthralgia yes /____/ no /____/; Polyneuritis yes /____/ no /____/; Urinary dipstick /_____/

❖ ***Additional examination***:

Creatininemia /_____; GFR according to MDRD /_____ 1 = CKD (≥90); 2 = Mild CKD (60 - 89); 3 = Moderate CKD (30 - 59); 4 = Severe CKD (15 - 29); 5 = Terminal CKD (<15); GFR CKD EPI /_____/

- CBC

Hemoglobin level in g/dl /_____/; VGM /____/ 1 = normocytic; 2 = microcytic; 3 = macrocytic; TGMH /_____/ 1 = normochromic; 2 = hypochromic; Reticulocytes /_____/ 1 = regenerative; 2 = aregenerative; GB: /_____/ 1 = normal (4000 – 10,000); 2 = diminished (<4000); 3 = increased (>100,000) Value: .....; PLAQ: /_____/ 1 = normal (150,000 – 400,000) ; 2 = Decreased (<150,000); 3 = increased (>400,000)

- Urinary sediment: Hematuria (≥10,000/ml) /_____/ yes /___/ no /___/ Value: ..... Leukocyturia (≥10,000/ml) /_____/ yes /___/ no /___/ Value: .....; Pyuria /_____/ yes /__/ no /___/; Culture/_____/ 1 = Positive; 2 = negative; If positive Germ: ..... sensitivity.....

24H proteinuria /_____/ 1 = nothing; 2 = minimal (<1 g); 3 = moderate (1 - 3 g); 4 = massive (>3 g) Value: .....

- Medical imaging

Renal ultrasound: Kidney size /_____/ 1 = diminished (<100 mm) 2 = normal (100 - 130 mm); 3 = increased (>130 mm)

CT scans: number of cysts in each kidney

Right kidney /_____/ Left kidney /_____/

DIAGNOSIS: Cystic fibrosis yes /___/ no /___/

Renal complication: Gross hematuria yes /___/ no /___/; CKD: yes /___/ no /___/; AKI: yes /___/ no /___/ ; Abdominal pain yes /___/ no /___/; Intracystic infection yes /___/ no /___/; Urinary tract infection yes /___/ no /___/; HTA yes /___/ no /___/; Lithiasis yes /___/ no /___/; Extrarenal complication; Cerebral aneurysm yes /___/ no /___/; Cardiac involvement yes /___/ no /___/; If yes: .....

➢ Other location: Liver: yes /___/ no /___/; Ovary yes /___/ no /___/; Pancreas yes /___/ no /___/

Other: .....

***Evolution*:**

- Favourable
- Death
- Discharge against medical advice