be seen in 5%–30% of patients and portends severe disease and development of lupus nephritis.[12] The U1 RNP antibody positivity is nearly 100% sensitive for mixed connective tissue disorder (MCTD), but may be associated with anti-Sm antibody in 20%–30% of SLE cases.[13]

Conclusion

We report a case of viral pneumonia (HRV) with rhabdomyolysis-related AKI, ANA positivity, renal dysfunction, bilateral lung infiltrates, and mild proteinuria who presented a diagnostic dilemma between viral infection and lupus flare. In view of the features not fulfilling the classification criteria of SLE, he needs to be carefully followed up for evolving lupus. Lupus and lupus-like illness lie on a spectrum from antibody only to evolving lupus to typical lupus to overlap disease.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Jha VK, Akal RS, Singh BP, Walia GK, Tejesh BS, Mahapatra D. Rhinovirus Pneumonia, Rhabdomyolysis-Induced Acute Kidney Injury, and Post-Viral Forme Fruste Lupus. Indian J Nephrol. 2024;34:389-92. doi: 10.4103/ijn.ijn_16_23

Received: 16-01-2023; **Accepted:** 09-02-2023
Online First: 04-08-2023; **Published:** 20-07-2024

DOI: 10.4103/ijn.ijn_16_23

Sporadic Form of Glomerulocystic Kidney Disease in a Child: A Case Report

Abstract

Glomerulocystic kidney disease (GCKD) is a rare form of cystic renal disease. We report a four-week-old baby girl born to non-consanguineous parents; their antenatal third-trimester ultrasound showed severe oligohydramnios that required amnioinfusion. Post-natal ultrasound examination showed few tiny cysts (2-3mm) involving the cortices in bilateral kidneys. Kidney biopsy showed dilatation of Bowman's space and cystically dilated glomeruli, suggestive of GCKD. Whole exome sequencing revealed no pathogenic or likely pathogenic variant.

Keywords: *Children, Glomerulocystic kidney disease, Renal failure, Ultrasound*

Introduction

Glomerulocystic kidney disease (GCKD) is a heterogeneous group of disorders characterised by tiny cortical cysts; due to the dilatation of Bowman's space.[1] GCKD occurs as sporadic and familial forms. It has been seen in infants and young children who present with renal failure.[2]

Case Report

A four-week-old baby girl born to non-consanguineous parents, was referred for evaluation of kidney dysfunction. The antenatal third-trimester ultrasound showed severe oligohydramnios that required amnioinfusion. The baby was born at 35 weeks by emergency lower segment caesarean section (LSCS) due to cord entanglement and with a birth weight of 2.2 kg. Postnatally, she experienced respiratory distress, which was treated as early-onset neonatal sepsis and with intravenous antibiotics. She had deranged renal function tests (serum creatinine 1.2 mg/dl). At three weeks of life, she was admitted for refusal of feeds and lethargy, with a serum creatinine level of 2.8 mg/dl, which increased to 4.1 mg/dl. At admission, investigations showed hyperkalemia (serum potassium at 5.6 mEq/L), deranged renal function tests (blood urea at 119 mg/dl, serum creatinine at 5.6 mg/dl), calcium at 8.7 mg/dl, phosphorus at 6.1 mg/dl, alkaline phosphatase at 224 IU/L, serum vitamin D3 at 29 ng/ml, and serum parathyroid hormone at 218 pg/ml. Ultrasonography showed a right kidney that measured 5 cm, a left kidney that measured 5.4 cm, and a few tiny cysts (2–3 mm) that involved the cortices were seen scattered at places bilaterally and had increased cortical echogenicity. Kidney biopsy was performed and showed dilatation of Bowman's space and cystically dilated glomeruli, both of which were suggestive of glomerulocystic disease [Figure 1]. No obvious extrarenal malformations or dysmorphic features were detected. There was no family history of cystic renal disorders. Whole exome sequencing revealed no pathogenic or likely pathogenic variant. Ultrasonography screening of parents was normal. At discharge, the serum creatinine level was 2.9 mg/dl [Table 1].

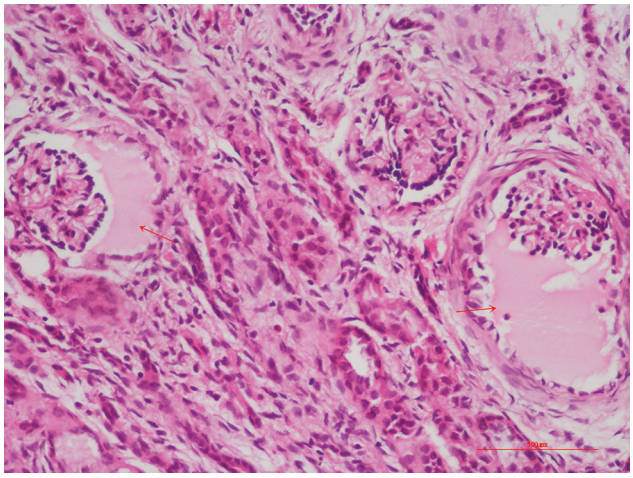

Figure 1: Renal biopsy depicting markedly dilated Bowman's space (glomerular cysts) in two glomeruli (200×, Hematoxylin and Eosin).

Discussion

Cystic glomeruli in an infant was first reported by Roos in 1941.[3] Taxy and Filmer[1] first used the term *glomerulocystic kidney disease* in 1976 to describe cystic dilatation of Bowman's space. Later, Bernstein defined glomerular cysts as the dilatation of Bowman's space to 2–3 times of the normal size in at least 5% of glomeruli.[4] Glomerulocystic kidneys can be broadly classified under three major categories: (1) GCKD, which includes sporadic forms and heritable non-syndromic forms like autosomal dominant polycystic kidney disease (ADPKD) in infants and familial hypoplastic GCKD; (2) glomerulocystic kidneys associated with heritable malformation syndromes such as tuberous sclerosis (TS), orofaciodigital syndrome type I, trisomy 13, and Zellweger cerebrohepatorenal syndrome; and (3) glomerular cystic changes in dysplastic kidneys.[4] Isolated GCKD can occur as a sporadic condition, a familial disorder, or as the infantile manifestation of ADPKD. Later on, a descriptive classification of GCKD was made.[2]

Glomerular cysts in GCKD are usually bilateral and tiny, with average size of 2–3 mm.[5] Thus, affected kidneys are of normal size or variably enlarged. Small-kidneys are seen in familial hypoplastic GCKD.[6] Most cases of GCKD are seen in the neonatal period.[7] It has a diverse presentation: renal failure is the usual presentation in newborns and infants, and hypertension, hematuria, or abdominal pain may be presented by older children and adolescents or may be detected incidentally.[8] The different clinical presentations and the course of the disease can be explained by the fact that glomerular cysts affect only a few glomeruli. The

Table 1: Serial monitoring of renal functions from birth till 11 months of age

Blood parameters	At birth	At admission (4 weeks)	At discharge	At 2 months	At 6 months	At 11 months
Blood urea (mg/dl)	57	119	48	54	84	67
S. Creatinine (mg/dl)	1.2	5.64	2.9	2.76	2.09	1.16
S. Sodium (mmol/L)		136	135	138	136	137
S. Potassium (mmol/L)		5.6	5.4	4.7	5.1	4.5
S. Calcium (mg/dl)		8.7	10	10	10.9	10
S. Phosphorus (mg/dl)		6.1	3.7	3.7	3.5	3.8
ALP (IU/L)		224	496	496	210	210

S.=Serum; ALP: Alkaline phosphatase

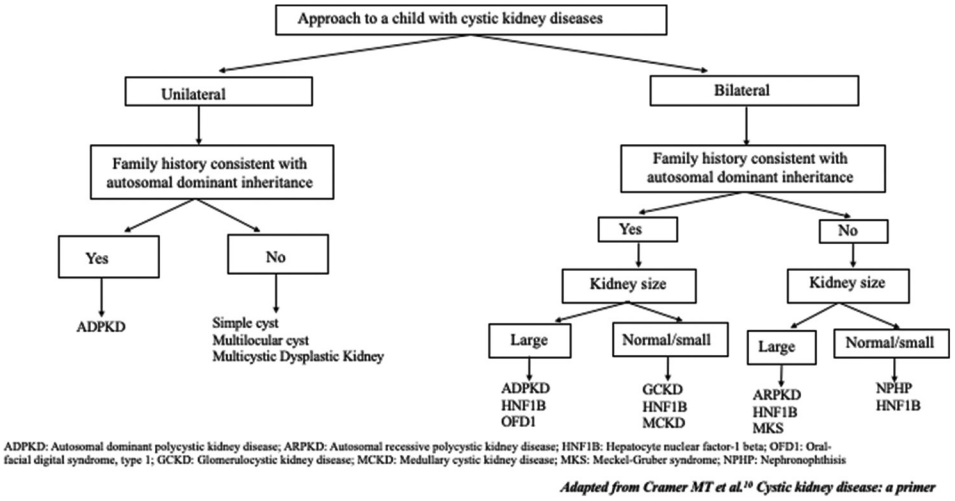

Figure 2: The flow diagram for differential diagnosis of cystic kidney disease in a child.

superimposed diseases hasten the onset of renal failure or bring the patient to clinical attention.

Ultrasonography can show tiny cortical cysts, increased cortical echogenicity, and loss of corticomedullary differentiation.[9,10] Contrast-enhanced magnetic resonance imaging (CEMRI) can be used to distinguish GCKD from other cystic renal diseases. Small renal cortical cysts can be seen on MRI and appear hypointense and hyperintense on T1- and T2-weighted images, respectively.[11] However, we did not perform MRI. Kidney biopsy is required to establish the diagnosis of GCKD and to differentiate it from other cystic renal disorders.[8] In 10% of the cases of GCKD (familial or sporadic), associated abnormalities of intrahepatic duct have been observed.[4]

The close differentials for GCKD are ADPKD, autosomal recessive polycystic kidney disease (ARPKD), and renal dysplasia [Figure 2]. The algorithm for screening children with kidney cysts have been described in literature [Figure 2].[10] The cortical distribution of cysts differentiates GCKD from other cystic renal disorders.[11] The presence of medullary cysts should raise the possibility of ARPKD, whereas tubular cysts should raise suspicion of ADPKD. In 50% of cases, GCKD in infants can be early onset ADPKD. Because there is an overlap in the clinical and pathological features of ADPKD and GCKD, the presence of renal cysts in one of the family members of a GCKD patient changes the diagnosis to ADPKD.[7] In renal dysplasia, apart from cysts, abnormal development of the renal cortex and medulla is seen.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Kaur N, Pradeep D, Nada R, Bhatia A, Dawman L, Tiewsoh K. Sporadic Form of Glomerulocystic Kidney Disease in a Child: A Case Report. Indian J Nephrol. 2024;34:392-5. doi: 10.4103/ijn.ijn_43_23

Received: 07-02-2023; **Accepted:** 28-02-2023
Online First: 04-08-2023; **Published:** 20-07-2024

DOI: 10.4103/ijn.ijn_43_23

Association of Acute Kidney Injury with Ammonia Poisoning: A Case Report

Abstract

Ammonia may cause poisoning due to inhalation or ingestion. Renal involvement in ammonia poisoning has been reported only once. A 30-year-old male working in an ice factory was accidentally exposed to liquid ammonia from a leaking hose, following which he had burns over his face and neck and severe abdominal pain. On day 2, he had deranged renal function, which was progressive. He was referred to us due to persistent renal dysfunction. A kidney biopsy was performed due to slow recovery of renal failure, which was suggestive of acute tubular necrosis. He was managed conservatively and showed gradual improvement over 12 days of his hospital stay. Renal functions normalized after 14 days of discharge. This case highlights the occurrence of renal involvement in ammonia poisoning.

Keywords: *Acute Kidney Injury, Ammonia, Poisoning*

Introduction

Ammonia is a colorless, pungent, and irritant caustic gas.[1,2] It is stored and transported as a pressurized liquid.[2] Poisoning may occur due to inhalation or ingestion and leads to local burns, gastrointestinal (GI) or respiratory manifestations, altered sensorium, shock, and even death. The first report of poisoning due to inhalation of ammonia was published in 1841, and since then, there have been various reports of ammonia poisoning and burns, but renal failure due to ammonia poisoning has been reported only once in the literature.[3,4] We report a case of accidental ammonia poisoning which led to acute kidney injury (AKI).

Case Report

A 30-year-old male who was working in an ice manufacturing factory in North India got accidentally exposed to liquid ammonia while attempting to repair a leaking hose. He developed burns over face, neck, and oral cavity, along with severe abdominal pain. He was taken to a local hospital where he was managed with proton pump inhibitors, drotaverine, and intravenous (iv) fluids. On day 2, he was found to have renal dysfunction (serum creatinine 2.1 mg/dl), although he did not have oliguria. Renal dysfunction was progressive, and serum creatinine rose to 8.2 mg/dl on day 7. He was given three sessions of hemodialysis over the next 7 days, and then he was referred to us for persistent renal failure. There was no history of vomiting, loose stools, or intake of nephrotoxic drugs (nonsteroidal anti-inflammatory drugs [NSAIDs], aminoglycosides, etc.). His medical records did not reveal any evidence of hypotension during his previous hospitalization, nor was any arterial blood gas analysis report available. On admission, he was afebrile and normotensive (blood pressure [BP] 122/80 mmHg). There were multiple shallow ulcers over lips and buccal mucosa, with superficial healing burns over nose, perioral region, and left side of neck. He had no respiratory distress or edema. Examination of chest, cardiovascular system, nervous system, and abdomen was unremarkable. His hemoglobin was 12 g%, total leucocyte count (TLC) was 7600/µl with 75% polymorphs and 21% lymphocytes, and platelet count was 1.51 lacs/µl. His serum chemistry revealed urea 164 mg%, creatinine 5.9 mg%, sodium 141 mEq/l, and potassium 3.57 mEq/l. Liver function tests, serum complements (C3 and C4), and serum creatine phosphokinase were within normal limits. Urine sediment was bland, and ultrasound of abdomen was also unremarkable. Upper GI endoscopy revealed multiple superficial erosions in esophagus and stomach. He was managed conservatively with H2 blockers and antispasmodics with local application of choline salicylate and benzalkonium chloride gel over buccal mucosa. Two sessions of hemodialysis were given over the first 3 days of admission. After 1 week of admission, serum creatinine was 3.9 mg/dl; hence, a kidney biopsy was performed due to a very slow recovery of renal function. On light microscopy, 13 glomeruli were seen, which were unremarkable. Tubules showed focal epithelial cell denudation and occasional red blood cell casts. There was a mild lymphohistiocytic infiltrate with normal blood vessels [Figure 1]. On immunofluorescence, 10 glomeruli were visualized, which did not show any deposits of IgG, IgA, IgM, C3, or C1q. Hence, a possibility of acute tubular necrosis was kept. His pain abdomen gradually resolved. Renal function and urine output gradually improved during hospital stay. After 12 days of hospital stay, he was discharged with a serum creatinine of 2.03 mg/dl. During follow-up, his