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Unravelling the Mysteries of Childhood Polyuria and Polydipsia: A Case of Senior-Loken Syndrome

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ABSTRACT

Case description: This case report emphasizes the critical importance of early evaluation after neglected or ignored symptoms of polyuria, and polydipsia, in children. We are presenting here a 12-year-old boy, presented with a gradual onset of progressive anorexia and full flared symptoms of advanced chronic kidney disease. Thorough investigations revealed End-stage kidney disease, including severe hyperparathyroidism leading to parathyroid hyperplasia and obstructive sleep apnoea. Detailed enquire revealed that the child had polyuria, and polydipsia from an early age.
Nursing care plan description: Peritoneal dialysis was initiated due to the severity of the condition and the progressive decline in kidney function and finally, renal transplantation was successfully done. Polyuria, Polydipsia, oculomotor apraxia, and whole genome mutation study confirmed the diagnosis of Senior-Loken Syndrome. However, the delay in diagnosis was partly attributed to the parents' ignorance of the significance of the early symptoms, such as polydipsia, polyuria, and involuntary eye movements, during the child's early years.
Evaluation and conclusion: This case highlights the critical importance of vigilant evaluation and prompt management in any child presenting with symptoms of polyuria, polydipsia, and ocular motor abnormalities. Timely detection and intervention play a crucial role in altering the course of Senior-Loken syndrome, effectively delaying the advancement towards end-stage renal disease. Primary care physicians and paediatricians should maintain a high index of suspicion for this genetic kidney disorder in children with such symptoms, as early diagnosis can lead to enhanced results and a higher quality of life for individuals who are affected.

Keywords: Chronic kidney disease, Parathyroid hyperplasia, Obstructive sleep apnoea, Senior-Loken Syndrome, Polydipsia and polyuria in children, Nephronophthisis

INTRODUCTION

Nephronophthisis (NPH), an autosomal recessive cystic kidney disease is one of the common causes of End Stage Renal Disease (ESRD) in children, accounting for approximately 15% of ESRD cases in children worldwide (1). NPH is caused by mutation of more than 20 genes. They produce proteins that are expressed in the centrosomes or primary cilia of renal

epithelial cells. NPHP1 (Nephrocystin-1), NPHP2 (Inversin), NPHP3 (Nephrocystin-3), and NPHP4 (Nephroretinin) coding for Nephrocystin-4 proteins (2). The gene NPHP4, situated on chromosome 1p36, is responsible for encoding a protein known as nephrocystin-4/nephroretinin, consisting of 1,426 amino acids (2). Nephrocystin, produced by the NPHP gene, is localized at the primary cilia-like proteins and the cell-cell junction, as well as the cell-matrix interface. In 10-20% of cases with NPHP, extra-renal symptoms are present. These include retinitis pigmentosa, cerebellar ataxia (Joubert syndrome), bone deformities, mental retardation, liver fibrosis, heart malformations, etc. (1). The initial signs of juvenile NPH include polyuria and polydipsia, typically appearing between 4-6 years ages of (3). Late signs, including anaemia, metabolic acidosis, nausea, anorexia, and growth retardation, as a consequence of the progression of renal insufficiency. ESRD typically manifest at age 13, however, it can potentially happen much later (4). The hallmark clinical features of juvenile NPH include small to normal-sized kidneys with distinguished tubulointerstitial fibrosis and thickened and disrupted tubular basement membranes. In the late stages of the disease, corticomedullary cysts may emerge from distal tubules (5). Anaemia and proteinuria appear with the progression of chronic kidney disease (CKD) and progressive retinal dystrophy leading to retinitis pigmentosa, often missed until the child goes to school. In this case report we present a case of juvenile nephronophthisis with an NPHP4 mutation, also known as Senior-Loken Syndrome. The patient exhibited childhood polyuria, polydipsia, and nystagmus, initially overlooked by parents. Subsequently, the condition progressed, leading to End Stage Renal Disease, and ultimately requiring a kidney transplant.

CASE REPORT

A 12-year-old boy, (figure 1) experiencing loss of appetite, nausea, and lethargy for the last 2-3 weeks was admitted to our hospital for treatment. We found him experiencing pain in his lower limbs with gait instability. When questioned about his daily activities, his father noted he was active and met developmental milestones, but often fell during outdoor play, complaining of leg pain. During the clinical examination, his heart rate was observed to be 100 beats per minute, blood pressure at 120/80 mmHg, respiratory rate at 20 per minute, and oxygen saturation at 97% on room air. His height was 140 cm (25-50th percentile), body weight 37.5 kg (50-75th percentile), with a body surface area of 1.2 square meters. His chest auscultation revealed normal heart sounds and clear breath sounds without crackles or wheezing. Upon examination, he appeared afebrile and pale, with no signs of icterus, cyanosis, clubbing, lymphadenopathy, or edema. His abdomen was soft and palpable without any organomegaly. The neurological evaluation showed nystagmus of both eyes, normal verbal and motor response, and gait instability. An ophthalmologist's opinion was sought. His visual acuity was 6/12, and colour vision was normal. Rapid, fine, horizontal pendular nystagmus was present. Examination of fundi showed narrowing of the retinal arterioles and waxy pallor of optic discs. No previous instances of oliguria, hematuria, edema, headaches, or hearing issues or events of urinary incontinence or recurrent urinary infection in the past. No dysmorphic features were noted. Additionally, no history of chest pain, palpitations, syncope, or seizures. His elder sister has no history of renal disease. His father was an anesthetist, and mother a schoolteacher. They had a non-consanguineous marriage and their son achieved normal developmental milestones. He was born as the second child at 34 weeks, pre-term, with a birth weight of 2.4 Kg. During pregnancy mother didn’t have any significant illness, her blood pressure was normal all through, and no history of any nephrotoxic drug exposure. He had received all the recommended vaccinations and achieved age-appropriate developmental milestones. Surprisingly after routine blood

biochemistry and urinalysis, we discovered that his serum creatinine was 8.19 mg dl^{-1}, with urea at 90.75 mg dl^{-1} and hemoglobin at 6. 1 gm l^{-1}. Potassium registered at 5.1 mmol L^{-1} and sodium at 142 mmol L^{-1}. The thyroid function was within the normal range. Serum calcium measured 10.1 mg dl^{-1}, and phosphorous was 5 mg dl^{-1}. Notably, his parathyroid hormone level was markedly elevated at 668 pg ml^{-1}. Urinalysis showed 2+ RBC with no proteinuria or cast. T-sat was 8% serum iron 28 mg dl^{-1} and Total Iron Binding Capacity (TIBC) was 331. Follow-up ultrasonography of the abdomen revealed bilateral hypodysplastic kidneys with a loss of corticomedullary differentiation, other organs were normal. Echocardiography showed normal heart function with an ejection fraction of 65%. His father was called for a detailed history of the child again. The boy had no history of analgesic abuse or any long-term exposure to any nephrotoxins, no history of prolonged infection, family history of chronic kidney disease, or complications during birth. The only notable history we found was the frequent urge to drink water. He used to drink 10-14 glasses of water a day since childhood and had an increased frequency of micturition, sometimes even every hour and at night peed 4/6 times. They once consulted their pediatrician who said not to worry about those symptoms. To rule out any evidence of collagen vascular disorder we tested complement proteins, C3 and C4 levels which were found to be in normal range. Antinuclear antibody (ANA) and Antineutrophil cytoplasmic antibody (ANCA) profiles, Antistreptolysin O (ASO) titer, and Anti-Deoxyribonuclease B (Anti DNase B) were all negative. His symptoms of anorexia aggravated hence we had to put him on hemodialysis through the right femoral catheter. A renal biopsy couldn't be done as the kidneys were shrunken and had fibrosis. We were still in the darkness to find a definite diagnosis. For anemia, he received injection of Darbepoetin 25 mcg subcutaneously once every two weeks. He was also advised to take intravenous iron at a dose of 500 mg monthly along with oral iron at a dose of 3mg kg^{-1} of body weight. Later, the dose of Darbepoetin was adjusted to a once-weekly dose. His CKD mineral bone disease abnormalities were managed with calcium-based phosphate binders and Calcitriol supplementation. He further developed high blood pressure which was managed by calcium channel blockers. After 4 sessions, he was shifted to peritoneal dialysis (PD) which was initiated with 100 ml for 1 hour, for three cycles. The cycle volume gradually increased to 200 ml in the next session, and he eventually achieved 1000 ml over 3 hours for four cycles. Ultrafiltration was found to be 400-600 ml, and his native urine output which was closely monitored was 1.8-2.0 liters. Within few days, he developed peritonitis and was treated with intravenous antibiotics (Ceftazidime, 500 mg twice a day, and Vancomycin, 1 g once in 5 days). The exit site swab pus culture showed the growth of *Staphylococcus aureus*, which was sensitive to vancomycin. His peritoneal equilibrium test was done using 2.5% PD solution showed his peritoneal membrane to be a low average transporter, so we changed his PD cycle to 1300ml for 4 hours for 2 cycles and 1400 ml for 4 hours for 1 cycle. The cause of his ESRD was still inconclusive. A doctor's review board was called, and the case was discussed. In view of polydipsia and polyuria since childhood, age, gait instability, features of oculomotor apraxia, and dysplastic kidneys, we suspected nephronophthisis, and a whole genome exon analysis was done. The genetic test revealed a mutation in the NPHP4 gene located at Exon 27 (variant c.3659del) and Exon 11 (variant 1302+1_1303-1) _ (1441+1_1442-1) exonic deletion. Both mutations were heterozygous with an autosomal recessive inheritance pattern. A diagnosis of Nephronophthisis type 4, Senior Loken syndrome was made. The boy was advised for a living donor renal transplantation as that was the best modality of treatment for him considering his age. He was discharged from our hospital in stable condition with advice to follow up in our

OPD every month. In a month they came with complaints of excessive daytime sleepiness snoring and parasomnia. Nasopharngo-laryngoscopic examination showed a collapse of the lateral pharyngeal wall. There was severe parathyroid gland hyperplasia which was due to secondary hyperparathyroidism. A polysomnogram (limited channel) was conducted which showed a severe apnea-hypopnea index (AHI) of 123 with severe desaturation and moderate snoring. He was initiated on BiPAP starting with a pressure of 11/6, which was then gradually increased to 15/8. In view of persisting parasomnia polysomnogram with synchronized EEG was done, revealing severe AHI 87.2 and normal EEG. MRI of the brain was done which showed mild cerebral volume loss with bilateral glooming of globus pallidus and hypointensity of calverial bones and vertebral bodies. Oral melatonin was added following which parasomnia episodes decreased. The child was continued on BiPAP 15/8 cm H2O, with which he maintained 90-95% saturation. Kidney transplantation was the only possible solution for him. The boy's blood group was AB positive and they were looking for donors. Unfortunately, being diabetic both parents were not suitable donors. His elder sister, though compatible, was unwilling to donate due to board exams. However, one of his distant aunt, having blood group A positive, altruistically agreed to donate her kidney to him. The patient was HLA-antibody negative, and his immunological risk status was determined to be low risk by donor-specific antibody and panel reactive antibody test. He finally underwent living donor-unrelated renal transplantation with triple immunosuppressant drugs Tacrolimus (0.1mg per day), Mycophenolate sodium (360 BD), and Prednisolone (30 mg once daily continues with tapering doses) along with ATG (3 mg kg^{-1} in 3 divided doses) as an induction agent. During the post-transplant recovery phase, patient's progress was uneventful. Follow-up ultrasound showed good pole-to-pole flow with a normal resistive index in the transplant kidney artery and Hilar artery. He gradually improved and was passing more than 3 liters of urine per day. He received Cytomegalovirus (CMV) prophylaxis with Valganciclovir (450 mg OD) and *Pneumocystis jiroveci* pneumonia (PCP) with trimethoprim-sulfamethoxazole ½ tablet once daily, along with other supportive medications. At discharge, his creatinine level was 1.1 mg dl^{-1}, and he was in a hemodynamically stable condition. The PD catheter was removed on the 25^{th} postoperative day. Over the weeks his Secondary hyperparathyroidism (SHPT) state improved with iPTH touching baseline in 4^{th} week and the need for CPAP at night also was not there. A repeat polysomnography study revealed poor sleep efficiency with disturbed sleep architecture (Figure 2A) and no significant Periodic Limb Movements of Sleep (PLMS) (Figure 2B). Severe oxygen desaturation episodes during the night with nadir SPO2 at 40% (Figure 2C) and only 2 central apneas and 1 hypopnea showing normal AH1 of 0.53 were found from the PLMS study (Figure 3).

Figure 1: Picture of the patient with Senior-Loken Syndrome.

This sleep study was obtained using the channels : REF - ECG, PUL - REF, EMG - REF, EO1 - REF, EO2 - REF, ARF - REF, THR - REF, ABD - REF, BP2 - REF, BP1 - REF, SNR - REF, SPO - REF, BDP - REF, FP2 - F4 , F4 - C4 , C4 - P4 , P4 - O2 , FP1 - F3 , F3 - C3 , C3 - P3 , P3 - O1 , FP2 - F8 , F8 - T4 , T4 - T6 , T6 - O2 , FP1 - F7 , F7 - T3 , T3 - T5 , T5 - O1 , FZ - CZ , CZ - PZ , O1 - O2

Sleep Summary

Lights out:	22:35:14
Lights on:	04:18:21
Total Recording Time (TRT):	342.5 Min
Total Sleep Time (TST):	261.5 Min
Sleep Period Time:	342.5 Min
Sleep Onset:	22:35:14
Sleep Efficiency:	76.4%
Sleep Latency (SL):	.0 Min
REM Latency (RL):	51.0 Min
Wake After Sleep Onset (WASO):	20.0 Min

Stage	Duration	%TST
Awake(W)	81 mins	24%
Stage 1(N1):	0.5 mins	00%
Stage 2(N2):	14 mins	04%
Stage 3(N3):	246.5 mins	72%
Stage REM(R):	0.5 mins	00%

A

Periodic Limb Movement & Other Related Events Summary

Total Limb Movements:	4	Total PLMS:	0
Limb Movement Index:	0.70	PLMS Index:	00.00
Rhythmic Movement Disorder:	0	Total Non-PLMS:	4
REM Sleep Behaviour Disorder:	0	Non-PLMS Index:	00.70
ALMA:	0		
Hypnagogic Foot Tremor:	0	Total Snoring Events:	336
Excessive Fragmentary Myoclanus:	0	Snoring Index:	58.95

B

Oximetry Data, Distribution & Summary

	Awake	REM	NREM	TOTAL
SPO2 Best Case:	98 %	92 %	98 %	100 %
SPO2 lowest Case:	89 %	92 %	87 %	40 %
SPO2 Average Case:	93.2 %	92. %	92.6 %	93.2 %
Time spent below 100% (min)	37.3 (10.87%)	0.5 (00.15%)	74.9 (21.82%)	235.6 (68.67%)
<95% (min)	29.1 (08.49%)	0.5 (00.15%)	60.4 (17.61%)	170.6 (49.73%)
<90% (min)	1.9 (00.56%)	0.0 (00.00%)	7.8 (02.26%)	13.9 (04.05%)
<85% (min)	0.0 (00.00%)	0.0 (00.00%)	0.0 (00.00%)	2.8 (00.82%)
<80% (min)	0.0 (00.00%)	0.0 (00.00%)	0.0 (00.00%)	2.0 (00.58%)
<70% (min)	0.0 (00.00%)	0.0 (00.00%)	0.0 (00.00%)	1.9 (00.54%)
<60% (min)	0.0 (00.00%)	0.0 (00.00%)	0.0 (00.00%)	1.0 (00.29%)
<50% (min)	0.0 (00.00%)	0.0 (00.00%)	0.0 (00.00%)	1.0 (00.29%)
Fail/Probe Disconnected (min)	43.7 (12.74%)	0.0 (00.00%)	46.6 (13.59%)	107.5 (31.34%)
Episodes with desat >= 02	10	0	19	29
>= 05	3	0	10	13
>= 15	0	0	0	0
Desaturation Index (#/hour)	**1.8**	**0.0**	**5.1**	**6.8**
Maximum Desat value	**6**	**0**	**8**	**8**
Maximum Desat Dur (sec)	**2674**	**0**	**1054**	**2674**

C

Figure 2: Sleep summary of the patient from Poly Somnographic Study (A), Periodic Limb movement and other related events summary from Poly Somnographic Study (B), Oximetry data, distribution and summary from Poly Somnographic Study (C)

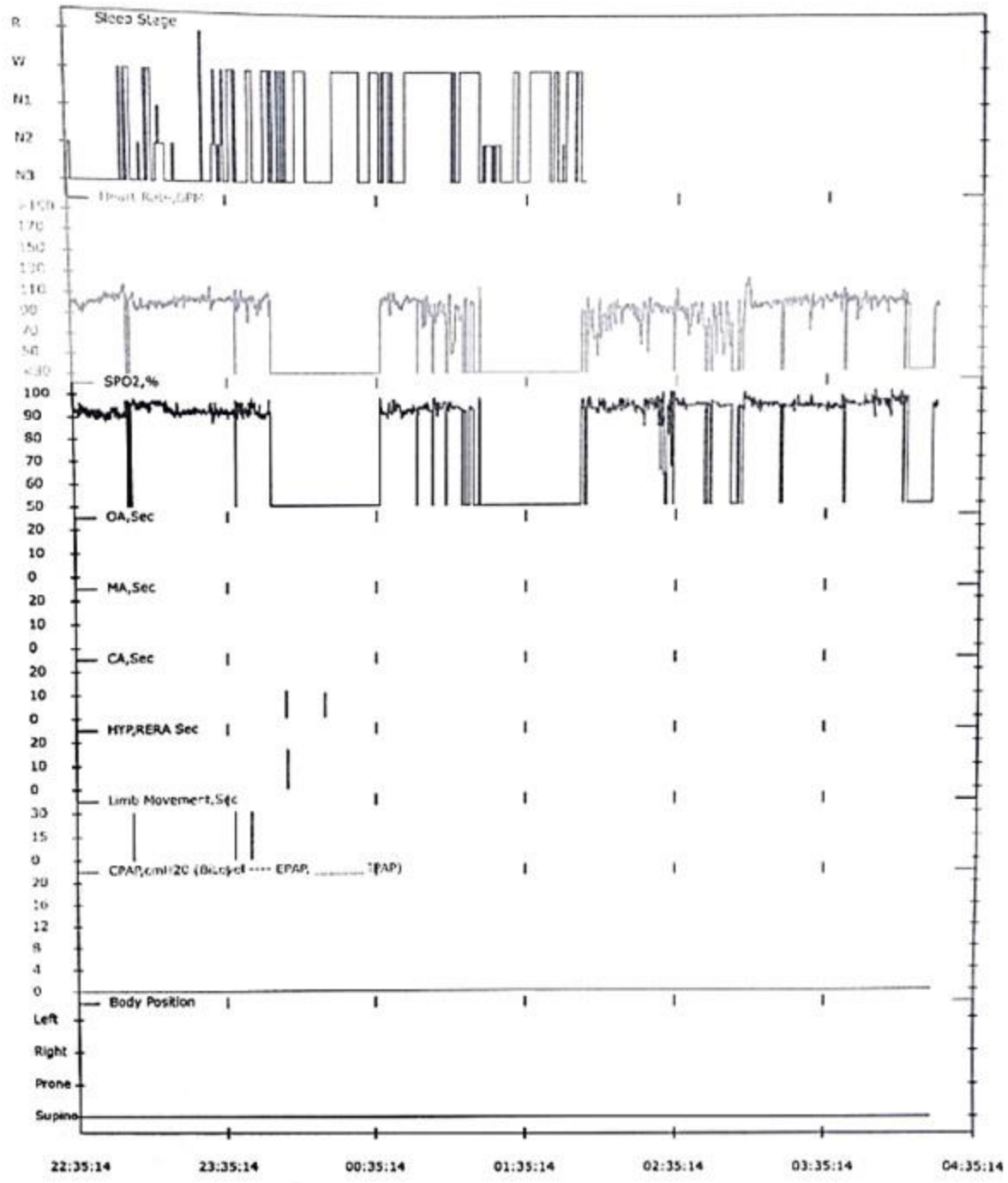

Figure 3: Night Hypnogram from Poly Somnographic Study

DISCUSSION

Familial Juvenile Nephronophthisis, manifests as a gradually advancing tubulointerstitial kidney disorder, especially in children characterized by an autosomal recessive pattern of inheritance (6). Although previous studies (7) reported consanguineous marriage among the parents might be a cause of this genetically recessive inheritance, in our case, the parents of the patient had no consanguineous marriage and had no family background of renal disorders. In this case, the early signs were polyuria, polydipsia, and enuresis like the previous studies which are frequently disregarded, especially when a urinalysis appears relatively normal and there is an absence of proteinuria, azotemia and hypertension (8). Polydipsia and polyuria are induced by sodium loss and impaired urine concentrating capacity in the

renal tubules and collecting ducts (9). While less common, some individuals may also exhibit features such as hypoplasia (underdevelopment) or agenesis (total lack) of the cerebellar vermis, which are frequently detected by the "molar tooth" sign on axial brain MRI scans (10); varying degrees of developmental delays/mental retardation; difficulty coordinating voluntary muscle movements or ataxia; impaired visual function, including nystagmus, photophobia, hyperopia, and slow pupillary responses; and vascular hypertension due to affected kidneys (11). Apart from nystagmus and later hypertension, our patient did not display any of these characteristics. However, the fundoscopic examination of the patient in this present case showed narrowing of retinal arterioles and waxy pallor of optic discs causing juvenile onset nephrophthisis with retinal involvement (12).

In the present case, the genetic test revealed a mutation in the NPHP4 gene confirming the diagnosis of nephronophthisis type 4, Senior Loken Syndrome. It is located on chromosome 1p36 and encodes a protein (1426 amino acids) called nephrocystin 4 or nephroretinin, expressed in the kidney, brain, and eye (2). Hemachandar (13) reported that in the Senior-Loken syndrome, the retinal lesion may be typical like Retinitis pigmentosa with night blindness progressing to day blindness due to the pigmentation of the retina's bone spicules or maybe as severe as Leber amaurosis, congenital retinal dystrophy causing early-onset blindness, nystagmus, extensive atypical retinal pigmentation, pallor of the optic disc, and early and full extinction of the electroretinogram (ERG) (14). Initially, our patient exhibited no significant symptoms, no hyponatremia, and maintained an active lifestyle without any health complaints. Despite being active in school, he experienced frequent urination and excessive thirst, involuntary movement of eyes, and gait instability likely due to nystagmus which his parents initially overlooked. However, at the age of 12, the symptoms intensified, including anemia, anorexia, and high blood pressure along with uremia, and secondary hyperparathyroidism leading to obstructive sleep apnoea (OSA), and renal replacement therapy became the only hope for a favorable outcome.

Unfortunately, there is no definitive treatment for Senior-Loken Syndrome. Management primarily emphasizes providing supportive care to slow down the advancement of CKD, avert complications, and promote the overall growth of the patient. Diagnosing renal tubular cell atrophy with medullary cyst formation and renal interstitial fibrosis are hallmarks of nephronophthisis in renal histopathology (15). The oculomotor apraxia with features of retinitis pigmentosa along with polyuria and polydipsia in our patients were crucial indicators in the identification of Senior-Loken syndrome. Increased knowledge about this syndrome could have aided in the early detection of renal illness. On a positive note, renal insufficiency does not recur after kidney transplantation, making kidney transplantation the established and most effective treatment option for the survival of the child.

CONCLUSION

In conclusion, our case report highlights the significance of early recognition of often neglected symptoms like polyuria and polydipsia in childhood, as they may be indicative of underlying genetic disorders such as Nephronophthisis. Kidney transplantation remains the most effective treatment for NPH patients who progress to end-stage renal disease as the disease does not recur after transplantation. Prompt identification, genetic testing, and proper intervention are crucial for delivering the best possible care to individuals diagnosed with Nephronophthisis. Enhancing awareness among pediatricians, primary care physicians, and parents regarding the importance of these symptoms can result in early diagnosis, thereby increasing the likelihood of improved outcomes and a better quality of life for individuals affected by them.

Declaration by Authors

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