by immunohistochemistry. The patient was prescribed an angiotensin receptor blocker and is being followed up.

Organized deposit disease must be considered in the differential diagnosis for any patient presenting with significant proteinuria, irrespective of age, gender, or systemic disease. Clinical history of comorbid conditions, light microscopy, immunofluorescence, and electronmicroscopy together play an important role in the diagnosis of this entity.

Conflicts of interest

There are no conflicts of interest.

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Hub and Spoke Model for Kidney Care – From Prevention to Treatment

Dear Editor,

India adds 200,000 new end-stage kidney disease (ESKD) patients to the existing pool annually. The country has seen a revolution in dialysis with 1452 dialysis centers in 748 out of 806 districts with 9902 machines under the Pradhan Mantri National Dialysis Program (PMNDP) model.[1] There are approximately 2500 nephrologists, with the majority practicing in urban areas. To address the shortage of nephrologists in rural peripheral districts, the state of Telangana has started a hub and spoke model of dialysis where the hub center, usually a teaching hospital in the city, monitors the spoke dialysis centers in the surrounding districts within a radius of 200–250 km [Table 1]. A local medical officer trained at a hub center is responsible for managing each spoke center under the guidance of the hub nephrologist.[2] This has markedly improved the quality of dialysis. Telemonitoring facilities have been initiated between the hub and spoke centers in some states, which is commendable.

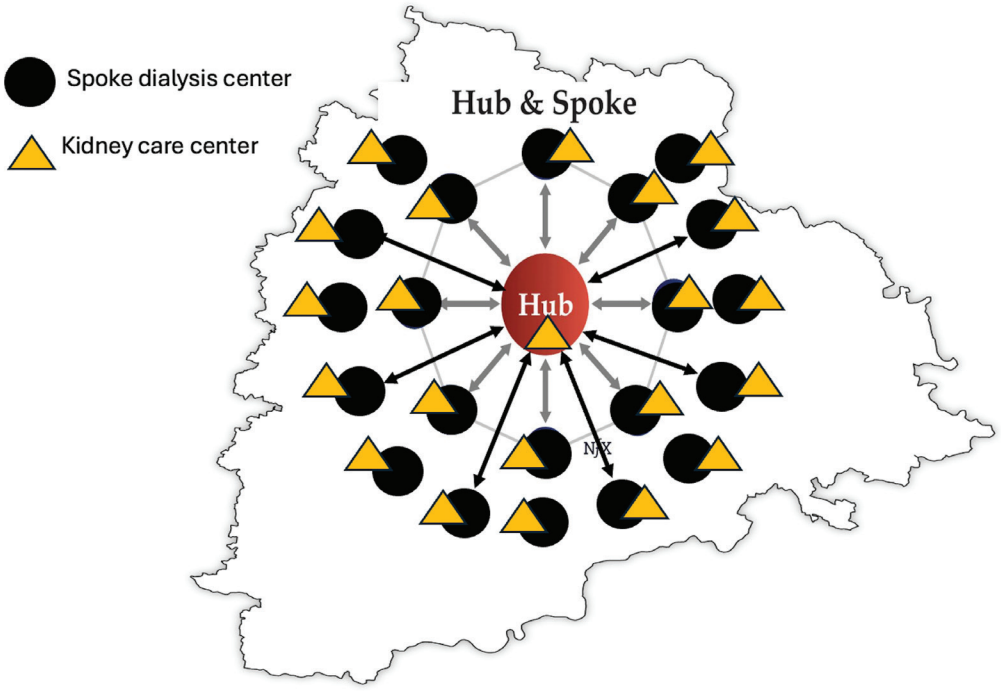

Figure 1: Hub and spoke model of kidney disease prevention and kidney replacement therapy (KRT).

Table 1: Hub and spoke model with Osmania Medical College and Hospital hub center

Name of district	Name of the facility	Distance from Hub in kilometers	Total number of functional dialysis machines	Number of new patients registered (March 2024)	Number of dialysis sessions held (March 2024)	Total patients who came for dialysis (March 2024)	Cumulative number of dialysis sessions held
Bodhan	Bodhan	205	5	1	497	46	4345
Hyderabad	Nampally	5	5	3	338	30	2590
	Golconda	11	5	1	265	26	2096
	Malakpet	5	10		997	82	7506
	Osmania	0	18	20	2318	247	20063
Kamareddy	Bichkunda	170	5	1	189	17	3817
	Yellareddy	158	5		309	26	2771
	Banswada	167	10	1	472	37	4419
	Kamareddy	113.6	9		560	54	5246
Medchal	Ghatkeshar	28	5	2	646	55	5850
Nalgonda	Nagarjuna Sagar	156	5	1	260	24	2606
	Devarakonda	107	5		452	37	5295
	Miryalaguda	145	5	1	611	71	5413
	Nalgonda	102	10	5	1145	109	10144
Nizamabad	Armoor	206	5	1	462	43	6490
	Dichpally	220	5	1	175	15	625
	Nizamabad	216	15		976	84	6381
Suryapet	Kodad	182	5	3	472	47	4592
	Huzurnagar	191	5	1	523	51	4811
	Suryapet	139	5	1	515	53	4662
Yadadri Bhongir	Choutuppal	50	5		424	41	5943
	Bhongir	80	5	1	551	46	6289
			155	46	13331	1259	124715

However, kidney replacement therapy (KRT), that is, dialysis or transplant, cannot match the ever-increasing burden of ESKD. KRT imposes a huge burden on the country's economy. The key should be prevention of CKD and/or its progression using renin angiotensin system inhibitors, sodium glucose transporter inhibitors, GLP1Ra, and finerenone.[3] Empagliflozin delays kidney failure, and the need for KRT from 1.9 years, if it is initiated at eGFR 20 mL/min/1.73 m^2, to 26.6 years, if initiated when eGFR is 85 mL/min/1.73 m^2.[4] This can happen if CKD is identified early by screening.[5] A practical solution for screening at grassroots level could be utilizing the already existing infrastructure of dialysis units in each district as "Kidney Care Centers" [Figure 1]. This does not need any infrastructure, and a medical worker at the dialysis center can be in charge of these. A starting point could involve screening the relatives of all patients who come for dialysis. Later, these centers can be made beacons for running community screening program in that district. MBBS students at the district medical colleges can be posted on rotation at these centers and can be tasked with door-to-door screening for CKD in that district. This will help inculcate interest in research in the young students.

The hub and spoke model for KRT as well as primary care can address the problem of shortage of nephrologists in peripheral centers and provide an opportunity for implementing early kidney disease detection programs in the community.

Conflicts of interest

There are no conflicts of interest.

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Novel Variations in KIRREL1 Gene and Infantile Onset Nephrotic Syndrome

Dear Editor,

More than 58 monogenic genes associated with steroid resistant nephrotic syndrome (SRNS) have been described to date.[1] Kin of IRRE-Like protein 1 (KIRREL1) has been described as an nephrotic syndrome type 1 (NPHS1) like Ig superfamily cell adhesion molecule.[2] We report two heterozygous missense mutations in the KIRREL1 gene leading to SRNS in a child with infantile nephrotic syndrome.

A 2-year-old male, a case of infantile-onset SRNS born out of a nonconsanguineous marriage, diagnosed at the age of 11 months was reported positive for the KIRREL1 gene mutation on exons 12 and 15. A mislocalization of both KIRREL1 mutants c.1513G>A (p.Ala505Thr) and c.1918C>T (p.Arg640Cys) was noted, which have not been reported previously as pathogenic variants. This was also confirmed by Sanger sequencing, as seen in Figure 1. Given the condition we found in our case of two different mutations in the same gene is classified as compound heterozygosity, it is difficult to ascertain any definite relationship between our findings and the clinical presentation. The child was started on Tacrolimus and ACE-I inhibitors and discharged. On 1-year follow-up, the child is in complete remission. There were no extrarenal manifestations or syndromic features in the child.

A direct interaction between NPHS1 and KIRREL1 because of their co-localization at the slit diaphragm has been described.[3] KIRREL1 is necessary for the rearrangement of the actin cytoskeleton of the slit diaphragm.[4] Previously, mutations in the KIRREL1 gene from two unrelated families were reported in children who presented at the ages of 5 and 14 with SRNS, respectively, which were p.Arg440Cys and p.Ser573Leu.[1] Patients with this mutation achieved complete remission upon treatments with tacrolimus,

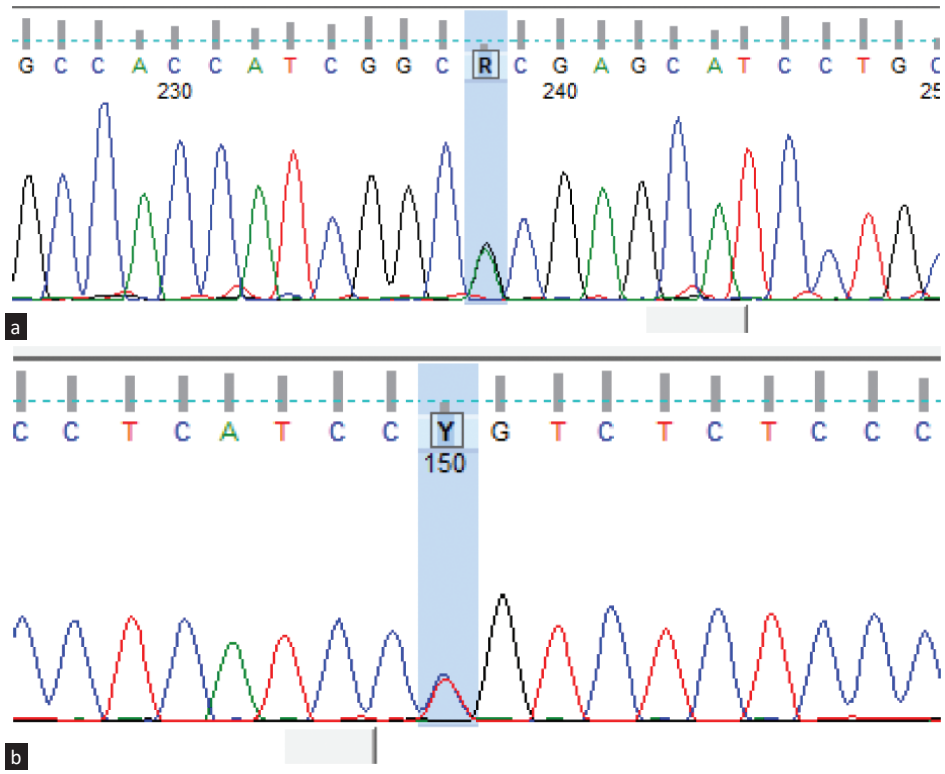

Figure 1: (a) Sanger sequencing data (electropherogram) shows a nucleotide change at c.1513G>A (p.Ala505Thr) in the KIRREL1 gene. (b) Sanger sequencing data (electropherogram) shows a nucleotide change at c.1918C>T (p.Arg640Cys) in the KIRREL1 gene.