

Supplementary Table S1. Barriers and facilitators described according to the Practical, Robust Implementation and Sustainability Model (PRISM) domains with corresponding implementation strategies according to the Expert Recommendations for Implementing Change (ERIC) taxonomy.

PRISM domains	Barriers and Facilitators	Implementation strategies
Intervention <i>Organization perspective</i>	No existing genetic service in the department	<i>Stakeholder inter-relationships</i> (1) Identified and prepared champions to form the implementation team <i>Support clinicians</i> (1) Created new clinical teams for genetic service
	No existing clinic where genetic service could be conducted	<i>Infrastructure</i> (1) Changed physical structure and arranged clinic room for genetic kidney disease clinic
	Implementation team lacked knowledge on performing genetic counselling and testing	<i>Evaluative and iterative strategies</i> (1) Geneticists in the multi-disciplinary Renal Alliance for Precision Diagnosis in Singapore (RAPIDS) collaboration audited and provided feedback to genetic nephrologists (2) Regular re-examination by geneticists and clinicians to refine patient selection, variant curation and approaches to resolution for suspicious variants of uncertain significance <i>Stakeholder inter-relationships</i> (1) Developed academic partnerships – research project with geneticists (2) Captured and shared local knowledge (3) Organized clinician implementation team meetings (4) Established collaboration with on-campus Ocular Genetics Clinic to evaluate patients with monogenic ophthalmological conditions and concomitant renal involvement <i>Train and educate stakeholders</i> (1) Geneticists conducted educational meetings for genetic nephrologists (2) Geneticists conducted ongoing training for genetic nephrologists <i>Interactive assistance</i>

		(1) Geneticists provided clinical supervision to implementation team
Intervention <i>Recipient perspective</i>	Nephrologists lack awareness about genetic counselling and testing	<i>Train and educate stakeholders</i> (1) Implementation team conducted educational meetings to nephrologists regarding referral criteria (2) Developed educational materials (3) Distributed educational materials
	Nephrologists uncertain about patients' suitability for genetic counselling and testing	<i>Support Clinicians</i> (1) Genetic nephrologists provided ongoing consultation via e-mail and secure healthcare messaging portal
	Patients lack awareness about genetic counselling and testing	<i>Engage patients</i> (1) Increased demand via advertisement (2) Increased demand via collaboration with the ocular genetics clinic
Implementation and Sustainability Infrastructure	Costly genetic test – no government subsidy or insurance cover	<i>Financial strategies</i> (1) Accessed new funding – genetic counselling and testing funded by research grant hence patients did not incur any out-of-pocket charges

Supplementary Table S2. The intervention and implementation strategies according to the Template for Intervention Description and Replication (TIDieR).

Brief name	Provision of genetic kidney disease service (GKS) for evaluation of suspected glomerular diseases by genetic nephrologists.
Why	There is a need to increase genetic testing for early diagnosis and management of adults with genetic glomerular kidney disease. By allowing genetic nephrologists to provide genetic services via standardized workflows and within a pre-defined scope, we aim to overcome the barriers that contribute to under-diagnosis and suboptimal management of genetic glomerular diseases.
What	Genetic nephrologists participated in a nation-wide multi-disciplinary team collaborative “Renal Alliance for Precision Diagnosis in Singapore” (RAPIDS). They received training in genetic counselling, results interpretation and variant curation. The training programme included recorded lectures, videos of simulated counselling encounters and written guides for pre-test and post-test counselling. In addition, a series of group training sessions via online video conferencing were conducted by geneticists and genetic counsellors to address knowledge gaps. The first few patient counselling sessions (two pre-tests and one post-test for each type of result: positive, negative and VUS) performed by genetic nephrologists were directly supervised by geneticists or genetic counsellors, who gave immediate feedback on the genetic nephrologist’s communication. Genetic nephrologists used templates from RAPIDS to (a) document patient counselling in electronic medical record (EMR); (b) obtain and document informed consent for genetic testing; (c) order genetic tests; and (d) reply to referring physicians about the genetic test results and clinical recommendations. Posters in the nephrology ambulatory clinics a layperson educational booklet provided information on genetic testing to patients. Posters in the nephrology ambulatory clinics and an introductory email provided information on the referral process to non-genetic nephrologists.
Who	Coordinators assisted in scheduling appointments and preparing redacted clinical phenotype and genetic results for genomic board meetings. Genetic nephrologists provided the pre- and post-test counselling to patients and clinical recommendations to referring non-genetic nephrologists. Clinical geneticists, genetic counsellors, and laboratory scientists provided guidance on results interpretation and variant curation during monthly genomic board meetings.
How	Patients with suspected monogenic glomerular diseases were referred by their nephrologists to the genetic nephrologists, who then assessed the indications for genetic testing and performed pre-test counselling at the genetic kidney clinic. Patients who agree to genetic testing had blood samples obtained for testing according to the indications. Genetic results were discussed at monthly multi-disciplinary genomic board meetings via online video conferencing to plan further genetic and clinical evaluation.

	Genetic nephrologists conducted the post-test counselling to explain the results to the patients in-person within 2-3 months after testing. A memo on results and clinical recommendations would be provided to the referring non-genetic nephrologist.
Where	Singapore General Hospital [SGH]
When and How much	Genetic nephrology clinics were held every 1-2 weeks. Implementation team meetings were held once a month. Genomic board meetings are held once a month.
Tailoring	As kidney cystic diseases can be phenocopies of glomerular diseases, the implementation team added a 41-gene panel that included kidney cystic disease genes to the 84-gene panel for glomerular diseases for patients with kidney cysts but no severe enough to explain the degree of haematuria, proteinuria or CKD. Additionally, whole exome sequencing test will be added for patients with CKD of unknown aetiology to better capture the breadth of possible genetic causes.
Adaptations	The SingHealth Duke-NUS Genomic Medicine Centre facilitated service and expertise sharing hence the implementation team was able to integrate genetic counsellors from an established pediatric clinical genetic service into our GKS as our case volume and complexity increased over time. We formed clinical collaborations and workflows for referrals with the Singapore National Eye Center's Ocular Genetics Clinic (OGC). The OGC referred patients with suspected genetic kidney and eye disease to the GKS for clinical testing, kidney function assessment and management, including some with pathogenic variants on research testing. The GKS referred patients with suspected and confirmed Alport's disease to the OGC for eye evaluation. The implementation team conducted continuing medical education (CME) in the form of case discussions at Peer Review Learning (PRL) sessions with practising nephrologists. Since our institution is the sponsor site for the healthcare clusters' senior residency program for nephrology trainees, the implementation team introduced lectures on monogenic kidney diseases and genetic counselling into the nephrology training program to increase awareness and genetic literacy.
How well	During the study period, 186 patients were referred by 32 nephrologists and nephrology trainees. 109 patients received pre-test counselling and 101 underwent genetic testing.

Supplementary Table S3. Implementation study methodology.

Implementation process model and setting

We conducted an implementation study with a post-design¹ between March 2023 and November 2024 at the Department of Renal Medicine, Singapore General Hospital (SGH). At the time of this study, this department comprised 35 nephrologists and 20 nephrology trainees who cared for more than 16,000 adults with kidney diseases.² Our trainees undertake a 3.5-year training programme to be accredited as nephrologists and had the experience of post-graduate year 5 and above. Despite having clinical genetic services within our healthcare cluster (Singapore Health Services), an in-house GKS would be synergistic in advancing precision medicine. Thus, a GKS led by genetic nephrologists was created. Genetic nephrologists first received training, supervision, and feedback to increase genetic knowledge and skills in the RAPIDS multidisciplinary collaboration.³ In addition to the potential barriers to genetic testing postulated by RAPIDS,³ we also established our center's specific barriers, facilitators and adapted implementation strategies during an iterative process that considered feedback from our implementation team, referring nephrologists and patients as established in **Supplementary Table S1**.^{4,5} The PRISM process model was relevant since our study aimed to translate low-use evidence-based interventions to routine clinical practice and the domains are hypothesized to determine implementation outcomes.⁶ The intervention is summarized in **Supplementary Table S2** according to the Template for Intervention Description and Replication (TIDieR).⁷

Implementation outcomes

Adoption among nephrologists was defined as the number and proportion of nephrologists and nephrology trainees within SGH who referred eligible patients with suspected monogenic glomerular disease to the GKS. Adoption among patients was defined as number and proportion of referred patients who had genetic tests performed. Utility was defined as the proportion of patients who had confirmed genetic diagnoses among those who were tested. We used the Normalization of Complex Interventions – Measure Development (NoMAD) survey to assess nephrologists' intent to sustain the service. The NoMAD is grounded in the Normalization Process Theory that explained the assimilation of practice modifications required for implementation activities.⁸ The 23-item instrument had good face validity, construct validity and internal consistency for assessing staff perceptions of factors relevant to implementing changes in work practices and covered the aspects of coherence insight (staff's comprehension of the purpose and perceived value of the service), cognitive participation (engagement and motivation to adopt the service), collective action (integration into existing practice), and reflexive monitoring (perceived impact on patient care and contribution to enhance existing practice).^{8,9} We conducted the self-administered, anonymized, online NoMAD survey among non-genetic nephrologists and nephrology trainees in SGH in February 2024, 12 months after the start of implementation. Educational sessions were held in March and May 2024 to address the concerns identified by NoMAD.

Participants

All referrals were screened for eligibility by a genetic nephrologist. The GKS coordinators contacted eligible patients for their agreement to schedule a pre-test genetic counselling session. The inclusion and exclusion criteria are detailed in **Supplementary Table S4**. Patients who consented to genetic testing post-counselling had their blood sampled for testing. All participants provided written informed consent for study participation and genetic testing. The study was conducted in accordance with the Declaration of Helsinki and approved by the National Healthcare Group's Domain Specific Review Board (DRSB Number 2022/00108).

Genetic Evaluation, Result Analysis and Costs

Pre-test counselling was initially performed by genetic nephrologists³. These were initially performed ad-hoc, subject to logistical constraints like availability of consultation rooms. Several months after implementation, the patient load increased and regular clinics were established (4-5 sessions monthly). To manage the increase in workload and complexity of cases, the implementation team collaborated with genetic counsellors in the KK Women's and Children's Hospital (KKWCH) to provide on-site genetic counselling services to ensure timely service delivery and return of results. Genetic counsellors facilitated a broader breadth of discussion topics with patients and families that would not have been possible with nephrologists alone, such as reproduction options and incidental findings with whole exome sequencing (WES). The collaboration reduced the need for additional referrals to a separate genetic service.

The genetic tests performed included an 84-gene panel from an exome backbone for patients with isolated glomerular phenotypes. As there may be phenocopies due to cystic kidney diseases, a 41-gene panel for cystic kidney disease was added for patients with kidney cysts but not severe enough to solely explain the degree of haematuria, proteinuria or CKD. For patients with CKD of unknown causes, WES was performed. The genetic tests were performed by a clinically accredited laboratory. The genes for targeted exome analysis are listed in **Supplementary Table S5**. The turnaround time was 6–8 weeks. Variants were classified according to American College of Medical Genetics and Genomics guidelines.¹⁰ A genetic report was generated for all patients who underwent genetic testing. The de-identified clinical information was discussed at monthly multi-disciplinary genomic board meetings. All participants had their genomic results along with post-test recommendations returned by genetic nephrologists or genetic counsellors. **Figure 1** summarized the patient journey. Research funding paid for the genetic tests, cascade testing and segregation analysis.

Statistical Analysis

Statistical analyses were carried out using R (R version 4.4.2, <https://www.R-project.org/>). In the analysis for the outcome of adoption, we included referrals from nephrologists in our own institution and excluded those from other institutions. For the outcome of intent to sustain the service, we included the non-genetic nephrologists in our center but excluded the implementation team (including the 6 genetic nephrologists). In the analysis for clinical utility of genetic testing, we included all patients who had genetic tests performed and excluded those who were uncontactable after referral and those who declined genetic testing. Categorical variables were reported as numbers (percentages). Non-normally distributed continuous variables were described as median (interquartile range; IQR) and compared using the Mann-Whitney U test for independent groups and the Wilcoxon signed-rank test for paired groups. Statistical significance was set at $p < 0.05$.

We reported this implementation study according to the Standards for Reporting Implementation Studies (StaRI) checklist.¹¹

References

1. Smith JD, Hwang S, Birken SA, Melvin CL, Rohweder CL, Smith JD. Designs and methods for implementation research: Advancing the mission of the CTSA program. *J Clin Transl Sci*. 2020;4(3):159-167. doi:10.1017/CTS.2020.16
2. Huang Z, Ng LC, Mok I, Tan CS, Lim CC. Implementing the Nurse-led Optimization of Volume and blood pressure – Enabling at multi-Levels using TechnologY (NOVELTY) program for Chronic Kidney Disease: a prospective cohort study. *Nephron*. Published online February 3, 2025:1-18. doi:10.1159/000543948

3. Lim C, Lim RS, Choo J, et al. Clinical implementation of Nephrologist-led Genomic Testing for Glomerular Diseases in Singapore: Rationale and Protocol. *Am J Nephrol*. Published online December 3, 2024;1-20. doi:10.1159/000542942
4. Powell BJ, Waltz TJ, Chinman MJ, et al. A refined compilation of implementation strategies: Results from the Expert Recommendations for Implementing Change (ERIC) project. *Implementation Science*. 2015;10(1):1-14. doi:10.1186/S13012-015-0209-1/TABLES/3
5. Feldstein AC, Glasgow RE. A practical, robust implementation and sustainability model (PRISM) for integrating research findings into practice. *Jt Comm J Qual Patient Saf*. 2008;34(4):228-243. doi:10.1016/S1553-7250(08)34030-6
6. McCreight MS, Rabin BA, Glasgow RE, et al. Using the Practical, Robust Implementation and Sustainability Model (PRISM) to qualitatively assess multilevel contextual factors to help plan, implement, evaluate, and disseminate health services programs. *Transl Behav Med*. 2019;9(6):1002-1011. doi:10.1093/TBM/IBZ085
7. Hoffmann TC, Glasziou PP, Boutron I, et al. Better reporting of interventions: template for intervention description and replication (TIDieR) checklist and guide. *BMJ*. 2014;348. doi:10.1136/BMJ.G1687
8. Rapley T, Girling M, Mair FS, et al. Improving the normalization of complex interventions: Part 1 - Development of the NoMAD instrument for assessing implementation work based on normalization process theory (NPT) 17 Psychology and Cognitive Sciences 1701 Psychology. *BMC Med Res Methodol*. 2018;18(1):1-17. doi:10.1186/S12874-018-0590-Y/TABLES/8
9. Finch TL, Girling M, May CR, et al. Improving the normalization of complex interventions: Part 2 - Validation of the NoMAD instrument for assessing implementation work based on normalization process theory (NPT) 17 Psychology and Cognitive Sciences 1701 Psychology. *BMC Med Res Methodol*. 2018;18(1):1-13. doi:10.1186/S12874-018-0591-X/TABLES/9
10. Richards S, Aziz N, Bale S, et al. Standards and guidelines for the interpretation of sequence variants: a joint consensus recommendation of the American College of Medical Genetics and Genomics and the Association for Molecular Pathology. *Genet Med*. 2015;17(5):405-424. doi:10.1038/GIM.2015.30
11. Pinnock H, Barwick M, Carpenter CR, et al. Standards for Reporting Implementation Studies (StaRI) Statement. *BMJ*. 2017;356. doi:10.1136/BMJ.I6795

Supplementary Table S4. Inclusion and Exclusion Criteria for Probands and Types of Genetic Tests.

Inclusion Criteria	Type of genetic test
1. Persistent glomerular hematuria (including gross hematuria) - Current active hematuria AND At least two prior separate episodes - Glomerular cause: Dysmorphic RBCs present, or glomerular haematuria is likely. - Kidney biopsy is not necessary. 2. Persistent A2 or A3 albuminuria - More than 1 occasion - May or may not have haematuria currently or previously. - No secondary causes of albuminuria - Kidney biopsy is not necessary. 3. Primary nephrotic syndrome with partial or no response to high-dose glucocorticoid monotherapy, with or without partial or no response to calcineurin inhibitors; OR - Initial-onset steroid resistance • Partial response after Prednisolone dose of $\geq 1\text{mg/ kg/ day}$ (max 60mg) (or steroid equivalent*) for 8-12 weeks or after IV Methylprednisolone pulses OR • No complete response after prednisolone dose of $\geq 1\text{mg/ kg/ day}$ (max 60mg) (or steroid equivalent*) for > 12 weeks - No complete response after adequate CNI treatment • Persistent A3 proteinuria AND • Adequate trough levels for ≥ 3 months (Cyclosporine level ≥ 100 ng/ml or Tacrolimus level ≥ 4 ng/mL) - Kidney biopsy must have been performed. - Patient must have never shown complete response to prednisolone given as a single immunosuppressive agent 4. Apparent “familial” IgA nephropathy - Biopsy-proven IgAN in both proband and first-degree family member - Biopsy-proven IgAN in proband AND presence of haematuria/ A3 albuminuria in first-degree family member - Haematuria/A3 albuminuria in proband and biopsy-proven IgAN in first-degree family member	Glomerular panel
5. Any of the above indications (1-4) with presence of >1 kidney cyst or kidney imaging that may suggest presence of renal cystic diseases	Glomerular and cystic panel

6. Chronic kidney disease Stage G3 to 5 (including dialysis and transplant) without obvious cause	Glomerular or Glomerular-Cystic Panel
7. Rapid kidney function decline (eGFR decline >5 ml/min/1.73 m ² per year) without obvious cause	Whole exome sequencing if necessary
8. Any of the above indications with personal or family history suspicious of mitochondrial disease (e.g. lactic acidosis, maculopathy, neurosensory hearing loss, early onset diabetes mellitus)	Whole exome sequencing with mitochondrial genes
Exclusion Criteria 1. Late-onset steroid resistance, or prior complete remission with steroid treatment as single immunosuppressive agent. 2. Confirmed or suspected secondary causes of glomerular diseases e.g. • Vasculitis e.g. lupus nephritis (positive anti-dsDNA), ANCA-associated vasculitis, • IgA nephropathy (except for familial IgAN) • Membranous nephropathy, Membranoproliferative GN, diffuse proliferative GN, • C3 glomerulopathy (serum C3 <80 mg/dL or <0.8 g/L) • Multiple myeloma, cryoglobulinemia • Infection (eg HIV, Hepatitis B or C, post infectious) or drug-related • glomerulonephritis • Secondary FSGS 3. Other co-morbidities that can explain the kidney phenotype, e.g. • Diabetes mellitus • Morbid obesity (body mass index > 30 kg/m²) • Cardiac, endocrinological, vascular or other conditions • Low birth weight < 2kg or small for gestational age, ex-prematurity < 32 weeks 4. Complex phenotypes involving multi-system malformations which may suggest complex genetic alterations outside the scope of current study. • Syndromic features are not absolute exclusion criteria. 5. Tubulointerstitial diseases (ADTKD, medullary cystic disease) 6. Cystic kidney disorders 7. Congenital anomalies of kidney and urinary tract (e.g. dysplasia, hypoplasia, agenesis, PUJO, PUV, VUJO, pelvic kidney, MCDK) 8. Scarred kidney 9. Nephrectomy or kidney surgery 10. Urinary / renal calculi	

Supplementary Table S5. List of genes tested for suspected inherited glomerular kidney disease.

No.	Gene	CIP Panel	MIM Entry	MIM Phenotype	MIM Phenotype Number	Mode of Inheritance
1	ACTN4	Glomerular; Glomerular and Cystic	604638	Glomerulosclerosis, focal segmental, 1	603278	AD
2	ADAMTS13	Glomerular; Glomerular and Cystic	604134	Thrombotic thrombocytopenic purpura, hereditary	274150	AR
3	AGXT	Glomerular; Glomerular and Cystic	604285	Hyperoxaluria, primary, type 1	259900	AR
4	ALG8	Glomerular and Cystic	608103	Congenital disorder of glycosylation, type 1h Polycystic liver disease 3 with or without kidney cysts	608104 617874	AR AD
5	AMN	Glomerular; Glomerular and Cystic	605799	Imerslund-Grasbeck syndrome 2	618882	AR
6	ANKS6	Glomerular and Cystic	615370	Nephronophthisis 16	615382	AR
7	ANLN	Glomerular; Glomerular and Cystic	616027	Focal segmental glomerulosclerosis 8	616032	AD
8	APOL1	Glomerular; Glomerular and Cystic	603743	{Glomerulosclerosis, focal segmental, 4, susceptibility to}	612551	AD
9	ARHGAP24	Glomerular; Glomerular and Cystic	610586	Focal segmental glomerulosclerosis	610586	?
10	ARHGDIA	Glomerular; Glomerular and Cystic	601925	Nephrotic syndrome, type 8	615244	AR
11	BICC1	Glomerular and Cystic	614295	{Renal dysplasia, cystic, susceptibility to}	601331	AD

12	C3	Glomerular; Glomerular and Cystic	120700	{Hemolytic uremic syndrome, atypical, susceptibility to, 5} {Macular degeneration, age- related, 9} C3 deficiency	612925 611378 613779	AD ? AR
13	CD2AP	Glomerular; Glomerular and Cystic	604241	Glomerulosclerosis, focal segmental, 3	607832	?
14	CD46	Glomerular; Glomerular and Cystic	120920	{Hemolytic uremic syndrome, atypical, susceptibility to, 2}	612922	AD, AR
15	CEP164	Glomerular and Cystic	614848	Nephronophthisis 15	614845	AR
16	CEP290	Glomerular and Cystic	610142	Bardet-Biedl syndrome 14 Joubert syndrome 5 Leber congenital amaurosis 10 Meckel syndrome 4 Senior-Loken syndrome 6	615991 610188 611755 611134 610189	AR AR ? AR AR
17	CEP83	Glomerular and Cystic	615847	Nephronophthisis 18	615862	AR
18	CEP89	Glomerular and Cystic	615470	Mitochondrial complex IV deficiency, nuclear type 1 Nephronophthisis	?	AR
19	CFB	Glomerular; Glomerular and Cystic	138470	Complement factor B deficiency {Hemolytic uremic syndrome, atypical, susceptibility to, 4} {Macular degeneration, age- related, 14, reduced risk of}	615561 612924 615489	AR AD DD
20	CFH	Glomerular; Glomerular and Cystic	134370	{Hemolytic uremic syndrome, atypical, susceptibility to, 1} {Macular degeneration, age- related, 4} Basal laminar drusen Complement factor H deficiency	235400 610698 126700 609814	AD, AR AD AD AD, AR
21	CFHR5	Glomerular; Glomerular and Cystic	608593	Nephropathy due to CFHR5 deficiency	614809	AD
22	CFI	Glomerular; Glomerular and Cystic	217030	{Hemolytic uremic syndrome, atypical, susceptibility to, 3} {Macular degeneration, age- related, 13, susceptibility to} Complement factor I deficiency	612923 615439 610984	AD AD AR

23	CLCN5	Glomerular; Glomerular and Cystic	300008	Dent disease 1 Hypophosphatemic rickets Nephrolithiasis, type I Proteinuria, low molecular weight, with hypercalciuric nephrocalcinosis	300009 300554 310468 308990	XLR XLR XLR XLR
24	COL4A1	Glomerular and Cystic	120130	Retinal arteries, tortuosity of {Hemorrhage, intracerebral, susceptibility to} Angiopathy, hereditary, with nephropathy, aneurysms, and muscle cramps Brain small vessel disease with or without ocular anomalies Microangiopathy and leukoencephalopathy, pontine, autosomal dominant	180000 614519 611773 175780 618564	AD ? AD AD AD
25	COL4A3	Glomerular; Glomerular and Cystic	120070	Alport syndrome 3A, autosomal dominant Alport syndrome 3B, autosomal recessive Hematuria, benign familial, 2	104200 620536 620320	AD ? AD
26	COL4A4	Glomerular; Glomerular and Cystic	120131	Alport syndrome 2, autosomal recessive Hematuria, familial benign, 1	203780 141200	AR AD
27	COL4A5	Glomerular; Glomerular and Cystic	303630	Alport syndrome 1, X-linked	301050	XLD
28	COQ2	Glomerular; Glomerular and Cystic	609825	{Multiple system atrophy, susceptibility to} Coenzyme Q10 deficiency, primary, 1	146500 607426	AD, AR AR
29	COQ6	Glomerular; Glomerular and Cystic	614647	Coenzyme Q10 deficiency, primary, 6	614650	AR
30	COQ8B	Glomerular; Glomerular and Cystic	615567	Nephrotic syndrome, type 9	615573	AR
31	CRB2	Glomerular; Glomerular and Cystic	609720	Focal segmental glomerulosclerosis 9 Ventriculomegaly with cystic kidney disease	616220 219730	AR AR
32	CTNS	Glomerular; Glomerular and Cystic	606272	Cystinosis, atypical nephropathic Cystinosis, late-onset juvenile or adolescent nephropathic	219800 219900	AR AR

				Cystinosis, nephropathic Cystinosis, ocular non-nephropathic	219800 219750	AR AR
33	CUBN	Glomerular; Glomerular and Cystic	602997	[Proteinuria, chronic benign] Imlerslund-Grasbeck syndrome 1	618884 261100	AR AR
34	DCDC2	Glomerular and Cystic	605755	?Deafness, autosomal recessive 66 Nephronophthisis 19 Sclerosing cholangitis, neonatal	610212 616217 617394	AR AR AR
35	DGKE	Glomerular; Glomerular and Cystic	601440	{Hemolytic uremic syndrome, atypical, susceptibility to, 7} Nephrotic syndrome, type 7	615008 615008	AR AR
36	DHTKD1	Glomerular; Glomerular and Cystic	614984	Charcot-Marie-Tooth disease, axonal, type 2Q Alpha-aminoacidic and alpha-ketoacidic aciduria	615025 204750	AD AR
37	DLC1	Glomerular; Glomerular and Cystic	604258	Colorectal cancer, somatic	114500	?
38	DNAJB11	Glomerular and Cystic	611341	Polycystic kidney disease 6 with or without polycystic liver disease	618061	AD
39	DZIP1L	Glomerular and Cystic	617570	Polycystic kidney disease 5	617610	AR
40	EMP2	Glomerular; Glomerular and Cystic	602334	Nephrotic syndrome, type 10	615861	AR
41	EYA1	Glomerular and Cystic	601653	Otofaciocervical syndrome Anterior segment anomalies with or without cataract Branchiootic syndrome 1 Branchiootorenal syndrome 1, with or without cataracts	166780 602588 602588 113650	AD AD AD AD
42	FAN1	Glomerular; Glomerular and Cystic	613534	Interstitial nephritis, karyomegalic	614817	AR
43	FAT1	Glomerular; Glomerular and Cystic	600976	Nephrotic syndrome Kidney dysplasia Ptosis, coloboma, microphthalmia	?	?
44	FN1	Glomerular; Glomerular and Cystic	135600	Glomerulopathy with fibronectin deposits 2 Spondylometaphyseal dysplasia, corner fracture type	601894 184255	AD AD

45	GANAB	Glomerular and Cystic	104160	Polycystic kidney disease 3	600666	AD
46	GLA	Glomerular; Glomerular and Cystic	300644	Fabry disease Fabry disease, cardiac variant	301500 301500	XL XL
47	GLIS2	Glomerular and Cystic	608539	Nephronophthisis 7	611498	AR
48	GRHPR	Glomerular; Glomerular and Cystic	604296	Hyperoxaluria, primary, type II	260000	AR
49	HNF1B	Glomerular and Cystic	189907	{Renal cell carcinoma} Renal cysts and diabetes syndrome Type 2 diabetes mellitus	144700 137920 125853	? AD AD
50	IFT172	Glomerular and Cystic	607386	Bardet-Biedl syndrome 20 Retinitis pigmentosa 71 Short-rib thoracic dysplasia 10 with or without polydactyly	619471 616394 615630	AR AR AR
51	INF2	Glomerular; Glomerular and Cystic	610982	Charcot-Marie-Tooth disease, dominant intermediate E Glomerulosclerosis, focal segmental, 5	614455 613237	AD ?
52	INVS	Glomerular and Cystic	243305	Nephronophthisis 2, infantile	602088	AR
53	IQCB1	Glomerular and Cystic	609237	Senior-Loken syndrome 5	609254	AR
54	ITGA3	Glomerular; Glomerular and Cystic	605025	Epidermolysis bullosa, junctional 7, with interstitial lung disease and nephrotic syndrome	614748	AR
55	ITGB4	Glomerular; Glomerular and Cystic	147557	Epidermolysis bullosa, junctional 5A, intermediate Epidermolysis bullosa, junctional 5B, with pyloric atresia	619816 226730	AR AR
56	JAG1	Glomerular and Cystic	601920	Deafness, congenital heart defects, and posterior embryotoxon Alagille syndrome 1 Charcot-Marie-Tooth disease, axonal, type 2HH Tetralogy of Fallot	617992 118450 619574 187500	AD AD AD AD
57	KANK1	Glomerular; Glomerular and Cystic	607704	Cerebral palsy, spastic quadriplegic, 2	612900	?

58	KANK2	Glomerular; Glomerular and Cystic	614610	Nephrotic syndrome, type 16 Palmoplantar keratoderma and woolly hair	617783 616099	AR AR
59	KANK4	Glomerular; Glomerular and Cystic	614612	Nephrotic syndrome	?	?
60	LAGE3	Glomerular; Glomerular and Cystic	300060	Galloway-Mowat syndrome 2, X-linked	301006	XLR
61	LAMA5	Glomerular; Glomerular and Cystic	601033	Bent bone dysplasia syndrome 2 Nephrotic syndrome, type 26	620076 620049	AR AR
62	LAMB2	Glomerular; Glomerular and Cystic	150325	Nephrotic syndrome, type 5, with or without ocular abnormalities Pierson syndrome	614199 609049	AR AR
63	LMX1B	Glomerular; Glomerular and Cystic	602575	Focal segmental glomerulosclerosis 10 Nail-patella syndrome	256020 161200	AD AD
64	LRP5	Glomerular and Cystic	603506	[Bone mineral density variability 1] Endosteal hyperostosis Exudative vitreoretinopathy 4 Osteopetrosis, autosomal dominant 1 Osteoporosis-pseudoglioma syndrome Polycystic liver disease 4 with or without kidney cysts	601884 144750 601813 607634 259770 617875	AD AD AD, AR AD AR AD
65	LYZ	Glomerular; Glomerular and Cystic	153450	Amyloidosis, hereditary systemic 5	620658	?
66	MAFB	Glomerular; Glomerular and Cystic	608968	Duane retraction syndrome 3 Multicentric carpotarsal osteolysis syndrome	617041 166300	AD AD
67	MAGI2	Glomerular; Glomerular and Cystic	606382	Nephrotic syndrome, type 15	617609	AR
68	MAPKBP1	Glomerular and Cystic	616786	Nephronophthisis 20	617271	AR
69	MYH9	Glomerular; Glomerular and Cystic	160775	Deafness, autosomal dominant 17 Macrothrombocytopenia and granulocyte inclusions with or without nephritis or sensorineural hearing loss	603622 155100	AD AD

70	MYO1E	Glomerular; Glomerular and Cystic	601479	Glomerulosclerosis, focal segmental, 6	614131	AR
71	NEK8	Glomerular and Cystic	609799	Nephronophthisis 9 Polycystic kidney disease 8 Renal-hepatic-pancreatic dysplasia 2	613824 620903 615415	? AD AR
72	NOTCH2	Glomerular and Cystic	600275	Alagille syndrome 2 Hajdu-Cheney syndrome	610205 102500	AD AD
73	NPHP1	Glomerular and Cystic	607100	Joubert syndrome 4 Nephronophthisis 1, juvenile Senior-Loken syndrome-1	609583 256100 266900	AR AR AR
74	NPHP3	Glomerular and Cystic	608002	Meckel syndrome 7 Nephronophthisis 3 Renal-hepatic-pancreatic dysplasia 1	267010 604387 208540	AR AR AR
75	NPHP4	Glomerular and Cystic	607215	Nephronophthisis 4 Senior-Loken syndrome 4	606966 606996	AR AR
76	NPHS1	Glomerular; Glomerular and Cystic	602716	Nephrotic syndrome, type 1	256300	AR
77	NPHS2	Glomerular; Glomerular and Cystic	604766	Nephrotic syndrome, type 2	600995	AR
78	NUP107	Glomerular; Glomerular and Cystic	607617	Ovarian dysgenesis 6 Galloway-Mowat syndrome 7 Nephrotic syndrome, type 11	618078 618348 616730	AR AR AR
79	NUP133	Glomerular; Glomerular and Cystic	607613	Galloway-Mowat syndrome 8 Nephrotic syndrome, type 18	618349 618177	AR AR
80	NUP160	Glomerular; Glomerular and Cystic	607614	?Nephrotic syndrome, type 19	618178	AR
81	NUP205	Glomerular; Glomerular and Cystic	614352	?Nephrotic syndrome, type 13	616893	AR
82	NUP85	Glomerular; Glomerular and Cystic	170285	Nephrotic syndrome, type 17	618176	AR
83	NUP93	Glomerular; Glomerular and Cystic	614351	Nephrotic syndrome, type 12	616892	AR
84	NXF5	Glomerular; Glomerular and Cystic	300319	?	?	?

85	OCRL	Glomerular; Glomerular and Cystic	300535	Dent disease 2 Lowe syndrome	300555 309000	XLR XLR
86	OFD1	Glomerular and Cystic	300170	?Retinitis pigmentosa 23 Joubert syndrome 10 Orofaciodigital syndrome I Simpson-Golabi-Behmel syndrome, type 2	300424 300804 311200 300209	XLR XLR XLD XLR
87	OSGEP	Glomerular; Glomerular and Cystic	610107	Galloway-Mowat syndrome 3	617729	AR
88	PAX2	Glomerular; Glomerular and Cystic	167409	Glomerulosclerosis, focal segmental, 7 Papillorrenal syndrome	616002 120330	AD AD
89	PDSS1	Glomerular; Glomerular and Cystic	607429	Coenzyme Q10 deficiency, primary, 2	614651	AR
90	PDSS2	Glomerular; Glomerular and Cystic	610564	Coenzyme Q10 deficiency, primary, 3	614652	AR
91	PKD1	Glomerular and Cystic	601313	Polycystic kidney disease 1	173900	AD
92	PKD2	Glomerular and Cystic	173910	Polycystic kidney disease 2	613095	AD
93	PKHD1	Glomerular and Cystic	606702	Polycystic kidney disease 4, with or without hepatic disease	263200	AR
94	PLCE1	Glomerular; Glomerular and Cystic	608414	Nephrotic syndrome, type 3	610725	AR
95	PODXL	Glomerular; Glomerular and Cystic	602632	Focal segmental glomerulosclerosis	?	AD
96	PRKCSH	Glomerular and Cystic	177060	Polycystic liver disease 1	174050	AD
97	PTPRO	Glomerular; Glomerular and Cystic	600579	Nephrotic syndrome, type 6	614196	AR
98	REN	Glomerular; Glomerular and Cystic	179820	[Hyperproreninemia] Renal tubular dysgenesis Tubulointerstitial kidney disease, autosomal dominant, 4	? 267430 613092	? AR AD
99	RPGRIP1L	Glomerular and Cystic	610937	COACH syndrome 3 Joubert syndrome 7 Meckel syndrome 5	619113 611560 611561	AR AR AR

100	SCARB2	Glomerular; Glomerular and Cystic	602257	Epilepsy, progressive myoclonic 4, with or without renal failure	254900	AR
101	SDCCAG8	Glomerular and Cystic	613524	Bardet-Biedl syndrome 16 Senior-Loken syndrome 7	615993 613615	AR AR
102	SEC61A1	Glomerular; Glomerular and Cystic	609213	Neutropenia, severe congenital, 11, autosomal dominant Immunodeficiency, common variable, 15 Tubulointerstitial kidney disease, autosomal dominant, 5	620674 620670 617056	AD AD AD
103	SEC63	Glomerular and Cystic	608648	Polycystic liver disease 2	617004	AD
104	SGPL1	Glomerular; Glomerular and Cystic	603729	RENI syndrome	617575	AR
105	SIX5	Glomerular and Cystic	600963	Branchiootorenal syndrome 2	610896	?
106	SMARCAL1	Glomerular; Glomerular and Cystic	606622	Schimke immunoosseous dysplasia	242900	AR
107	TBC1D8B	Glomerular; Glomerular and Cystic	301027	Nephrotic syndrome, type 20	301028	XL
108	THBD	Glomerular; Glomerular and Cystic	188040	{Hemolytic uremic syndrome, atypical, susceptibility to, 6} Thrombophilia 12 due to thrombomodulin defect	612926 614486	AD AD
109	TMEM67	Glomerular and Cystic	609884	RHYNs syndrome {Bardet-Biedl syndrome 14, modifier of} COACH syndrome 1 Joubert syndrome 6 Meckel syndrome 3 Nephronophthisis 11	602152 615991 216360 610688 607361 613550	AR AR AR AR AR AR
110	TP53RK	Glomerular; Glomerular and Cystic	608679	Galloway-Mowat syndrome 4	617730	AR
111	TPRKB	Glomerular; Glomerular and Cystic	608680	Galloway-Mowat syndrome 5	617731	AR
112	TRIM8	Glomerular; Glomerular and Cystic	606125	Focal segmental glomerulosclerosis and neurodevelopmental syndrome	619428	AD

11 3	TRPC6	Glomerular; Glomerular and Cystic	603652	Glomerulosclerosis, focal segmental, 2	603965	AD
11 4	TSC1	Glomerular and Cystic	605284	Focal cortical dysplasia, type II, somatic Lymphangioleiomyomatosis Tuberous sclerosis-1	607341 606690 191100	? ? AD
11 5	TSC2	Glomerular and Cystic	191092	Focal cortical dysplasia, type II, somatic Lymphangioleiomyomatosis, somatic Tuberous sclerosis-2	607341 606690 613254	? ? AD
11 6	TTC21B	Glomerular; Glomerular and Cystic	612014	Nephronophthisis 12 Short-rib thoracic dysplasia 4 with or without polydactyly	613820 613819	AD, AR AR
11 7	TTR	Glomerular; Glomerular and Cystic	176300	[Dystransthyretinemic hyperthyroxinemia] Amyloidosis, hereditary, transthyretin-related Carpal tunnel syndrome, familial	145680 105210 115430	AD AD AD
11 8	UMOD	Glomerular; Glomerular and Cystic	191845	Tubulointerstitial kidney disease, autosomal dominant, 1	162000	AD
11 9	VHL	Glomerular and Cystic	608537	Erythrocytosis, familial, 2 Hemangioblastoma, cerebellar, somatic Pheochromocytoma Renal cell carcinoma, somatic von Hippel-Lindau syndrome	263400 ? 171300 144700 193300	AR ? AD ? AD
12 0	WDR19	Glomerular and Cystic	608151	Spermatogenic failure 72 Cranioectodermal dysplasia 4 Nephronophthisis 13 Senior-Loken syndrome 8 Short-rib thoracic dysplasia 5 with or without polydactyly	619867 614378 614377 616307 614376	AR AR AR AR AR
12 1	WDR4	Glomerular; Glomerular and Cystic	605924	Galloway-Mowat syndrome 6 Microcephaly, growth deficiency, seizures, and brain malformations	618347 618346	AR AR
12 2	WDR73	Glomerular; Glomerular and Cystic	616144	Galloway-Mowat syndrome 1	251300	AR

12 3	WT1	Glomerular; Glomerular and Cystic	607102	Denys-Drash syndrome Frasier syndrome Meacham syndrome Mesothelioma, somatic Nephrotic syndrome, type 4 Wilms tumor, type 1	194080 136680 608978 156240 256370 194070	AD, SMu AD, SMu AD ? AD AD, SMu
12 4	XPO5	Glomerular; Glomerular and Cystic	607845	Focal segmental glomerulosclerosis	?	?
12 5	ZNF423	Glomerular and Cystic	604557	Joubert syndrome 19 Nephronophthisis 14	614844 614844	AD, AR AD, AR

Abbreviations. AD, Autosomal Dominant; AR, Autosomal Recessive; XLD, X-linked Dominant; XLR, X-linked Recessive; SMu, Somatic Mutation

Supplementary Table S6. Implementation outcomes of adoption, utility and intent to maintain the service.

Adoption

Supplementary Figure S1 shows the trend in the number of referrals to the GKS in each quarter during implementation. The number of referrals in the first six months after implementation (42 referrals per quarter) then reduced to a median of 20 (IQR 16 – 21) referrals per quarter.

In SGH, 6 genetic nephrologists referred 107 patients (57.5% of all referrals) and 26 non-genetic nephrologists and trainees referred 79 patients (42.5% of all referrals). **Supplementary Figure S2** shows that referrals were higher among genetic nephrologists compared to non-genetic nephrologists (median 14 referrals (IQR 8 – 23.8) per nephrologist versus 2 (IQR 2 – 4) respectively, $p=0.005$). **Supplementary Figure S3** shows the number of referrals made per genetic nephrologist and non-genetic nephrologists each quarter. Each genetic nephrologist made a median of 2 referrals per quarter (IQR 0 – 4) while each non-genetic nephrologist referred a median of 0.5 patients per quarter (IQR 0 – 1) for genetic testing ($p<0.001$). There was no difference between the referral rates of non-genetic nephrologists in the first twelve months (median 0.5 per quarter, IQR 0 – 1) and in the nine months after educational sessions were conducted (median 0 per quarter, IQR 0 – 1) ($p = 0.208$).

Among 201 patients referred to the GKS, 109 (54.2%) consented to receive pre-test genetic counselling. Thirty-seven (18.4%) were not contactable, three (1.5%) wanted more time to consider, and 52 (25.9%) declined genetic counselling citing uncertainty about the benefits of genetic testing (25 patients, 12.4%), concerns regarding future insurability for themselves or family members (23 patients, 11.4%) and unwillingness for additional visits (4 patients, 2.0%). Among 109 patients who received pre-test counselling, 101 (92.7%) received genetic testing. Six patients declined testing and two withdrew consent after testing. These eight patients cited concerns about future insurability for themselves or family members.

Utility

Among 101 patients who underwent genetic testing, 49 patients (48.5%) were tested for the glomerular gene panel and the rest had the glomerular and cystic panel. The clinical genetics lab initially found that 13 patients (12.9%) had pathogenic variants that explained their kidney condition, while 65 (64.4%) had variants of uncertain significance (VUS). The genomic board variant curation meetings determined that 18 (17.8%) patients had VUS suspicious for pathogenicity.²⁵ VUS resolution efforts included segregation analysis, family phenotyping, research tests such as functional studies and further genetic analysis e.g. 10 patients (9.9%) had additional WES. Subsequently, 8 additional patients (7.9%) had genetic diagnoses so that a total of 21 patients (20.8%) had genetic diagnoses. Among these 21 patients, the most common genetic diagnosis was Alport's syndrome (17 patients; 81.0%). The other diagnoses included *INF2*-associated FSGS, *PKD2*-associated Autosomal Dominant Polycystic Kidney Disease and *HNF1 β* -associated kidney disease.

Intent to maintain the service

Twelve months after implementation, 22 (44.9%) of 49 non-genetic nephrologists completed the NoMAD survey. Among these 22 respondents, 21 (95.5%) had patients with suspected genetic etiology but only 10 (47.6%) had ever arranged for genetic testing. Sixteen (72.7%) were aware of the GKS implementation. These 16 respondents were further surveyed on their perceptions of the GKS (**Supplementary Table S10**). In the domain of coherence insight, most respondents recognised its potential value and benefits to patient care. In the domain of cognitive participation, the majority agreed that there was shared understanding on the purpose of the service and would support the service. There was also recognition that the genetic nephrologists were key drivers and advocates for the service. In the domain of collective action,

approximately one third of surveyed nephrologists expressed concerns about the adequacy of training and resources, integration of the service into existing workflows and potential disruptions to working relationships. For example, only 43.8% agreed or strongly agreed there was sufficient resources to support the service and 62.5% agreed or strongly agreed that they could easily integrate the GKS into their existing work. However, most respondents felt that the genetic nephrologists had the necessary skills or support to run this service and that there was overall adequate support from the leadership. In the domain of reflexive monitoring, most staff members expressed appreciation for the impact of this service on their work. All believed that feedback would be instrumental in enhancing the program further.

Supplementary Table S7. Standards for Reporting Implementation Studies: the StaRI checklist.

Checklist item		Reported on page #	Implementation Strategy	Reported on page #	Intervention
			“Implementation strategy” refers to how the intervention was implemented		“Intervention” refers to the healthcare or public health intervention that is being implemented.
Title and abstract					
Title	1	1	Identification as an implementation study, and description of the methodology in the title and/or keywords		
Abstract	2	N.A.	Identification as an implementation study, including a description of the implementation strategy to be tested, the evidence-based intervention being implemented, and defining the key implementation and health outcomes.		
Introduction					
Introduction	3	3	Description of the problem, challenge or deficiency in healthcare or public health that the intervention being implemented aims to address.		
Rationale	4		The scientific background and rationale for the implementation strategy (including any underpinning theory/framework/model, how it is expected to achieve its effects and any pilot work).	3	The scientific background and rationale for the intervention being implemented (including evidence about its effectiveness and how it is expected to achieve its effects).
Aims and objectives	5	3	The aims of the study, differentiating between implementation objectives and any intervention objectives.		
Methods: description					
Design	6	Supp	The design and key features of the evaluation, (cross referencing to any appropriate methodology reporting standards) and any changes to study protocol, with reasons		
Context	7	3	The context in which the intervention was implemented. (Consider social, economic, policy, healthcare, organisational barriers and facilitators that might influence implementation elsewhere).		
Targeted ‘sites’	8		The characteristics of the targeted ‘site(s)’ (e.g locations/personnel/resources etc.) for implementation and any eligibility criteria.	Supp	The population targeted by the intervention and any eligibility criteria.
Description	9		A description of the implementation strategy	Supp	A description of the intervention
Sub-groups	10	N.A.	Any sub-groups recruited for additional research tasks, and/or nested studies are described		
Methods: evaluation					

Outcomes	11		Defined pre-specified primary and other outcome(s) of the implementation strategy, and how they were assessed. Document any pre-determined targets	Supp	Defined pre-specified primary and other outcome(s) of the intervention (if assessed), and how they were assessed. Document any pre-determined targets
Process evaluation	12	Supp	Process evaluation objectives and outcomes related to the mechanism by which the strategy is expected to work		
Economic evaluation	13	N.A.	Methods for resource use, costs, economic outcomes and analysis for the implementation strategy	N.A.	Methods for resource use, costs, economic outcomes and analysis for the intervention
Sample size	14	Supp	Rationale for sample sizes (including sample size calculations, budgetary constraints, practical considerations, data saturation, as appropriate)		
Analysis	15	Supp	Methods of analysis (with reasons for that choice)		
Sub-group analyses	16	4	Any a priori sub-group analyses (e.g. between different sites in a multicentre study, different clinical or demographic populations), and sub-groups recruited to specific nested research tasks		

Results					
Characteristics	17		Proportion recruited and characteristics of the recipient population for the implementation strategy	4	Proportion recruited and characteristics (if appropriate) of the recipient population for the intervention
Outcomes	18		Primary and other outcome(s) of the implementation strategy	4	Primary and other outcome(s) of the Intervention (if assessed)
Process outcomes	19	Supp	Process data related to the implementation strategy mapped to the mechanism by which the strategy is expected to work		
Economic evaluation	20	N.A.	Resource use, costs, economic outcomes and analysis for the implementation strategy	N.A.	Resource use, costs, economic outcomes and analysis for the intervention
Sub-group analyses	21	4	Representativeness and outcomes of subgroups including those recruited to specific research tasks		
Fidelity/adaptation	22		Fidelity to implementation strategy as planned and adaptation to suit context and preferences	Supp	Fidelity to delivering the core components of intervention (where measured)
Contextual changes	23	N.A.	Contextual changes (if any) which may have affected outcomes		
Harms	24	N.A.	All important harms or unintended effects in each group		
Discussion					
Structured discussion	25	5	Summary of findings, strengths and limitations, comparisons with other studies, conclusions and implications		

Supplementary materials to: Tung LL, Lim CC, Mok I, et al. Genetic counselling and testing for inherited glomerular kidney diseases: A single-centre experience. Ann Acad Med Singap 2025;54:798-799. DOI: <https://doi.org/10.47102/annals-acadmedsg.2025118>

Implications	26		Discussion of policy, practice and/or research implications of the implementation strategy (specifically including scalability)	5	Discussion of policy, practice and/or research implications of the intervention (specifically including sustainability)
General					
Statements	27	Supp	Include statement(s) on regulatory approvals (including, as appropriate, ethical approval, confidential use of routine data, governance approval), trial/study registration (availability of protocol), funding and conflicts of interest		

Supplementary Table S8. Characteristics of patients who underwent genetic testing.

Characteristic	Results (n = 101)
Age, years	43 (IQR 34 – 55)
Ethnicity	
- Chinese, n (%)	83 (82.1)
- Malay, n (%)	11 (10.9)
- Indian, n (%)	4 (4.0)
- Others, n (%)	3 (3.0)
Kidney manifestation	
- Isolated haematuria, n (%)	18 (17.8)
- Albuminuria (A2 or A3) , n (%)	43 (42.6)
- Nephrotic syndrome , n (%)	4 (4.0)
- CKD stages G3 – 5, n (%)	27 (26.7)
- Kidney failure on KRT, n (%)	9 (8.9)
-- Age at KRT initiation, years	36 (28 – 57)
Family history of kidney disease, n (%)	76 (75.2)
Previous kidney biopsy finding, n (%)	35 (34.7)
<i>(can have > 1 finding)</i>	
- FSGS, n (%)	4/35 (11.4)
- Thin basement membrane, n (%)	6/35 (17.1)
- IgA nephropathy, n (%)	5/35 (14.2)
- Alport's syndrome, n (%)	1/35 (2.9)
- Minor changes, n (%)	16/35 (45.7)
- Others, n (%)	6/35 (17.1)
Indication for genetic testing	
<i>(can have > 1 indication)</i>	
- Isolated haematuria	20 (19.8%)
- Persistent albuminuria	61 (60.4%)
- Steroid-resistant nephrotic syndrome	3 (3.0%)
- CNI-resistant nephrotic syndrome	0
- Familial IgA nephropathy	3 (3.0%)
- CKD stages G3-5 of unknown aetiology	20 (19.8%)
- Rapid eGFR decline	2 (2.0%)

Abbreviations: CKD, chronic kidney disease; KRT, Kidney Replacement Therapy; FSGS, focal segmental glomerulosclerosis; Immunoglobulin A, IgA; CNI, calcineurin inhibitor; eGFR, estimated glomerular filtration rate.

Categorical variables were presented as number (proportion). Continuous variables summarized as medians with interquartile ranges [IQR (25th percentile, 75th percentile)].

Supplementary Table S9. Genes detected with pathogenic/likely pathogenic variants and variants of uncertain significance (VUS) of high pathogenic suspicion.

Gene	Number
Pathogenic/ Likely Pathogenic	21
<i>COL4A3</i>	2
<i>COL4A4</i>	13
<i>COL4A5</i>	2
<i>INF2</i>	1
<i>PKD2</i>	1
<i>HNF1B</i>	2
Carrier of Pathogenic/ Likely Pathogenic Variant	2
<i>INVS</i>	1
<i>PKHD1</i>	1
VUS of high pathogenic suspicion	10
<i>COL4A3</i>	3
<i>COL4A4</i>	1
<i>NPHS1</i>	1
<i>LRP5</i>	1
<i>EYA1</i>	1
<i>MYO1E</i>	1
<i>OCRL</i>	1
<i>UMOD</i>	1
Carriers for Pathogenic Variants associated with monogenic glomerular disease inherited in Autosomal Recessive pattern Two patients (2.0%) were carriers for pathogenic variants that can cause disease in an autosomal recessive pattern of inheritance. The first patient had CKD of unknown aetiology with hypertension and bilateral kidney cysts and was subsequently found to have a heterozygous pathogenic variant in the <i>INVS</i> gene (associated with autosomal recessive nephronophthisis). The second patient had asymptomatic microscopic haematuria and found to have a heterozygous pathogenic variant in the <i>PKHD1</i> gene (associated with autosomal recessive polycystic kidney disease). Both patients received counselling for carrier status during return of results.	

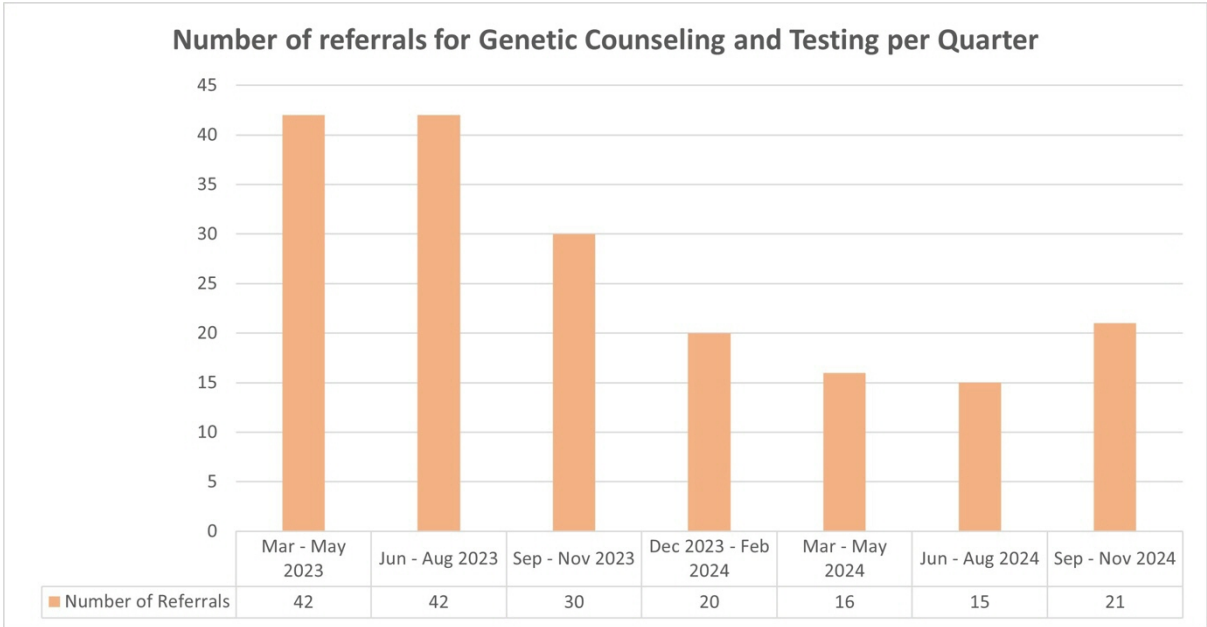
Supplementary Table S10. The Normalization of Complex Interventions – Measure Development (NoMAD) survey responses at 12 months after implementation of the genetic kidney disease service.

Question	At 12-months		
When you use the program, how familiar does it feel?	2 (0, 6) ^a		
Do you feel the program is currently a normal part of your work?	4 (2, 6) ^a		
Do you feel the program will become a normal part of your work?	7 (4, 9) ^a		
	Strongly disagree	Neither agree nor disagree	Strongly agree
I can see how the program differs from usual ways of working (CI)	6.2%	6.2%	87.6%
Staff involved have a shared understanding of the purpose of the program (CI)	6.2%	6.2%	87.6%
I understand how the program affects the nature of my own work (CI)	0%	12.4%	87.6%
I can see the potential value of the program for my work (CI)	0%	0%	100.0%
There are key people who drive the program forward and get others involved (CP)	0%	6.2%	93.8%
I believe that participating in the program is a legitimate part of my role (CP)	0%	6.2%	93.8%
I'm open to working with colleagues in new ways to use the program (CP)	0%	0%	100.0%
I will continue to support the program (CP)	0%	6.2%	93.8%
I can easily integrate the program into my existing work (CA)	6.2%	31.3%	62.5%
The program disrupts working relationships (CA)	62.5%	25.0%	12.5%
I have confidence in other people's ability to use the program (CA)	6.2%	12.5%	81.3%
Work is assigned to those with skills appropriate to the program (CA)	0%	0%	100.0%
Sufficient training is provided to enable staff to implement the program (CA)	0%	37.5%	62.5%
Sufficient resources are available to support the program (CA)	18.7%	37.5%	43.8%
Management adequately supports the program (CA)	0%	6.2%	93.8%
I am aware of reports about the effects of the program (RM)	12.5%	31.3%	56.3%
The staff agree that the program is worthwhile (RM)	6.2%	12.5%	81.3%

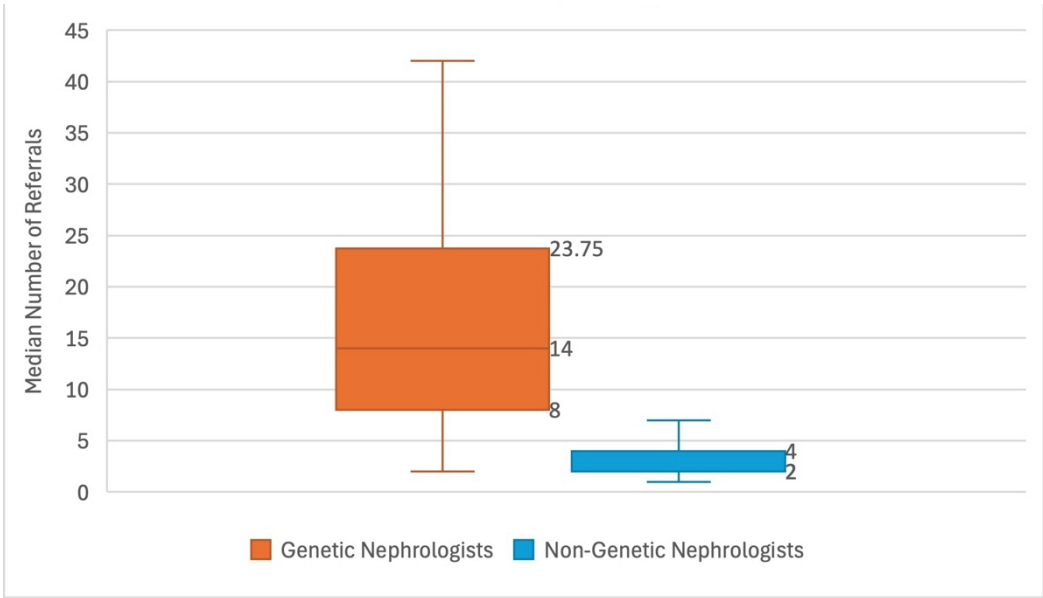
I value the effects that the program has had on my work (RM)	0%	6.2%	93.8%
Feedback about the program can be used to improve it in the future (RM)	0%	0%	100.0%
I can modify how I work with the program (RM)	0%	6.2%	93.8%

^aScored from 0 “not at all” to 10 “completely” and reported as median (25th centile, 75th centile).
Abbreviations: CI, coherence insights (staff’s comprehension of the purpose and perceived value within their practice), CP, cognitive participation (engagement and motivation levels among healthcare staff to adopt the tool); CA, collective action (integration into daily workflows and influence on existing practice assessed by questions); RM, reflexive monitoring (perceived impact on patient care and contribution to enhance existing practice)

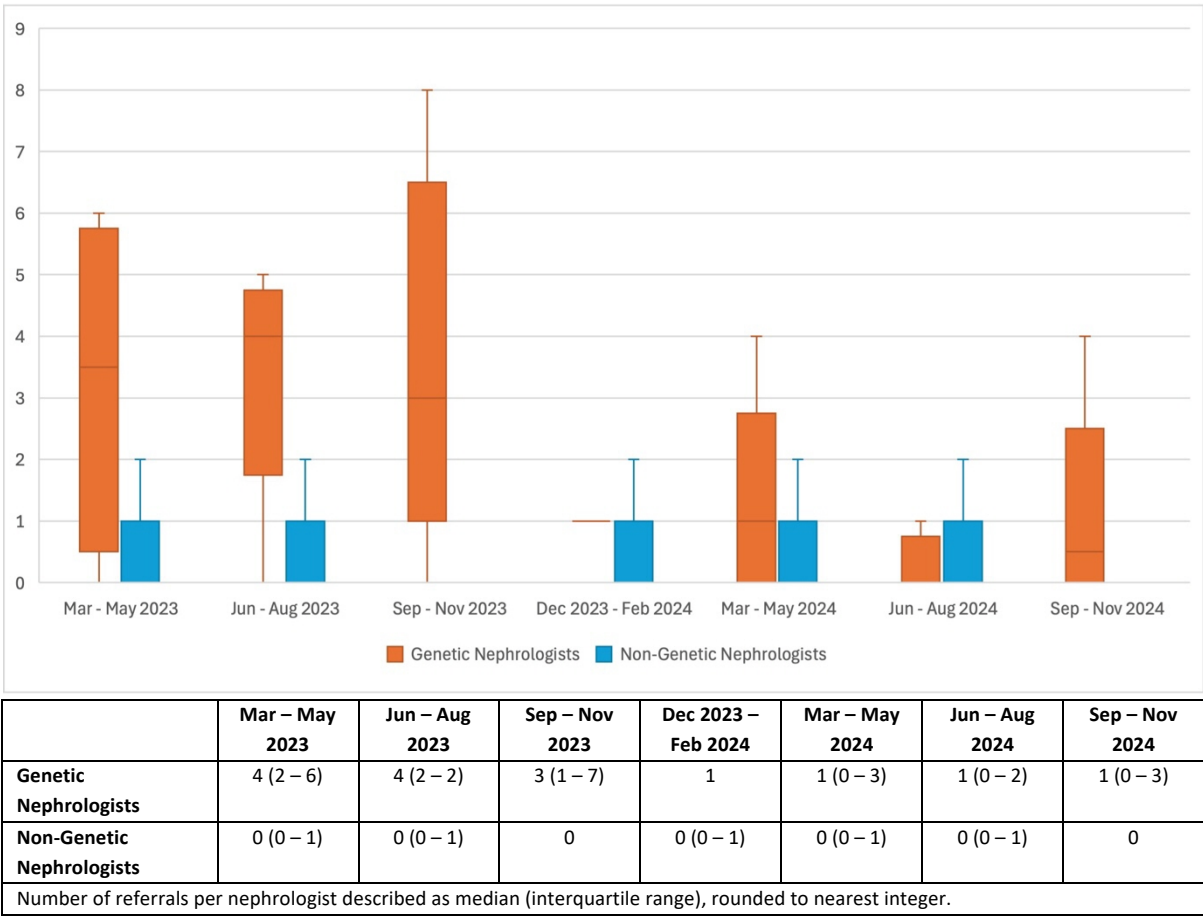
Supplementary Figure S1. Number of referrals for genetic counselling and testing per quarter.



Supplementary Figure S2. Box plots of referrals per nephrologist from genetic nephrologist compared with non-genetic nephrologists.



Supplementary Figure S3. Box plots of referrals per nephrologist from genetic vs non-genetic nephrologists per quarter.



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