

### RaDaR Inclusion and Exclusion Criteria

Diagnosis	Cohort	Inclusion Criteria	Exclusion Criteria	Date of Diagnosis
<b>Adenine Phosphoribosyltransferase Deficiency (APRT-D)</b>	APRT Deficiency	APRT Deficiency confirmed Abolished APRT enzyme activity or confirmed disease-causing mutation	None, if APRT Deficiency not confirmed	Date that clinical diagnosis was first made
<b>Alport Syndrome and Type IV collagenopathies</b>	Alport	Alport Syndrome definite or probable Alport carrier definite or probable Female heterozygote for X-linked Alport Syndrome (COL4A5) Heterozygote for autosomal Alport Syndrome (COL4A3, COL4A4) Thin basement membrane nephropathy	None stated	Date that clinical diagnosis was first made
<b>Autoimmune distal renal tubular acidosis</b>	Tubulopathy	Autoimmune distal renal tubular acidosis	None stated	Date that clinical diagnosis was first made
<b>Autosomal dominant distal renal tubular acidosis</b>	Tubulopathy	Autosomal dominant distal renal tubular acidosis Genetically confirmed heterozygous pathogenic variant in SLC4A1	None stated	Date that clinical diagnosis was first made
<b>Autosomal recessive distal renal tubular acidosis</b>	Tubulopathy	Autosomal recessive distal renal tubular acidosis Genetically confirmed homozygous pathogenic variant in ATP6V0A4, ATP6V1B1 or FOXI1	None stated	Date that clinical diagnosis was first made
<b>Autosomal recessive proximal renal tubular acidosis</b>	Tubulopathy	Autosomal recessive proximal renal tubular acidosis with ocular abnormalities and intellectual disability Genetically confirmed homozygous pathogenic variant in SLC4A4	None stated	Date that clinical diagnosis was first made

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<b>Bartter Syndrome types 1 and 2</b>	Tubulopathy	Bartter Syndrome, infantile onset Hypokalaemic alkalosis, infantile onset without hypertension Hypokalaemic alkalosis, infantile onset with raised renin	Acidosis Persistent Hyperkalaemia	Date that clinical diagnosis was first made
<b>Bartter Syndrome type 3 Gitelman Syndrome</b>	Tubulopathy	Bartter Syndrome type 3 Gitelman Syndrome Hypokalaemic alkalosis with hypomagnesaemia Hypokalaemic alkalosis with raised renin Hypokalaemic alkalosis without hypertension	Acidosis Hyperkalaemia	Date that clinical diagnosis was first made
<b>Bartter Syndrome Type 4</b>	Tubulopathy	Bartter Syndrome, infantile onset with deafness Hypokalaemic alkalosis, infantile onset without hypertension with deafness Hypokalaemic alkalosis, infantile onset with raised renin, with deafness	Acidosis Persistent Hyperkalaemia	Date that clinical diagnosis was first made
<b>BK Nephropathy</b>	BK Nephropathy	Significant BK viraemia, with polymerase chain reaction (PCR) greater than or equal to 10 log 4 copies per ml. A confirmatory biopsy is <b>not</b> required.	None stated	Date that PCR first equalled or exceeded 10 log 4

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<b>Calciophylaxis</b>	Calciophylaxis	Any patient with a diagnosis of clinical diagnosis of Calciophylaxis; tissue diagnosis not required	None stated	Date that the diagnosis was made by a nephrologist or dermatologist
<b>Cystinosis (Nephropathic Cystinosis)</b>	Cystinosis	Cystinosis	None stated	Date that biochemical testing first showed an elevated level of white blood cell cysteine
<b>Cystinuria</b>	Cystinuria	Biochemically proven cystine kidney stone  Urinary cystine level > 3X reference range of the laboratory it was taken in  Cystine crystals in the urine (biochemically proven)	Another cause of proximal tubular dysfunction accounting for the raised cystine level e.g. Fanconi's syndrome	Date that any of the inclusion criteria first occurred
<b>Dent Disease</b>	Dent & Lowe	Dent Disease	None stated	Date that the clinical label of Dent Disease was first applied
<b>Dominant hypophosphatemia with nephrolithiasis or osteoporosis</b>	Tubulopathy	Dominant hypophosphatemia with nephrolithiasis or osteoporosis  Genetically confirmed heterozygous pathogenic variant in SLC34A1, SLC9A3R1, SLC34A3	None stated	Date that clinical diagnosis was first made
<b>Drug induced Fanconi syndrome</b>	Tubulopathy	Drug induced Fanconi syndrome	None stated	Date that clinical diagnosis was first made
<b>Drug induced hypomagnesemia</b>	Tubulopathy	Drug induced hypomagnesemia	None stated	Date that clinical diagnosis was first made

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<b>Drug induced Nephrogenic Diabetes Insipidus</b>	Tubulopathy	Drug induced Nephrogenic Diabetes Insipidus	None stated	Date that clinical diagnosis was first made
<b>EAST syndrome (Epilepsy, Ataxia, Sensorineural deafness, Tubulopathy)</b>	Tubulopathy	Gitelman/Bartter-type syndrome in childhood with epilepsy /ataxia	Normal CNS examination	Date that clinical diagnosis was first made
<b>Fabry Disease</b>	Fabry	Confirmed diagnosis of Fabry Disease	None stated	Date that genetic diagnosis was made and/or, for males, the date that low alpha gal levels were first recorded
<b>Familial Hypomagnesaemia with hypercalciuria and nephrocalcinosis CLDN16/19</b>	Tubulopathy	Familial Hypomagnesaemia with Hypercalciuria and Nephrocalcinosis Genetically confirmed homozygous pathogenic variant in CLDN 16/19	None stated	Date that clinical diagnosis was first made
<b>Familial primary hypomagnesemia with hypocalcuria FXYD2</b>	Tubulopathy	Familial primary hypomagnesemia with hypocalciuria Genetically confirmed homozygous pathogenic variant in FXYD2	None stated	Date that clinical diagnosis was first made
<b>Familial primary hypomagnesemia with normocalcuria EGF</b>	Tubulopathy	Familial primary hypomagnesemia with normocalcuria Genetically confirmed homozygous pathogenic variant in EGF	None stated	Date that clinical diagnosis was first made
<b>Familial renal glucosuria SLC5A2</b>	Tubulopathy	Familial renal glucosuria Genetically confirmed homozygous pathogenic variant in SLC5A2	None stated	Date that clinical diagnosis was first made

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<b>Fanconi Renotubular syndrome 1 (FRTS1)</b>	Tubulopathy	Fanconi Renotubular syndrome 1	None stated	Date that clinical diagnosis was first made
<b>Fanconi Renotubular syndrome 2 (FRTS2)</b>	Tubulopathy	Fanconi Renotubular syndrome 2 Genetically confirmed homozygous pathogenic variant in SLC34A1	None stated	Date that clinical diagnosis was first made
<b>Fanconi Renotubular syndrome 3 (FRTS3)</b>	Tubulopathy	Fanconi Renotubular syndrome 3 Genetically confirmed homozygous pathogenic variant in EHHADH	None stated	Date that clinical diagnosis was first made
<b>Fibromuscular Dysplasia</b>	Fibromuscular Dysplasia	Diagnosis of FMD established on radiological or histological grounds  FMD of any arterial bed	None stated	Date that FMD was diagnosed by radiological (or histological) methods
<b>Generalized pseudohypoaldosteronism type 1</b>	Tubulopathy	Generalized pseudohypoaldosteronism type 1 Genetically confirmed homozygous pathogenic variant in SCNN1A/ SCNN1B/SCNN1G	None stated	Date that clinical diagnosis was first made

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<b>Haemolytic Uraemic Syndrome - Atypical</b>	aHUS	<p>Diarrhoea-negative HUS, includes congenital and familial HUS</p> <p>Renal biopsy showing a TMA and/or the triad of microangiopathic haemolytic anaemia, thrombocytopenia, renal failure.</p>	<p>Shiga toxin associated HUS</p> <p>Secondary causes:</p> <ul style="list-style-type: none"> <li>• Drugs</li> <li>• Infection (HIV, pneumonia, streptococcus)</li> <li>• Transplantation (bone marrow, liver, lung, cardiac but not de-novo renal)</li> <li>• Cobalamin deficiency</li> <li>• SLE</li> <li>• APL Ab syndrome</li> <li>• Scleroderma</li> <li>• ADAMTS13 antibodies or deficiency</li> </ul>	Date of first presentation

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<p style="text-align: center;"><b>Haemolytic Uraemic Syndrome-Shiga toxin (Verocytotoxin)-associated</b></p>	<p style="text-align: center;">STEC-HUS</p>	<p>Acute kidney injury (AKI) with elevated creatinine for age and/or oligoanuria (urine output &lt;0.5ml/kg/hr over 24hr period) with either:</p> <ul style="list-style-type: none"> <li>• Microangiopathic haemolytic anaemia (MAHA) - defined as Hgb &lt; 10mg/dl with fragmented RBCs</li> </ul> <p style="text-align: center;">or</p> <ul style="list-style-type: none"> <li>• Thrombocytopenia - defined as platelet count less than 130, 000 x 10<sup>9</sup>/l</li> </ul> <p style="text-align: center;">and</p> <ul style="list-style-type: none"> <li>• Occurring with Shiga-toxin producing E Coli (STEC) infection defined as:</li> <li>• Positive STEC culture</li> <li>• Positive PCR for Stx gene directly from a faecal specimen</li> <li>• Positive antibodies to the lipopolysaccharide</li> <li>• antigen of E. coli serogroups O157, O26, O103, O111 and O145</li> </ul>	<p>Septicaemia</p> <p>Malignant hypertension</p> <p>Primary vascular disease</p> <p>Familial HUS not being part of the same</p>	<p style="text-align: center;">Date on which the STEC-HUS was suspected.</p>
<p style="text-align: center;"><b>Heavy metal induced Fanconi syndrome</b></p>	<p style="text-align: center;">Tubulopathy</p>	<p>Heavy metal induced Fanconi syndrome</p>	<p>None stated</p>	<p style="text-align: center;">Date that clinical diagnosis was first made</p>

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<b>Hepatocyte Nuclear Factor-1B mutation</b>	HNF1b	Hepatocyte nuclear factor-1B mutation Renal cysts and diabetes (RCAD) Inherited genetic diabetes type 2 (MODY 5).	None stated	Date of genetic diagnosis
<b>Hereditary renal hypouricemia</b>	Tubulopathy	Hereditary renal hypouricemia Genetically confirmed homozygous pathogenic variant in SLC22A12, SLC2A9	None stated	Date of genetic diagnosis
<b>Hereditary hypophosphatemic rickets with hypercalciuria</b>	Tubulopathy	Hereditary hypophosphatemic rickets with hypercalciuria Genetically confirmed homozygous pathogenic variant in SLC34A3	None stated	Date of genetic diagnosis
<b>Hyperoxaluria (Primary hyperoxaluria, Oxalosis)</b>	Hyperoxaluria	Primary Hyperoxaluria Type 1 Primary Hyperoxaluria Type 2 Primary Hyperoxaluria Type 3 Primary Hyperoxaluria awaiting genetic confirmation (Urine oxalate excretion $\geq 0.8$ mmol/1.73 m <sup>2</sup> /24 hrs) Primary Hyperoxaluria Unclassified Primary Hyperoxaluria Unclassified but with systemic oxalate deposition	Secondary hyperoxaluria associated with gastrointestinal disease  Renal failure without systemic oxalate deposits	Date that definitive diagnosis by genetic confirmation with gene mutation was first made.  If in doubt use the earliest date that PH was suspected or the date when treatment was first introduced



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<p style="text-align: center;"><b>Hyperuricaemic Nephropathy (Primary/Familial Hyperuricaemic nephropathy)</b></p> <p style="text-align: center;"><b>Medullary cystic kidney disease</b></p>	ADTKD	<p>Autosomal Dominant Tubulointerstitial Kidney Disease (ADTKD; previously known as FUAN)</p> <p>Familial juvenile hyperuricaemic nephropathy</p> <p>Familial gouty nephropathy</p> <p>Familial urate nephropathy</p> <p>Familial interstitial nephropathy</p> <p>Uromodulin-associated nephropathy</p> <p>Medullary cystic kidney disease (type I or II)</p>	None stated	Date that genetic confirmation was received
<b>IgA Nephropathy</b>	IgA Nephropathy	Biopsy proven IgA Nephropathy plus proteinuria >0.5g/ day or eGFR<60ml/min	All forms of secondary IgA nephropathy, including Henoch Schonlein purpura	Date of renal biopsy
<b>Isolated autosomal dominant hypomagnesemia, Glaudemans type</b>	Tubulopathy	<p>Isolated autosomal dominant hypomagnesemia</p> <p>Genetically confirmed homozygous pathogenic variant in KCNA1</p>	None stated	Date that clinical diagnosis was first made

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<b>Liddle syndrome</b>	Tubulopathy	Liddle syndrome Hypertension with hypokalaemia, suppressed aldosterone Hypertension with suppressed aldosterone Autosomal dominant hypertension, suppressed aldosterone	Hyperaldosteronism	Date that clinical diagnosis was first made
<b>Low Syndrome</b>	Dent & Lowe	Low Syndrome	None Stated	Date that the clinical label of Low Syndrome was first applied

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<p><b>Membranoproliferative glomerulonephritis</b></p> <p><b>Mesangiocapillary glomerulonephritis</b></p> <p><b>Dense Deposit Disease</b></p> <p><b>C3 Glomerulonephritis</b></p> <p><b>C3 Glomerulopathy</b></p>	MPGN	<p>Child or adult with histological finding of:</p> <p>MPGN Type I</p> <p>Dense Deposit Disease (morphological pattern may or may not be MPGN)</p> <p>Other pattern of MPGN</p> <p>C3 Glomerulonephritis (Characterised by C3 deposits in the absence of immunoglobulin with electron dense deposits (morphological pattern may or may not be MPGN)</p> <p>Unclassified GN with capillary wall immune deposits</p>	<p>MPGN known to be secondary to:</p> <p>Chronic bacterial infection</p> <p>Hepatitis B or C infection</p> <p>Malignancy</p> <p>Systemic lupus erythematosus (by ACR criteria)</p>	Date of biopsy
<b>Membranous Nephropathy</b>	Membranous Nephropathy	Membranous nephropathy confirmed by kidney histology	Lupus nephritis	Date of biopsy
<b>Mitochondrial Renal Disease</b>	Mitochondrial	Mitochondrial Disease or Mitochondrial Cytopathy	None Stated	Date that clinical diagnosis was first made

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<b>Monoclonal Gammopathy of Renal Significance</b>	MGRS	<p>Renal biopsy proven confirmation of:</p> <ul style="list-style-type: none"> <li>• AH amyloidosis*</li> <li>• AHL amyloidosis*</li> <li>• AL amyloidosis*</li> <li>• C3 glomerulonephritis with monoclonal gammopathy</li> <li>• Crystalglobulinaemia</li> <li>• Crystal-storing histiocytosis</li> <li>• Fibrillary Glomerulonephritis</li> <li>• Immunotactoid/Glomerulonephritis with Organised Microtubular Monoclonal Immunoglobulin Deposits (GOMMID)</li> <li>• Intracapillary monoclonal IgM without cryoglobulin</li> <li>• Intraglomerular/capillary lymphoma/leukaemia</li> <li>• Light chain cast nephropathy</li> <li>• Light chain proximal tubulopathy, crystalline</li> <li>• Light chain proximal tubulopathy, non crystalline</li> <li>• Monoclonal Immunoglobulin Deposition Disease (MIDD; includes Light Chain Deposition Disease - LCDD; Heavy Chain Deposition Disease - HCDD; and Light and Heavy Chain Deposition Disease - LHCDD)</li> <li>• Proliferative glomerulonephritis with monoclonal immunoglobulin deposits – PGNMID</li> <li>• Thrombotic Microangiopathy with monoclonal gammopathy</li> <li>• Type 1 cryoglobulinaemic Glomerulonephritis</li> <li>• Unclassified MGRS</li> </ul> <p>*Patients with systemic amyloidosis may have a renal biopsy confirming AL amyloidosis or a biopsy of other tissue with confirmation of renal involvement by the UK National Amyloidosis Centre.</p>	None Stated	Date of biopsy

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<b>Nephrogenic diabetes insipidus</b>	Tubulopathy	Nephrogenic diabetes insipidus Genetically confirmed homozygous pathogenic variant in AVPR2, AQP2	None stated	Date that clinical diagnosis was first made
<b>Nephrogenic syndrome of inappropriate antidiuresis</b>	Tubulopathy	Nephrogenic syndrome of inappropriate antidiuresis Genetically confirmed homozygous pathogenic variant in AVPR2	None stated	Date that clinical diagnosis was first made
<b>Nephronophthisis</b>	ARPKD/NPHP	Histological or radiological features of Nephronophthisis Genetic diagnosis of Nephronophthisis or Nephronophthisis-related ciliopathy	None stated	Date that histological /radiological or genetic diagnosis was first made

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<p><b>Nephrotic Syndrome - Steroid Sensitive or Steroid Resistant</b></p> <p><b>(Congenital nephrotic syndrome, nephrotic syndr ome with focal segmental glomerulosclerosis)</b></p>	INS	<p>Children and adults with idiopathic Nephrotic Syndrome (nephrotic range proteinuria and hypoalbuminaemia)</p> <p>Congenital NS (presumed Steroid Resistance)</p> <p>Childhood or adult onset with primary Steroid Resistance</p> <p>Childhood or adult onset with late onset Steroid Resistance</p> <p>Steroid Sensitive Nephrotic Syndrome (full or partial remission in response to steroids)</p> <p>As part of a syndrome e.g. Nail Patella Syndrome and Denys-Drash Syndrome</p> <p>Those with a biopsy diagnosis of FSGS or minimal change disease can be included if they fall in the above categories but biopsy is not a prerequisite for inclusion</p>	<p>Secondary causes of Nephrotic Syndrome</p> <ul style="list-style-type: none"> <li>• Primary diagnosis of Glomerulonephritis (IgA Nephropathy, Membranoproliferative Glomerulonephritis, Membranous Nephropathy)</li> <li>• Vasculitis</li> <li>• Systemic Lupus Erythematosus</li> <li>• Diabetes</li> <li>• Obesity</li> <li>• Hypertension</li> </ul>	Date of presentation to secondary or tertiary centre
<b>Oncogenic osteomalacia</b>	Tubulopathy	Oncogenic osteomalacia	None stated	Date that clinical diagnosis was first made
<b>Osteopetrosis with renal tubular acidosis</b>	Tubulopathy	<p>Osteopetrosis with renal tubular acidosis</p> <p>Genetically confirmed homozygous pathogenic variant in CA2</p>	None stated	Date that clinical diagnosis was first made

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<p><b>Polycystic Kidney Disease - Autosomal Dominant</b></p>	<p>ADPKD</p>	<p>Clinical features of Autosomal Dominant Polycystic Kidney Disease meeting current image based diagnostic criteria</p> <p>Clinical features compatible with ADPKD in the absence of a family history</p> <p>Pathogenic or likely pathogenic PKD1 or PKD2 mutation with or without clinical features</p>	<p>Autosomal dominant polycystic liver disease with no evidence of renal cysts</p>	<p>Date that the clinical diagnosis was first made.</p> <p>This may be reported by the clinician as the date of the diagnostic scan or by the patient if scans were performed at another centre</p>
<p><b>Polycystic Kidney Disease - Autosomal Recessive</b></p>	<p>ARPKD</p>	<p>Autosomal Recessive Polycystic Kidney Disease</p> <p>Congenital Hepatic Fibrosis</p> <p>Caroli Syndrome with kidney malformation or cyst</p>	<p>None stated</p>	<p>Date that clinical diagnosis was first made.</p>

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<b>Pregnancy and Chronic Kidney Disease</b>	Pregnancy	<p>Pregnancy in all women known to have CKD 1-5 prior to pregnancy or those with a serum creatinine &gt;85umol/l on two occasions during pregnancy</p> <p>Pregnancy in all women with renal transplants regardless of function</p> <p>Pregnancy in all women with previous or current lupus nephritis regardless of function</p>	None stated	Date of last menstrual period
<b>Primary hypomagnesemia with secondary hypocalcemia</b>	Tubulopathy	<p>Primary hypomagnesemia with secondary hypocalcemia</p> <p>Genetically confirmed homozygous pathogenic variant in TRPM6</p>	None stated	Date that clinical diagnosis was first made
<b>Pseudohypoaldosteronism type 2A</b>	Tubulopathy	Pseudohypoaldosteronism type 2A	None stated	Date that clinical diagnosis was first made
<b>Pseudohypoaldosteronism type 2B</b>	Tubulopathy	<p>Pseudohypoaldosteronism type 2B</p> <p>Genetically confirmed homozygous pathogenic variant in WNK1</p>	None stated	Date that clinical diagnosis was first made
<b>Pseudohypoaldosteronism type 2C</b>	Tubulopathy	<p>Pseudohypoaldosteronism type 2C</p> <p>Genetically confirmed homozygous pathogenic variant in WNK4</p>	None stated	Date that clinical diagnosis was first made
<b>Pseudohypoaldosteronism type 2D</b>	Tubulopathy	<p>Pseudohypoaldosteronism type 2D</p> <p>Genetically confirmed homozygous pathogenic variant in KLHL3</p>	None stated	Date that clinical diagnosis was first made



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<b>Pseudohypoaldosteronism type 2E</b>	Tubulopathy	<p>Pseudohypoaldosteronism type 2E</p> <p>Genetically confirmed homozygous pathogenic variant in CUL3</p>	None stated	Date that clinical diagnosis was first made
<b>Pure Red Cell Aplasia</b>	PRCA	<p>Treatment with any injectable form of erythropoiesis stimulating agent for at least four weeks.</p> <p>Haemoglobin &lt;70 g/l without transfusion or transfusion dependence.</p> <p>Normal leucocyte and platelet count</p> <p>Reticulocyte count &lt; 20.000 / mm<sup>3</sup></p> <p>Bone marrow aspirate showing well preserved myeloid and megakaryocyte development, and &lt;5% erythroblasts.</p> <p>Presence of anti-erythropoietin antibodies.</p>	Pre-established PRCA due to myeloproliferative disorder	Date of positive antibody test
<b>Renal pseudohypoaldosteronism type 1</b>	Tubulopathy	<p>Renal pseudohypoaldosteronism type 1</p> <p>Genetically confirmed homozygous pathogenic variant in NR3C2</p>		Date that clinical diagnosis was first made

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<b>Retroperitoneal Fibrosis</b>	Retroperitoneal Fibrosis	<p>Any radiologically confirmed retroperitoneal fibrosis (RPF), presumed to be 'idiopathic' or associated with primary conditions including (but not exclusively):</p> <ul style="list-style-type: none"> <li>• Aortitis</li> <li>• Periaortitis</li> <li>• IgG4-related Vasculitis</li> <li>• Perivascular fibrosis</li> <li>• Atherosclerotic or aneurysmal disease</li> </ul> <p><b>Note:</b> There is no specific ICD code for retroperitoneal fibrosis although the diagnosis term links to two ICD codes:</p> <ul style="list-style-type: none"> <li>• ICD10:N13.5 - Crossing vessel and stricture of ureter without hydronephrosis</li> <li>• ICD-9-CM 593.4 - Other ureteric obstruction</li> </ul>	Neoplastic disease within retroperitoneal fibrosis mass defined histologically	Date of diagnostic imaging study report
<b>Tuberous Sclerosis</b>	<b>Tuberous Sclerosis</b>	<p>Clinical or molecular diagnosis of Tuberous Sclerosis Complex (TSC)</p> <p>Multiple renal angiomyolipomas</p> <p>Multiple renal angiomyolipomas (&gt; 3) +/- pulmonary lymphangiomyomatosis (LAM) without other signs of TSC</p>	None stated	Date that clinical diagnosis was first made

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<b>Vasculitis (Primary systemic Vasculitis)</b>	<b>Vasculitis</b>	<p><b>Small vessel Vasculitis (ANCA associated)</b></p> <p>Microscopic polyangiitis (including renal limited Vasculitis)</p> <p>Granulomatosis with polyangiitis (Wegener)</p> <p>Eosinophilic granulomatosis with polyangiitis (Churg Strauss)</p> <p>ANCA Vasculitis unclassified</p> <p><b>Small vessel Vasculitis (Immune complex)</b></p> <p>anti-GBM disease</p> <p>Cryoglobulinemic Vasculitis</p> <p>IgA Vasculitis (Henoch-Schönlein)</p> <p><b>Medium vessel Vasculitis</b></p> <p>Classical PAN</p> <p>Kawasaki disease</p> <p><b>Large vessel Vasculitis</b></p> <p>Giant cell arteritis</p> <p>Takayasu's arteritis</p>	None stated	<p>Date of biopsy.</p> <p>In the absence of a biopsy, the date of a positive antibody test should be used</p>

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<b>Vasculitis (Primary systemic Vasculitis)</b>	Vasculitis	<p><b>Variable vessel Vasculitis</b></p> <p>Behçet's disease</p> <p>Cogan's syndrome</p> <p><b>Single organ Vasculitis</b></p> <p>Isolated aortitis</p> <p>Primary cerebral angiitis</p>	None stated	<p>Date of biopsy.</p> <p>In the absence of a biopsy, the date of a positive antibody test should be used</p>
<b>Inherited Renal Cancer Syndrome</b>	Renal Cancer Inherited	<p>1. A molecular or clinical diagnosis according to standard criteria of any of the following conditions:</p> <p>Von Hippel Lindau disease (VHL) OMIM 193300</p> <p>PTEN hamartoma tumour syndrome (Cowden syndrome) OMIM 158350</p> <p>Birt Hogg Dube syndrome (BHD) OMIM 135150</p> <p>Hereditary leiomyomatosis and renal cell cancer syndrome(HLRCC) OMIM 150800</p> <p>Succinate dehydrogenase-related tumour predisposition syndrome</p> <p>BAP1-related tumour predisposition</p>	None stated	<p>Date of molecular or clinical diagnosis according to standard criteria</p>

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		<p>syndrome OMIM 614327</p> <p>Hereditary Type 1 papillary renal cell carcinoma syndrome (MET oncogene) OMIM 605074</p> <ol style="list-style-type: none"><li>2. Two or more cases in first degree relatives of any type of renal cancer without an established molecular or clinical diagnosis</li><li>3. Bilateral, multiple primary renal cancers of any histopathological type with or with a family history</li></ol>		
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