Unilateral renal agenesis and the congenital solitary functioning kidney: developmental, genetic and clinical perspectives

Adrian S. Woolf and Katherine A. Hillman*

*Nephro-Urology Unit, UCL Institute of Child Health, and *Centre for Nephrology, Royal Free and University College Medical School, London, UK*

Accepted for publication 3 July 2006

KEYWORDS

agenesis, dysplasia, gene, hypertension, proteinuria, renal failure, syndrome

INTRODUCTION

We review the condition termed 'unilateral renal agenesis' (URA), i.e. individuals born with non-ectopic, solitary functioning kidneys (SFKs), with contralateral kidneys which fail to form. We discuss the causes, diagnosis and long-term 'renal prognosis' of URA. We do not consider in detail other anomalies resulting in SFK, such as unilateral multicystic dysplastic kidney (MCDK) and fused renal tracts [1,2]. SFK also follows unilateral nephrectomy for disease and living renal transplant donation; the long-term outcomes for these scenarios have been reviewed [3].

DEVELOPMENTAL BIOLOGY

'RA' implies that the embryonic kidney has failed to begin to form. The human metanephros is the direct precursor of the mature kidney; it becomes a distinct entity in the fifth gestational week when the ureteric bud epithelium branches from the mesonephric duct [4]. Thereafter, renal mesenchyme condenses to envelop the advancing bud, and forms primitive nephron vesicles; the bud forms the ureter and branches to generate collecting ducts. Thus, RA must be the result of either failure of the ureteric bud to arise, or failure of the bud to engage with renal mesenchyme.

INCIDENCE AND DIAGNOSIS

Kiprov *et al.* [5], in a histopathology study, reported an incidence of URA of ≈1 : 1000, based on >9000 (postnatal) autopsies. However, in clinical practice the clinician is generally faced with making a radiological diagnosis of 'URA' in a child or adult, or perhaps in a fetus in which just one kidney has been visualized on ultrasonography (US) during mid-gestation. Using US, URA can be 'missed' in the fetus or neonate because the adrenal, which then occupies the renal bed, can be mistaken for a kidney [6]. Using US, Roodhooft *et al.* [7] reported that URA occurred in ≈1 : 500 of the general population, which is more frequent than the autopsy value cited above [5]. There are caveats about using an 'empty renal bed' assessed by US to diagnose URA. Renal ectopia (e.g. pelvic and cross-fused) kidneys should be considered and sought by US [8] and, if necessary, by functional scanning with IVU and/or isotope renography (e.g. with ^{99m}Tc-DMSA, concentrated in functioning tubules). Furthermore, all these radiological techniques might fail to detect a very small (<2 cm across) contralateral kidney affected by renal 'aplasia' (a tiny, dysplastic organ, containing undifferentiated and metaplastic tissues) or atrophy secondary to renal artery stenosis or neonatal renal venous thrombosis [9–11]. Such 'remnants', confirmed at laparotomy, have rarely been implicated in causing hypertension [10,12]; MRI might prove a sensitive technique with which to find such rudiments [10].

With the advent of routine fetal US screening, it became apparent that dysplastic kidneys, either of modest size, or even greatly enlarged MCDK, can spontaneously involute prenatally or in the first years after birth [10,13,14]. This process might be driven by apoptosis, a metabolically active form of 'cell-suicide', i.e. excess apoptosis [15]. Possibly, URA is rarer than previously considered because some individuals so classified might actually have regressed, malformed kidneys rather than true agenesis.

ASSOCIATED ANOMALIES, AND GENETIC AND TERATOGENIC CAUSATION

Anomalies outside the renal tract can be associated with URA, including absence of a uterine horn [16] or vas deferens [17] ipsilateral to the absent kidney, respectively, emphasizing close relationships between paramesonephric and mesonephric duct development and nephrogenesis itself. Dursun *et al.* [18] detected non-urological anomalies in 44% of 87 consecutive cases of congenital SFK, with cardiac and gastrointestinal malformations being especially common. Sometimes RA occurs as part of a multi-organ syndrome (Table 1), and several of these have defined genetic bases [19–31]. Not unexpectedly, the normal versions of genes mutated in such individuals are expressed during differentiation of normal kidneys and ureters [4]. Gene mutations in branchio-oto-renal [19], Townes Brockes [30] and the renal cysts and diabetes [29] syndromes encode transcription factor and related proteins that modulate expression of other genes; in Fraser [22] and X-linked Kallmann [23,24] syndromes, the relevant proteins are on surfaces of renal epithelia, and probably mediate cell–cell and cell–matrix interactions; in Rokitansky-Kuster-Hauser syndrome, there is a report of mutation of a locally acting growth factor [26].

Even in the absence of non-renal tract features, RA can be familial, sometimes apparently inherited as a dominant trait with incomplete penetrance [7,32,33]. The risk to close relatives might be greater when the index case has bilateral RA, and Roodhooft *et al.* [7] recommend 'ultrasonographic screening for parents and siblings of infants born with agenesis or dysgenesis of both kidneys or with agenesis of one kidney and dysgenesis of the other, since renal malformations may have medical implications even for asymptomatic patients'.

Finally, regarding teratogenic causation, poorly controlled maternal diabetes mellitus and use of specific drugs during pregnancy (e.g. those which inhibit the renin-angiotensin system) have been associated with RA in progeny [34,35]. Other agents, such as high doses of vitamin A derivates, can cause RA in experimental animals [36].

TABLE 1 Multi-organ syndromes associated with RA

Syndrome
Branchio-oto–renal syndrome: *EYA1* (*Eyes Absent 1*) dominant mutation; hearing loss, pre-auricular pits, branchial clefts [19].
DiGeorge syndrome: *deletion of 22q11*: congenital heart disease, hypocalcaemia, immunodeficiency, and neurocognitive disorders [20].
Fanconi anaemia: caused by recessive mutation of several genes; pancytopenia [21].
Fraser syndrome: *FRAS1* autosomal recessive mutations; cryptophthalmos, cutaneous syndactyly, malformations of the larynx and ambiguous genitalia [22].
Kallmann syndrome: *Anosmin-1* X-linked recessive mutation: hypogonadotrophic hypogonadism and anosmia [23,24].
Klinefelter syndrome: *47,XXY*: small, firm testis, gynaecomastia, azoospermia and hypergonadotrophic hypogonadism [25].
Rokitansky-Kuster–Hauser syndrome: *WNT4* (*wingless-type MMTV integration site family member 4*) dominant mutation; absent/rudimentary upper vagina and uterus [26].
MURCS association: genetic basis undefined; Müllerian duct aplasia-hypoplasia (MU), renal malformations (R) and cervicothoracic somite dysplasia (CS) [27].
Poland syndrome: genetic basis undefined; unilateral hypoplasia of pectoralis major muscle and ipsilateral syndactyly [28].
Renal cysts and diabetes syndrome: *HNF1β* (*hepatocyte nuclear factor β*) dominant mutations; diabetes mellitus, hyperuricaemia and uterine malformations [29].
Townes–Brocks syndrome: *SALL1* (*sal-like 1/homologue of Drosophila spalt*) dominant mutation: imperforate anus, triphalangeal/bifid thumb, rocker bottom feet, sensorineural hearing loss, hypospadias [30].
Williams–Beuren syndrome; *deletion of 7q11.23*; developmental delay, cardiovascular anomalies, mental retardation and facial dysmorphology [31].

THE CONTRALATERAL RENAL TRACT

Having diagnosed URA, the next step is to exclude disease of the contralateral renal tract. First, is the SFK 'normal'? Congenital SFKs are often larger than normal [37,38], and overgrowth initiated prenatally; in one study, the lengths of human fetal SFKs were increased in 44% of cases assessed by US, a phenomenon detectable as early as mid-gestation [37]. The remarkable process has been called 'hypertrophy', implying simply an increase in cell size, although other mechanisms are probably operative. In sheep, after experimental unilateral nephrectomy during the period of normal nephrogenesis, final glomerular numbers in the SFK are strikingly greater than in control kidney, bringing the final complement of nephrons in the single kidney to ≈70% of that in two normal kidneys [39]. One histological report which compared a 'healthy (human) congenitally solitary kidney' with a normal control, suggested the former contained more nephrons [40]; however, larger scale studies quantifying glomerular numbers by the 'gold-standard' technique of stereology [41] are needed to confirm this impression. Only 20–40% of individuals with URA have a significant increase in renal length above the upper normal age-adjusted limits [37,38]; one explanation for this is that kidney length is highly variable even in individuals with two normal kidneys [37,42,43].

A congenital SFK of 'normal length' which is echogenically bright, or one which is shorter than the normal range, might well be considered abnormal, and might itself either have a degree of dysplasia/hypoplasia, or have been damaged postnatally. The most severe contralateral tract anomaly (apart from RA itself), is severe renal dysplasia. Indeed, URA and MCDK can coexist [44]; many such fetuses will be terminated or, if born, will soon die from renal failure and lung hypoplasia associated with oligohydramios. Cascio *et al.* [45] reviewed 46 consecutive children with URA diagnosed in one hospital: all had US and renography, and most had cystography; almost half had an anomaly of the contralateral renal tract, including VUR (often high-grade) and 'obstruction', especially at the PUJ. Kaneyama *et al.* [46] found a similar spectrum and incidence of anomalies, and even suggested that 'All children with (congenital) solitary kidney should undergo a screening voiding cysto-urethrography . . .'. While these studies show the range of contralateral anomalies that can occur, index cases in such series might not have been typical of all individuals with URA (e.g. half of the cases analysed by Cascio *et al.* [45] presented with UTI), and hence suggestions for extra investigations in addition to US are difficult to generalize into clinical imperatives.

LONG-TERM PROGNOSIS

The tendency for 'normal' kidneys opposite URA to increase in length can be viewed as a positive, adaptive response. Indeed, in children with uncomplicated URA, SFKs maintain GFRs similar to that of two normal kidneys [38]. However, it has been contended, from rat experiments (reviewed in [3]), that such compensatory growth and functional responses might be detrimental in the long term, with the onset of hypertension, glomerulosclerosis, proteinuria and even progressive renal failure. Is there any evidence that humans, who initially have overtly normal SFKs and lower renal tracts, might acquire a similar spectrum of dysfunction? The answer is 'yes', although frustratingly we cannot yet predict the risk for a specific individual with URA.

In a survey of children with URA, Wasilewska *et al.* [38] found that serum cystatin C, a substance cleared by glomerular filtration, tended to be increased in those over 12 years old, a rise positively correlated with kidney overgrowth. Mei-Zahav *et al.* [47] used ambulatory blood pressure monitoring and concluded that daytime and night-time systolic blood pressures were significantly higher (4–5 mmHg) in children with URA than in controls; increased blood pressure correlated with increased renal length. By contrast, children with unilateral nephrectomy for renal disease had pressures similar to healthy controls. Perhaps these

studies give indications of 'trouble ahead' for children with URA, who are born with presumed nephron deficits. Keller *et al.* [41] quantified glomerular numbers in kidneys of adults (with two kidneys), aged 35–59 years, who died in road traffic accidents, and found that individuals with histories of 'essential hypertension' had on average only half the number of glomeruli/kidney than had age-matched normotensive controls. Although not a study of URA, perhaps there is a message here for the long-term prognosis of individuals with congenital SFKs.

Heinonen [16] studied pregnant women with URA and associated uterine anomalies; outcomes were compared with a control group of age-matched pregnancies in women with similar uterine malformations who had two normal kidneys. The relative risk of gestational hypertension, pre-eclampsia or gestational proteinuria was 2.3, significantly higher. Argueso *et al.* [48] studied 157 adults with 'URA and a normal contralateral kidney' diagnosed at a mean age of 37 years; proteinuria (>50 mg/day) was found in 19% of 37 patients tested, hypertension in 47% of 47 patients, renal function was impaired in 13% of 32 patients and, on follow-up, there were six deaths from chronic renal failure. Duke *et al.* [23] reported that URA, in young adults with Kallmann's syndrome, could be associated with hypertension, proteinuria and progressive renal failure. Gonzalez *et al.* [49], in a retrospective study of 33 adults with URA, found that those with hypertension, proteinuria and renal insufficiency at diagnosis had a higher body mass index than had those lacking these signs at diagnosis; in the former group, progressive renal impairment was less common in those treated with drugs which block angiotensin II, while in the latter group, onset of proteinuria and renal impairment was more frequent in those patients with an increased body mass index.

With hindsight, it is difficult to be sure that some individuals in these reports did not have regressed unilateral MCDKs; this might be important because hypertension has (rarely) be attributed to such remnants themselves [12]. However, such studies raise concerns about the long-term renal prognosis of individuals with URA. The histology of SFKs in URA patients with a history of proteinuria generally shows glomerulosclerosis [5,33], but the decision to biopsy a SFK during life must be weighed against the greater significance of complications after such a procedure than in an individual with two functioning kidneys. Kiprov *et al.* [5] reviewed 29 cases of histologically confirmed focal glomerulosclerosis and found that five had URA; in the same study, in 9200 autopsies, seven had URA and, of these, two had died from chronic renal failure and had glomerulosclerosis.

CONCLUSIONS

URA occurs in ≈1 : 500–1000 individuals; from the practical clinical perspective, we suggest that the diagnosis of 'URA' should prompt the clinician to consider several points:

- Give consideration to alternative diagnoses of ectopic kidney or a regressed dysplastic kidney; in the latter, remnants have been reported to cause hypertension.
- Did the individual's mother have diabetes mellitus or was she treated for hypertension during the index cases' gestation?; hyperglycaemia and drugs inhibiting angiotensin II action can be teratogenic and are associated with RA.
- Anomalies of other organ systems can occur, and sometimes the picture will fit a specific syndrome (Table 1); in addition, RA can be familial, even with disease confined to the renal tract. Such kindreds might benefit from genetic counselling and screening of asymptomatic relatives.
- Ascertain the structure of the contralateral renal tract, at least by US; in particular, check whether the solitary kidney shows normal 'compensatory hypertrophy' (i.e. is longer than normal) and exclude hydronephrosis (in which case further ascertainment might be needed to define/exclude urine flow impairment or VUR).
- For 'uncomplicated cases', secure life-long follow-up of blood pressure and urinary protein checks every year or two. Consider dietary advice to normalize an increased body mass index.
- The finding of structural anomalies of the solitary kidney and/or lower renal tract might demand appropriate specialized urological observation or surgery.
- Finding hypertension, proteinuria or impaired renal function will demand appropriate specialized nephrological follow-up; such patients might benefit from specific interventions such as treatment with angiotensin-converting enzyme inhibitors and dietary advice to normalize an increased body mass index.

Suggestions for long-term follow-up and treatments are given with the caveat that we currently lack good evidence with which to accurately predict the risk of life-long complications for any individual with URA.

CONFLICT OF INTEREST

None declared.

REFERENCES

1 **Woolf AS.** Unilateral multicystic dysplastic kidney. *Kidney Int* 2006; **69**: 190–3
2 **Fekak H, Mezzour MH, Rabii R, Joual A, Bennani S, El Mrini M.** [Management of horseshoe kidney based on a series of 36 cases]. *Prog Urol* 2004; **14**: 485–8
3 **Woolf AS.** The single kidney. In Stringer MD, Oldham KT, Mouriquand PD eds, *Pediatric Surgery and Urology: Long-Term Outcomes*, Chapt. 54. Cambridge, UK: Cambridge University Press, in press
4 **Woolf AS, Jenkins D.** Development of the kidney. In Jennette JC, Olson JL, Schwartz MM, Silva FG eds *Heptinstall's Pathology of the Kidney*, 6th edn, Chapt. 2. Philadelphia, New York: Lippincott-Raven, in press
5 **Kiprov DD, Calvin RB, McLuskey RT.** Focal and segmental glomerulosclerosis and proteinuria associated with unilateral renal agenesis. *Lab Invest* 1982; **46**: 275–81
6 **Barwick TD, Malhotra A, Webb JA, Savage MO, Reznek RH.** Embryology of the adrenal glands and its relevance to diagnostic imaging. *Clin Radiol* 2005; **60**: 953–9
7 **Roodhooft AM, Birnholz JC, Holmes LB.** Familial nature of congenital absence and severe dysgenesis of both kidneys. *N Engl J Med* 1984; **310**: 1341–5
8 **Yuksel A, Batukan C.** Sonographic findings of fetuses with an empty renal fossa and normal amniotic fluid volume. *Fetal Diagn Ther* 2004; **19**: 525–32
9 **Luque-Mialdea R, Martín-Crespo R, Cebrian J, Moreno L, Carrero C, Fernández A.** Does the multicystic dysplastic kidney really involute? The role of the retroperitoneoscopic approach.

J Pediatr Urol 2006 [Epub ahead of publication], doi 10.1016/j.jpurol.2006.01.012.

10 **Valentini RP, Langenburg S, Imam A, Mattoo TK, Zerin JM.** MRI detection of atrophic kidney in a hypertensive child with a single kidney. *Pediatr Nephrol* 2005; **20**: 1192–4

11 **Winyard PJ, Bharucha T, De Bruyn R *et al.*** Perinatal renal venous thrombosis: presenting renal length predicts outcome. *Arch Dis Child Fetal Neonatal* 2006; **91**: F273–8

12 **Webb NJ, Lewis MA, Bruce J *et al.*** Unilateral multicystic dysplastic kidney: the case for nephrectomy. *Arch Dis Child* 1997; **76**: 31–4

13 **Hiraoka M, Tsukahara H, Ohshima Y, Kasuga K, Ishihara Y, Mayumi M.** Renal aplasia is the predominant cause of congenital solitary kidneys. *Kidney Int* 2002; **61**: 1840–4

14 **Rabelo EA, Oliveira EA, Diniz JS *et al.*** Natural history of multicystic kidney conservatively managed: a prospective study. *Pediatr Nephrol* 2004; **19**: 1102–7

15 **Winyard PJ, Nauta J, Lirenman DS *et al.*** Deregulation of cell survival in cystic and dysplastic renal development. *Kidney Int* 1996; **49**: 135–46

16 **Heinonen PK.** Gestational hypertension and preeclampsia associated with unilateral renal agenesis in women with uterine malformations. *Eur J Obstet Gynecol Reprod Biol* 2004; **114**: 39–43

17 **Kolettis PN, Sandlow JI.** Clinical and genetic features of patients with congenital unilateral absence of the vas deferens. *Urology* 2002; **60**: 1073–6

18 **Dursun H, Bayazit AK, Buyukcelik M, Soran M, Noyan A, Anarat A.** Associated anomalies in children with congenital solitary functioning kidney. *Pediatr Surg Int* 2005; **21**: 456–9

19 **Pierides AM, Athanasiou Y, Demetriou K, Koptides M, Deltas CC.** A family with the branchio-oto-renal syndrome: clinical and genetic correlations. *Nephrol Dial Transplant* 2002; **17**: 1014–8

20 **Miyane S, Itoh S, Imai T *et al.*** Case of CATCH 22 syndrome complicated with pseudohypoparathyroidism and unilateral renal aplasia. *Pediatr Int* 2002; **44**: 109–11

21 **Habib Z, Abudaia J, Bamehriz F, Ahmed S.** Fanconi's anemia with solitary crossed renal ectopia, vesicoureteric reflux, and genital abnormalities. *Pediatr Surg Int* 2000; **16**: 136–7

22 **McGregor L, Makela V, Darling SM *et al.*** Fraser syndrome and mouse blebbed phenotype caused by mutations in FRAS1/Fras1 encoding a putative extracellular matrix protein. *Nat Genet* 2003; **34**: 203–8

23 **Duke V, Quinton R, Gordon I, Bouloux PM, Woolf AS.** Proteinuria, hypertension and chronic renal failure in X-linked Kallmann's syndrome, a defined genetic cause of solitary functioning kidney. *Nephrol Dial Transplant* 1998; **13**: 1998–2003

24 **Hardelin JP, Julliard AK, Moniot B *et al.*** Anosmin-1 is a regionally restricted component of basement membranes and interstitial matrices during organogenesis: implications for the developmental anomalies of X chromosome-linked Kallmann syndrome. *Dev Dyn* 1999; **215**: 26–44

25 **Ozata M, Yesilova Z, Saglam M, Tunca Y.** A case of Klinefelter's syndrome associated with unilateral renal aplasia. *Med Sci Monit* 2000; **6**: 1000–2

26 **Biason-Lauber A, Konrad D, Navratil F, Schoenle EJ.** A WNT4 mutation associated with Mullerian-duct regression and virilization in a 46,XX woman. *N Engl J Med* 2004; **351**: 792–8

27 **Pittock ST, Babovic-Vuksanovic D, Lteif A.** Mayer-Rokitansky-Kuster-Hauser anomaly and its associated malformations. *Am J Med Genet A* 2005; **135**: 314–6

28 **Assadi FK, Salem M.** Poland syndrome associated with renal agenesis. *Pediatr Nephrol* 2002; **17**: 269–71

29 **Bingham C, Ellard S, Cole TR *et al.*** Solitary functioning kidney and diverse genital tract malformations associated with hepatocyte nuclear factor-1β mutations. *Kidney Int* 2002; **61**: 1243–51

30 **Salerno A, Kohlhase J, Kaplan BS.** Townes–Brocks syndrome and renal dysplasia: a novel mutation in the SALL1 gene. *Pediatr Nephrol* 2000; **14**: 25–8

31 **Sforzini C, Milani D, Fossali E *et al.*** Renal tract ultrasonography and calcium homeostasis in Williams-Beuren syndrome. *Pediatr Nephrol* 2002; **17**: 899–902

32 **McPherson E, Carey J, Kramer A *et al.*** Dominantly inherited renal adysplasia. *Am J Med Genet* 1987; **26**: 863–72

33 **Arfeen S, Rosborough D, Luger AM, Nolph KD.** Familial unilateral renal agenesis and focal and segmental glomerulosclerosis. *Am J Kidney Dis* 1993; **21**: 663–8

34 **Boix E, Zapater P, Pico A, Moreno O.** Teratogenicity with angiotensin II receptor antagonists in pregnancy. *J Endocrinol Invest* 2005; **28**: 1029–31

35 **Nielsen GL, Norgard B, Puho E, Rothman KJ, Sorensen HT, Czeizel AE.** Risk of specific congenital abnormalities in offspring of women with diabetes. *Diabet Med* 2005; **22**: 693–6

36 **Tse HK, Leung MB, Woolf AS *et al.*** Implication of Wt1 in the pathogenesis of nephrogenic failure in a mouse model of retinoic acid-induced caudal regression syndrome. *Am J Pathol* 2005; **166**: 1295–307

37 **Hill LM, Nowak A, Hartle R, Tush B.** Fetal compensatory renal hypertrophy with a unilateral functioning kidney. *Ultrasound Obstet Gynecol* 2000; **15**: 191–3

38 **Wasilewska A, Zoch-Zwierz W, Jadeszko I *et al.*** Assessment of serum cystatin C in children with congenital solitary kidney. *Pediatr Nephrol* 2006; **21**: 688–93

39 **Douglas-Denton R, Moritz KM, Bertram JF, Wintour EM.** Compensatory renal growth after unilateral nephrectomy in the ovine fetus. *J Am Soc Nephrol* 2002; **13**: 406–10

40 **Maluf NS.** On the enlargement of the normal congenitally solitary kidney. *Br J Urol* 1997; **79**: 836–41

41 **Keller G, Zimmer G, Mall G, Ritz E, Amann K.** Nephron number in patients with essential hypertension. *N Engl J Med* 2003; **348**: 101–8

42 **Rosenbaum DM, Korngold E, Teele RL.** Sonographic assessment of renal length in normal children. *AJR Am J Roentgenol* 1984; **142**: 467–9

43 **Emamian SA, Nielsen MB, Pedersen JF, Ytte L.** Kidney dimensions at sonography: correlation with age, sex, and habitus in 665 adult volunteers. *AJR Am J Roentgenol* 1993; **160**: 83–6

44 **Damen-Elias HA, Stoutenbeek PH, Visser GH, Nikkels PG, de Jong TP.** Concomitant anomalies in 100 children with unilateral multicystic kidney. *Ultrasound Obstet Gynecol* 2005; **25**: 384–8

45 **Cascio S, Paran S, Puri P.** Associated urological anomalies in children with unilateral renal agenesis. *J Urol* 1999; **162**: 1081–3

46 **Kaneyama K, Yamataka A, Satake S *et al.*** Associated urologic anomalies in children with solitary kidney. *J Pediatr Surg* 2004; **39**: 85–7
47 **Mei-Zahav M, Korzets Z, Cohen I *et al.*** Ambulatory blood pressure monitoring in children with a solitary kidney – a comparison between unilateral renal agenesis and uninephrectomy. *Blood Press Monit* 2001; **6**: 263–7
48 **Argueso LR, Ritchey ML, Boyle ET Jr, Milliner DS, Bergstralh EJ, Kramer SA.** Prognosis of patients with unilateral renal agenesis. *Pediatr Nephrol* 1992; **6**: 412–6
49 **Gonzalez E, Gutierrez E, Morales E *et al.*** Factors influencing the progression of renal damage in patients with unilateral renal agenesis and remnant kidney. *Kidney Int* 2005; **68**: 263–70

Correspondence: Prof Adrian S. Woolf, Nephro-Urology Unit, UCL Institute of Child Health, 30 Guilford Street, London WC1N 1EH, UK.
e-mail: a.woolf@ich.ucl.ac.uk

Abbreviations: **MCDK**, multicystic dysplastic kidney; **RA**, renal agenesis; **SFK**, solitary functioning kidney; **URA**, unilateral renal agenesis; **US**, ultrasonography.