Unilateral Renal Agenesis: A Case Report

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Abstract

Unilateral renal agenesis is the congenital absence of one kidney. The incidence ranges from 1:500 to 1:3200 with a slight male preponderence. The diagnosis is usually made as an incidental finding. Complications such as hypertention, proteinuria and renal impairemnt are commoner in this condition than in those with both kidneys, though majority will lead a normal life.

We present a four year old girl who presented with anarsaca and proteinuria and was found to have a solitary left kidney. She reponded to prednisolone but is currently on captopril and levamisol and has been asymptomatic for five years

Keywords:

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Introduction

Unilateral renal agenesis is development of a sngle kiney. It can occur in isolation or combined with other abnormalities.[1] the condition has been known since the time of Aristotle.[2,3] About a quarter of all renal agenesis is associated with genital abnormalities, making it possible to determine the point in time when the anomaly appeared in the emryonic life.[4] It may occur with mullerian duct abnormalities,[5] or as an association with other systemic congenital defects.[6] Its association with mullerian agenesis could be an autosomal dominantly inherited disorder[7]. The incidence in adult life ranges from 1:500 and 1:320. [8].

We present here a four year old girl who presented with unilateral renal agenesis and review of the literature on the subject.

Case Presentation

A four year old girl presented in our clinic with a three week history of periorbital swelling, initially worse in the morning but later became persistent throughout the day. There was associated abdominal and leg swellings with passage of cola-coloured urine but with no change in the frequency or volume of urine. There was no history of rashes or sore throat ante-dating the onset of symptoms. She had severe neonatal jaundice at age four days requiring an exchange blood transfusion, and has been apparently well before the onset of symptoms, apart from occasional malaria. The mother is not a diabetic and neither smokes or drinks, and had no illness during the pregnancy of the child.

Clinical examination showed her to have normal vital signs with a blood pressure of 80/50mmHg. She had anarsaca with fine basal crepitation in both lungs. There was no tenderness or palpable

organs in the abdomen.

A diagnosis of nephrotic syndrome was made witha differential dianosis of acute glomerulonephritis. Broncho-puemonia waas also diagnosed. The investigations ordered for include, full blood count, ESR, serum uera, electrolytes and creatinine, serum protein, ASO titer, urinalysis, urine microscopy culture and sensitivity, 24hour urinary protein and plain chest X-ray. The laboratory results are shown in Tables 1 and 2. The haemoglobin was 9.6g/dl, packed cell volume, 28%, (Table1). The electrolyte profile showed sodium of 129mmol/l and chloride 85mmol/l. The serum albumin was 2.5g/dl and the ASO titre was 125Todds unit (normal range 120-160Todss units). Urinalysis showed 1+ of protein on dipstick examination and the 24hour urinary protein was 110mg, with a urine volume of 1.2litres in 24hours. Plain chest X-ray showed bilateral patchy opacities.

She was started on prednisolone, paracetamol, atermasimin, amodiaquine and frusemide.

The edema regressed but she represented one month later with similar symptoms. An abdominal ultrasonography revealed a single kidney on the left with absent right kidney. (Fig 1). She was then given another course of prednisolone for four weeks and tapered down for another two weeks. She is currently on captopril and levamisol and has remained asymptomatic for four years now.

Discussion.

Unilateral renal agenesis occurs more in males with a male:female ratio of 1.2-2.3: [1].This has been attributed to the fact that the Wolfian duct

Table 1: *Haematological indices of case (values in bracket are the normal range)*

Haematologic Index	Laboratory Value
Haemoglobin	9.6g/dl (12-15g/dl)
Packed Cell Volume	28% (36-46%)
Total White Cell Count	6800 (4000-10000)
Neutrophils	46% (40-75%)
Lymphocytes	49% (20-40%)
Monocytes	2% (0-10%)
Esinophils	3% (0-6%)
Basophils	0% (0-1%)

Table 2: *Clinical Chemistry Results of Case (values in bracket are the normal range)*

Substance	Laboratory Value
Serum Urea	20mg/dl (10-55mg/dl)
Serum Creatinine	0.7mg/dl (0.6-1.2mg/dl)
Serum Sodium	129mmol/l (133-146mmol/l)
Serum Potasium	4.0mmol/l (3.5-5.5mmol/l)
Serum Chloride	85mmol/l (97-106mmol/l)
Serum Bicarbonate	26mmol/l (24-32mmol/l)
Serum Protein (Total)	5.4g/dl (6-8g/dl)
Serum Albumin	2.5g/dl (3.2-5.5g/dl)
Serum Globulins	2.9g/dl (2-3g/dl)
Aso Titer:	125Todds units (120-160T U)
24hour Urine Protein	110mg (less than 1g)
24hour Urine Volume	1.2litres (normal)

differentiates earlier than the Mullerian duct and at about the time of the ureteric bud formation. 2Unilateral renal agenesis occurs more on the left3. This is unlike in our patient who is a girl and with right renal agenesis.. The laboratory data showed she had anaemia, hyponatremia and hypochloraemia, all of which are dilutional. There is also hypoalbuminaemia probably due to the proteinuria, which is however not in the nephrotic range.

Renal agenesis is a developmental abnormality occuring at 4-6 weeks of embryonic life. Normal kidney development requires the following three events take place: first the ureteric buds must arise bilaterally from the mesonephric (wolffian) ducts, secondly the bilateral metanephric blastema must form from the mesoderm in the caudal region of the nephrogenic cord and thirdly, ureteric buds must grow, contact and invaginate the metanephric blastema, thereby inducing differentiation of theblastema into two mature kidneys.[9]. Failure of the metanephros to develop results in complete absence of the kidney. This can either be due to nonexistence of the ureteric bud or failure of the unilateral bud to develop from the metanephric duct.[9] Most cases of unilateral renal agenesis results from lack of induction of the metanephric blastema by the ureteric bud, though some may be due to in utero regression of a multicystic dysplastic kidney.10 Maternal febrile illness, medication use especially those that affect the renin-angiotensin system, cocaine,smoking and alcohol comsunption have been implicated in this condition.[2,11] So also maternal diabetes12Others have implicated diabetes.[13]

Diagnosis in majority of cases is made as an incidental finding.[14] Our patient however presented with generalized edema and proteiunuria, making us make a diagnosis of nephrotic syndrome. Occassionally, what is diagnosed assolitary kidney may be a rudimentry dysplastic nonfunctional kidney.[15]

Renal agenesis survellance in the USA, has shown an increase in its incidence[16]

Unlike bilateral renal agenesis which is not compartible with life, or infact babies born as stillbirths, patients with single kidney often lead a normal life, with compensatory hypertrophy of the single kidney by upto 10%.[17] This begins prenatally[18.] There is however a controversy about people with this condition participating in vigorous/contact sports. Those against their involvement are of the opinion that damage to that kidney im those events will be catastrphic.[19] however those in favour argue that there other single organs in the body which have not caused exclusion or limitation in sports participation.[20] Infact research has shown that head injuries are more likely to occur in such sports as cycling than kiney injuries.[21]

Prognosis of of this condition has shown that the relative risk of gestational hypertention, pre-eclampsia or gestational proteinuria is about 2-3 times higer. Argueso et al in a study of 157adults with this condition diagnosed at mean age of 37yrs, proteinuria (>50mg/day), was found in 19% of 37patients tested, hypertension in 47%, of 47 tested, and renal impairement in 13% of 32patients with six deaths on follow up.[22]. Gonzalez *et al* in a retrospective study of 33 adults with this condition found that those with hypertension, proteinuria and renal insufficiency had higher body mass index than those lacking this sign at diagnosis. They also found that progressive renal failure was less common in those treated with drugs that block angiotensiin II.[23]. Our patient is currently on captopril and levamisol to slow proteinura

Conclusion

Unilateral renal agenesis is an uncommon condition and if documented, follow up of such patients is necessary especially if evidence of renal pathology is present. Although the

proteinuria in our patient is not in the nephrotic range, persistent proteinuria can lead to chronic renal impairement[23] An excretory urography and magnetic resonance imaging are necessary in our patient to rule out such conditions as hypoplastic or rudimentary kidney which may cause hypertension as shown in a study.[24]

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