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Unilateral Shrunken Kidney in a 58 Year Old Nigerian Woman with Kidney Stone

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ABSTRACT

Background Kidney stones are formed as a result of interplay between factors promoting and those inhibiting stone formation in the kidneys/urinary tracts. Kidney stones cause a lot of complications including urinary tract obstruction. Failure or delay in relieving the obstruction causes irreversible damage to the kidney and urinary tract leading to loss of function and atrophy of the kidney.

Mrs. AT was a 58 year old woman referred from a general hospital with an 8 year history of right lumbar pain. The clinical examination and investigations revealed that she had atrophy and loss of function of the right kidney secondary to nephrolithiasis.

The objective of this case study is to highlight that kidney stone can lead to unilateral kidney atrophy and loss of function.

INTRODUCTION

Renal stone is a crystal aggregate embedded in a small amount of glycoprotein matrix. The initial formation is usually at the collecting duct resulting either from super saturation of stone forming constituents or damage and dysfunction of the renal tubule. The prevalence is 36-100 per 105, increasing with age, more in whites and males [1, 2]. This variability has been attributed to various demographic, geographic and physiological factors [3,4,5,6]

The complications associated with renal stones are pyelonephritis, pyonephrosis, septicaemia, urinary fistula, ureteric scaring, stenosis and perforation. Obstruction of the urinary tracts by renal stones leads initially to enlargement/dilatation of the urinary tract (hydroureter) and kidneys (hydronephrosis), but failure/delay in relieving the obstruction leads to kidney atrophy. There is associated impairment in renal function in presence of urinary tract obstruction manifesting as acute or chronic renal failure[7].

The objective of this case report is to draw attention to the fact that kidney stones can lead to unilateral kidney atrophy and loss of function.

CASE REPORT

Mrs. AT was a 58 year old woman referred from a general hospital with an 8 year history of right lumbar pain. The pain was initially colicky but later became continuous and dull in nature, moderate to severe, radiating to the groin with no relieving or aggravating factors. The pain was recurrent with periods of remission and exacerbation, but later became non-remitant for about a year. She has had recurrent low grade fever associated with rigors but no nausea, vomiting or anorexia. She had taken a variety of analgesics for the pain but denied use of any herbal preparation.

A month prior to presentation, she noticed reduction in her urine volume. This was not associated

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with dysuria, haematuria, nocturia or frothiness of urine.

There was no body, leg or facial swelling and no seizures or loss of consciousness. She had no cough, dyspnoea, orthopnoea or paroxysmal nocturnal dyspnoea. She was a petty trader and her diet consisted mainly of staple food in her locality.

She had been managed in various private hospitals for the abdominal pain and was diagnosed hypertensive 3 years earlier in the referral hospital and was on alpha methyl dopa and nifedipine. She is not a known diabetic. She had never used tobacco in any form and denied use of alcohol.

Clinical examination revealed middle aged anxious looking woman, who was not pale, not dehydrated and had no peripheral oedema. Her pulse rate was 84 beats/minute; blood pressure 194/90mmHg supine; JVP not raised. There was no clinical evidence of cardiomegaly or left ventricular hypertrophy. First and second heart sounds only were heard, no murmur. Chest was clinically clear on auscultation.

Abdominal examination revealed no area of tenderness, no organ palpably enlarged, no ascites and no bruit heard.

Laboratory investigations from the referral hospital revealed that the urinalysis was normal; urine microscopy showed many calcium oxalate crystals as the only abnormality, urine culture yielded no significant growth. The abdominal scan done 5 months prior to referral showed hydronephrosis of the right kidney, other viscera were normal and no ascites.

A working diagnosis of right hydronephrosis secondary to kidney stones in poorly controlled hypertension was made. Repeat laboratory investigations in our centre revealed many calcium oxalate crystals on urine microscopy, serum urea of 45mg/dl, creatinine 1.6mg/dl, sodium 135mmol/l, potassium 4.5mmol/l, bicarbonate 23mmol/l, chloride 108mmol/l, calcium 8.2mg/dl, and phosphate 4.8mg/dl and estimated glomerular filtration rate of 46ml/minute.

Abdominal ultrasound showed right kidney that was shrunken and atrophic measuring 4.5 × 2.8 cm, hyperechoic with ablation of the corticomedullary junction. The left kidney was 12.1 × 4.2 cm in size with normal echotexture and cortico medullary differentiation. The liver, adrenals and billiary tract were normal.

An intravenous urography showed radio opaque density in the right pelvis just above the right iliac bone in the scout film. There was prompt excretion of contrast by the left kidney. The left kidney was enlarged but normal in shape, position, outline and alignment. The left pelicalyceal system and ureter were grossly normal. There was no excretion of contrast by the right kidney, and the right ureter was not opacified. The bladder filled out well with regular margin and the post micturition film showed no residual urine.

The chest X ray, electrocardiography, packed cell volume and blood film was normal. A final diagnosis of acute renal failure secondary to kidney stones with unilateral kidney atrophy and poorly controlled hypertension was made.

She was placed on tablets nifedipine retard 20mg twice daily, atenolol 50mg daily, and alpha methyl dopa 250mg thrice daily, dietary counseling, and advised on liberal fluid intake. She was referred to the urologist for review and possible nephrectomy of the atrophied kidney.

She has been compliant on medications and has been regular on follow up. Her blood pressure control has been fair, and was 130/90mmHg at last visit. During her outpatient visit serum urea was 20mg/dl, creatinine 1.2mg/dl, and electrolytes were within normal. The surgeons reviewed the patient for possible nephrectomy of the atrophic kidney but she declined surgery.

DISCUSSION

The theories on the formation of stones in the kidney are the free, and the fixed particles theory. The free particle theory is that there is super saturation of the stone-forming product in the urine while the fixed theory is that there is injury or dysfunction in the renal tubules, which attracts the stone forming substances. Inhibitors and promoters of renal stone formation plays significant role. The inhibitors are Tammhorsfall protein, nephrocalcin, glycosaminoglycans, ribonucleic acid, pyrophosphate, citrate and some trace elements. The promoters include presence of calcium oxalate in urine, hypercalcaemia, hypercalciuria, uricosuria, hyperuricaemia and dehydration8. Reduction of inhibitors or elevation of promoters encourages stone formation[3]. Although this patient's urinary calcium and uric acid were not estimated she had significant calcium oxalate in the urine.

The clinical manifestation of patients with renal stone depends on the site, size, mobility, and types of stone. It also depends on the presence or absence of

concomitant infection. Patients with small stones are usually asymptomatic and may be diagnosed incidentally by abdominal imaging or passage of the stone in urine. In the moderately sized or large stone, there is usually flank abdominal pain, which may be unilateral as in our patient, or bilateral if both kidneys are affected. The pain is either colicky when the stone is in the ureter or continuous when the stone is in the kidney or completely obstructing the ureter. Pain may be mild or severe associated with nausea and vomiting, usually radiating to the groin. Other manifestations are haematuria, recurrent urinary tract infection. Post renal acute renal failure as in our patient may be the mode of presentation in patients with complete obstruction of ureter (in a solitary kidney), bladder, urethra or rarely bilateral obstruction of both ureters. Loss of affected kidneys as in our patient occurs when the obstruction is longstanding without relief [9, 10. 11, 12]. Our patient had initially recurrent colicky flank pain, which later became continuous and severe. The onset of oliguria was the only pointer to impaired renal function due to post renal urinary obstruction from stones. The renal calculi probably led to complete loss of function on the affected kidney as detected by the IVU.

Complete urinary tract obstruction lasting more than 6 weeks leads to irreversible loss of renal function [7, 13,] Sequence of events following the obstruction shows that there is vasodilatation of preglomerular blood vessels leading to increase renal blood flow thus maintaining the glomerular filtration rate (GFR) for few hours following the obstruction.

Twenty – four hours later there is increase in preglomerular vascular resistance with reduction in renal blood flow, and GFR falls to 40-50% of normal. Persistence of the obstruction leads to further fall in GFR to 30% in 6 days, 20% in 2 weeks and 12% in 8 weeks, after which the obstruction is irreversible[9].

These changes are mediated through various chemical mediators like nitric oxide, prostaglandins, angiotensin etc released by the macrophages, T-cells and juxtaglomerular cells[14].

Depending on duration of obstruction, kidneys completely obstructed by stone shows either hydronephrosis, or atrophy as in our patient. The histology varies from interstitial inflammation with cellular infiltration, tubular dilation and tubulo interstitial fibrosis[15]. The right kidney of our patient was atrophic without evidence of function on intravenous urography. It is possible that our patient may have had incomplete ureteric obstruction over the years and later became complete before presentation. The IVU detected a radiopaque object, possibly kidney stone, at the point the right ureter crosses the pelvic brim, and this we suspect to be the cause of the obstruction and subsequent atrophy of the right kidney.

Prevention of stone formation and its enlargement, with elimination of uric acid and cystine stones is achieved by avoiding dehydration, maintaining appropriate urinary flow and PH16. Patients are encouraged to take liberal fluid, to void at least 2 litres of urine daily. Urine PH is made acidic in patients with calcium oxalate stones, and alkaline in uric acid stones. Non-steroidal or narcotic analgesic is used to control pain. There were ongoing trials of steroid and nifedipine to reduce ureteric inflammation and ureteric colic respectively, but are yet to be approved [2, 17].

Various surgical interventions in nephrolithiasis are available. Indications for surgery are intractable pain, recurrent infections, and obstructions. Surgery is contraindicated in patient with active urinary tract infection, bleeding diathesis and pregnancy. The surgical procedures are extracorporal ballistic short wave lithotripsy (EBSWL), ureteroscopy with lithotripter, percutaneous nephrostolithotomy and nephrectomy which can be partial or total.[18]

In our environment, the medical management is available, and effective when diagnosis is early and patient compliant to therapy. This prevents formation of stone in 98% of patients at risk, prevents recurrence in 60%, and eliminates stones in 70% of patients[2, 18]. This can ameliorate morbidity, and prevent loss of the kidney as in our patient.

We advocate early diagnosis and prompt intervention to forestall complications such as was encountered in this case reported.

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