

Unilateral hypertrophied right kidney and contralateral atrophy of left kidney: In a setting of malignant hypertension – A case report

Rupshikha Dutta,^a Manash Jyoti Phukon,^b G Anandam^c

^aAssociate Professor, Department of Anatomy, ^bProfessor, Department of Anatomy, ^cProfessor and Head, Department of Pathology, Prathima Institute of Medical Sciences, Karimnagar, Andhra Pradesh, India.

Address for correspondence: Dr. Manash Jyoti Phukon, Professor, Department of Anatomy, Prathima Institute of Medical Sciences, Karimnagar, Andhra Pradesh, India.

Email: manash06@yahoo.co.in

ABSTRACT

During routine dissection of cadavers for 1st MBBS students in 2009-2010 a normal built adult male cadaver presented with a hypertrophied right kidney and an atrophied left kidney. In the right kidney (16cmx6.5cmx4.5cm) the capsule was difficult to strip off. The cut section showed well demarcated cortex and medulla. Blood clots were present in renal sinuses. Histopathology showed glomerulosclerosis, thyroid casts in atrophic tubules thickening and hyalinization of blood vessels, inflammatory changes in the interstitium. The left kidney (4cmx2.5cmx1cm) showed thinned out

cortex on cut section. Calyces were not well distinguished. Capsule was adherent to its surface. Glomerulosclerosis and predominant atrophic changes in the tubules observed in histopathology.

Vascular compromise secondary to hypertension caused ischaemic changes in the left kidney. This superimposed with chronic renal infection had resulted into an atrophic left kidney which led to a compensatory hypertrophy of the right kidney.

Key words –Hypertrophied kidney, atrophic kidney, renal failure, hypertension, renal infection

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INTRODUCTION

The kidneys are a pair of essential excretory organs which are placed retroperitoneally on each side of vertebral column, extending from T12 to L3 vertebra. The kidneys in the fresh state are reddish brown in colour. They are bean shaped with hilum directed medially. It is enclosed by a fibrous capsule which can be stripped off except at the hilum. The kidneys are supplied by the renal arteries and drained by the renal veins into the inferior vena cava. The kidney is composed of multiple uriniferous tubules bounded by a delicate connective tissue in which runs blood vessels, lymphatics and nerves. Each tubule consist of two embryologically distinct parts the nephron and the collecting duct. Any insult to the kidneys in the form of infection(acute or chronic), toxins, systemic disease like hypertension, stenosis of renal artery etc. can cause macroscopic and microscopic changes in the kidney which may lead to renal failure.^{1,2}

CASE REPORT

During routine dissection of cadavers for 1st MBBS students in 2009-2010 a normal built adult male cadaver presented with a hypertrophied right kidney and an atrophied left kidney.

The right kidney measured 16cmx6.5cmx4.5cm and was heavier than the normal. The capsule could not be easily peeled off. The cortical surface was smooth but appeared dark reddish brown. On cut section, cortex and medulla were well demarcated. The number of calyces were increased. The renal artery showed multiple branching at the hilum.

The left kidney was found to be atrophic, measuring about 4cm x 2.5cmx 1cm. The capsule was adherent to the underlying scar tissue. Surface of the kidney appeared reddish white and uneven. On cut section, the cortex and medulla were found to be ill defined. The kidney was supplied by a single renal artery at the hilum.

In the present case, histopathology of the right kidney showed glomeruli-shrunken, sclerosed, bowmen space increased. Tubules-atrophied, degenerated with thyroid cast. Interstitium- showed dense lymphoid and neutrophilic aggregates and interstitial fibrosis. Vasculature- thickened and hyalinized large and medium size vessels. Small arteries show arteriosclerosis at places showing onion skin appearance. All these findings are suggestive of renal hypertension. Left kidney showed same morphology but predominant atrophic changes in tubules without much inflammation and interstitial fibrosis.

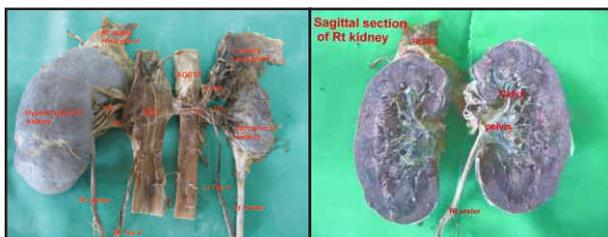


Figure 1

Figure 2

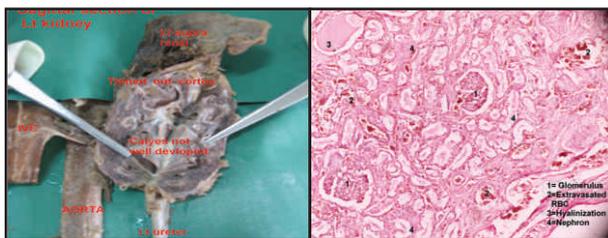


Figure 3

Figure 4

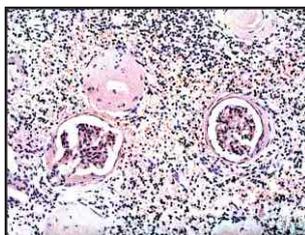


Figure 5

Fig1 – Specimen of both kidneys

Fig2- Cut section of right kidney

Fig 3- Cut section of left kidney

Fig 4 : Histo-pathological features of hypertrophied kidney

Fig 5: Histo-pathological features of atrophied kidney

DISCUSSION

Hypertrophy means enlargement or overgrowth of an organ or part of the organ due to increase in the size of its constituent cells. The cells of the kidney are particularly prone to hypertrophy. Hypertrophy

of kidney may be due to physiologic or pathologic conditions.

The common causes of enlarged kidney are medullary sponge kidney, malignant hypertension, rapidly progressive glomerulonephritis, membranous glomerulonephritis, ischaemic acute tubular necrosis, toxic acute tubular necrosis.³

Atrophy is the reduction of the number and size of parenchymal cells of an organ, or its parts which were once normal. Atrophy may be physiological or pathological. Small atrophic kidney is due to atherosclerosis of renal artery.⁴ Size may also be reduced and scarred because of pre existing arterial narrowing or stenosis.⁵

Hypertensive renal disease is the second most common form of end stage renal disease after diabetic nephropathy. The incidence is high in African Americans than in the whites. It is also mentioned that nephrosclerosis is probably the most common finding in renal pathologic condition found at autopsy. The incidence increases with age commonly associated with low grade essential hypertension. Diabetes mellitus is a predisposing factor.⁶

The gross size of the kidney depends on the duration of severity of hypertensive disease. Haemorrhage in the kidney may appear due to the rupture of arterioles, or glomerular capillaries.

The histopathological changes in malignant hypertension shows glomerulus increase in mesangial matrix which results in either segmental or global solidification (sclerosis) of the glomerular tuft. Tubules may be atrophic and sometimes contains hyaline casts. The epithelial cells are flattened and surrounded by thickened tubular basement membrane, which may be wrinkled. Interstitium is widened in areas with atrophic tubules. Increased collagen is noted. Chronic inflammatory cells usually small lymphocytes may be widely dispersed in areas of scarring.⁷

According to Sheldon C et al, arteriolar sclerosis, tubular degeneration was the most frequent parenchymal lesion observed and was the earliest recognizable renal abnormality accompanying hypertension. Tubular lesion accompanying pyelonephritis were distinguished by presence of ordinary inflammatory and reparative process.

Finely pitted cortical surface reflects the scarring of kidneys in individuals with hypertension. Hyalinized glomeruli were found more frequently in association with advanced arteriolar sclerosis.⁸

Acute post streptococcal glomerulonephritis is a common form of glomerulonephritis in developing countries like India. Though it mostly affects children between 2-14 years of age, 10% cases are seen in adults above 40 years of age. The kidneys are found to be symmetrically enlarged weighing 1.5 to twice the normal weight. Development of hypertension is a poor prognostic sign. In adults renal failure may result as a complication. According to Michie et al, pyelonephritis provokes a functional deficit which in turn induces compensatory hypertrophy in nondiseased kidney.⁹

Compensatory hypertrophy results from an increased workload due to some physical defect, such as, in an organ where one part is defective, or in one kidney when other is absent or nonfunctioning.¹⁰

The present case showed a unilateral hypertrophic right kidney in an adult male. The left kidney was atrophic. The surface of the kidney was irregularly scarred and the capsule was adherent to the scar. There is blunting and dilatation of calyces. Hypertension and chronic renal infection such as pyelonephritis led to scarring and atrophy of the left kidney which led to compensatory hypertrophy and hypertensive changes on the right kidney.¹¹

CONCLUSION

Vascular compromise secondary to malignant hypertension caused ischemic changes in the left kidney. This superimposed with chronic renal infection, ultimately resulted in an atrophic kidney. The right kidney became hypertrophied to compensate the work load of the left kidney. The histopathologic changes in the right kidney revealed that renal hypertension as well as infection had set in.

As regardless of cause, any renal disease ultimately leads to renal failure (End stage renal disease). This might have been the cause of death for this present case.

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REFERENCES

- 1) Healy J C. Kidney. In, Standring S (ed). Gray's Anatomy, 39th edition, Edinburgh, Elsevier Churchill Livingstone, 2005; 1269-1277
- 2) Dutta A K. The Urinary System . In Dutta A K(ed). Essentials of Human Anatomy, Part I, 8th edition, Kolkata, Current Books International, 2009; 289
- 3) Mohan H . The Kidney and lower Urinary Tract. In Mohan H (ed) Text book of Pathology , 6th edition, New Delhi ,Jaypee Brothers, 2010; 653-666
- 4) Laszik ZG, Silva F G. Hemolytic Uremic Syndrome, Thrombotic Thrombocytopenic Purpura and other Thrombotic Microangiopathies. In, Jennette C J(ed) . Heptinstall's Pathology of the Kidney, 6th edition Philadelphia, PA 19106 USA, Lippincott Williams & Wilkins, 2007; 732-733
- 5) Nadasdy T, Silva F G. Adult Renal Disease. In, Carter D (ed) Sternberg's Diagnostic Surgical Pathology, 4th edition, Philadelphia PA 19106 USA, Lippincott Willams & Wilkins, 2004; 1927
- 6) Alpers C E. The Kidney, In Kumar V(ed). Robbins and Cotran Pathologic Basis of Disease , 8th edition, New Delhi , Elsevier publication ,2012 ; 950-51
- 7) Olson Jean L. Renal disease caused by hypertension. In Jennet C J(ed) Heptinstall's Pathology of the Kidney,6th edition Vol 2, Philadelphia, PA 19106 USA, Lippincott Willams & Wilkins, 2007; 944-947
- 8) Sommers SC, Relman A S, Smithwick RH . Histologic studies of kidney biopsy specimens from patient of hypertension. Am J Pathol 1958; 34:685-715
- 9) Michie AJ, Michie CR, Ragni MC. Kidney function in unilatetral pyelonephritis II, Physiologic interpretation . The Am J of Med 1957; 2:22.
- 10) Dossetor R S. Renal compensatory hypertrophy in adults. Br J Radiol 1975; 48: 993-95
- 11) Bengtsson C, Hood B. The unilateral small kidney with special reference to hypoplastic kidney . Int J of uro and neph 1971; 3: 337-51