RENAL HISTOPATHOLOGY FOLLOWING RUSSELL'S VIPER (*VIPERA RUSSELLI*) BITE

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Abstract. Renal lesions in ten patients following Russell's viper bite were studied. Renal biopsies were available in six and autopsies in four patients. Autopsied tissues from two cases of traumatic death served as controls. Both qualitative and quantitative changes in the glomeruli, tubules, interstitium and blood vessels were evaluated. Tubular necrosis was detected in five, tubular degeneration in nine, glomerular changes in nine and interstitial changes in four cases. Generally tissues from expired cases had more severe and extensive renal lesions than those that survived.

INTRODUCTION

Russell's viper envenomation is one of the important causes of acute renal failure (Aung Khin, 1978; Chugh *et al,* 1975) in tropical countries. In most reports, renal failure was attributed to tubular necrosis and cortical necrosis (Date and Shastry, 1981, 1982). Case reports regarding other renal lesions like nephrotic syndrome, proliferative glomerulonephritis, acute interstitial nephritis and necrotising arteritis have been described by different authors from different countries (Steinbech, 1960; Sant and Purandare, 1972; Tembe and Sant, 1975; Seedat *et al,* 1974; Sitprija and Benjapati, 1974; Mittal *et al,* 1986). Reports from our country (Aung Khin, 1978; Than Than *et al,* 1989; Soe Soe *et al,* 1990) mentioned tubular necrosis in autopsied cases only. Thus it is necessary to study both autopsied and biopsied tissues following Russell's viper induced renal failure for other possible renal lesions.

MATERIALS AND METHODS

Ten patients who were admitted to Renal Dialysis Unit, Yangon General Hospital for renal failure following Russell's viper envenomation were studied. Four autopsied tissues and six biopsies were available. Renal biopsy was taken when the renal insufficiency persisted even after three rounds of peritoneal dialysis in acute cases and in chronic cases when there was unexplained renal dysfunction. Sources of tissues are summarized in Table 1.

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Renal tissues were fixed in 10% buffered formalin, embedded in paraffin, sections cut at 3 μm thickness and stained with Hematoxylin and Eosin, Periodic Acid Schiff, methanamine silver and fibrin stain using standard procedures.

RESULTS

Glomerular changes

Changes in the cells, capillary loop, basement membrane and Bowmen's space were noted and semiquantitatively graded into mild, moderate and severe.

A moderate degree of endothelial cell swelling was noted in three out of four autopsy tissues and two out of six biopsies showed only mild degree (Table 2). A mild increase in glomerular cells was observed in three and one cases of postmortem and biopsy tissues respectively. Apart from one biopsy all cases had a mild degree of glomerular necrosis (Fig 1). Fluid collections (Fig 2) and adhesions between glomerulus and Bowmen's capsule were visualized in all tissues, however they were more marked in expired cases. Ballooning of capillary loops with thickening of basement membrane (Fig 2) was seen in only three biopsies from patients who presented with proteinuria, nocturia and panhypopituitarism (Table 1).

Tubular changes

Cellular swelling, vacuolar or acidophilic degeneration, necrosis and regeneration of the lining

Table 1

Clinical course of ten snake-bite patients.

Sr no.	Age/sex	Type of tissue	Interval between bite and tissue obtained	Main clinical features
1	13 M	Autopsy	1 day	ARF
2	15 F	Autopsy	14 days	ARF
3	15 M	Autopsy	3 days	ARF
4	49 M	Autopsy	6 days	ARF
5	55 M	Biopsy	6 months	Hypertention
6	32 M	Biopsy	2 months	Hypopituitarism
7	20 M	Biopsy	2 months	Hypopituitarism
8	40 M	Biopsy	5 months	Dependent edema
9	17 M	Biopsy	8 months	Proteinuria
10	52 M	Biopsy	11 months	Nocturia

ARF = acute renal failure

Table 2

Glomerular changes of ten snake bite cases.

Sr no.	Endothelial swelling	GBM thickening	Capillary ballooning	Glomerular necrosis	Increased cells	Fluid or adhesion
1	(++)	(−)	(−)	(+)	(+)	(++)
2	(++)	(−)	(−)	(+)	(+)	(++)
3	(++)	(−)	(−)	(+)	(+)	(++)
4	(−)	(−)	(+)	(+)	(−)	(+)
5	(+)	(−)	(−)	(+)	(−)	(+)
6	(+)	(−)	(−)	(+)	(−)	(+)
7	(−)	(+)	(+)	(−)	(+)	(+)
8	glomeruli not included in the biopsy					
9	(−)	(+)	(++)	(+)	(−)	(+)
10	(−)	(+)	(++)	(+)	(−)	(+)

(−) no, (+) mild, (++) moderate
GBM = Glomerular basement membrane

tubular epithelium were studied and graded. The presence of casts inside tubules was also noted.

Apart from one biopsy, a mild to severe degree of tubular cell degeneration was noted in all tissues (Table 3). However, necrosis of tubular cells (Fig 3) was noted in all autopsies and one biopsy which presented with nocturia. A mild to moderate degree of tubular regeneration was observed in 50% and 83% of autopsies and biopsies, respectively. Apart from one case each in autopsy and biopsy tissues, all had casts in their tubules (Fig 3).

Interstitial changes

Changes in the interstitium such as cellular infiltration, fibrosis, fibrin deposition and vasculitis were scrutinized.

Inflammatory cells infiltration was observed in

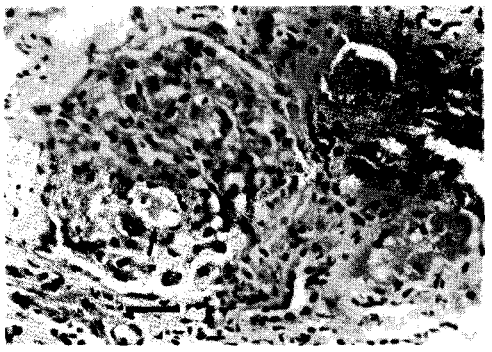

Fig 1—Micrograph of a glomerulus showing swelling and cellularity with necrosis (arrow) and adhesions partially obliterating the Bowmen's space. Bar = 50 μm.

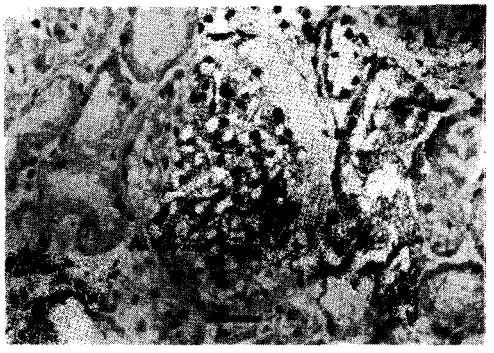

Fig 2—Micrograph of a glomerulus which was hypocellular and shrunken with ballooned-out capillary loops and collection of fluid inside Bowmen's space. Bar = 50 μm.

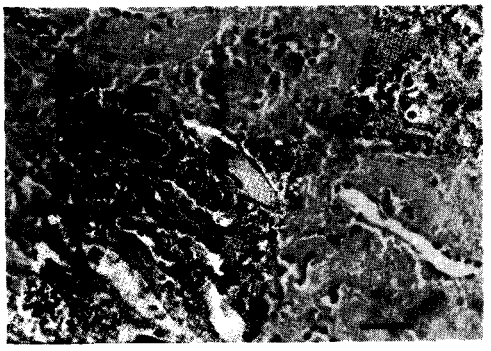

Fig 3—Micrograph showing necrotic tubules with casts. Bar = 50 μm.

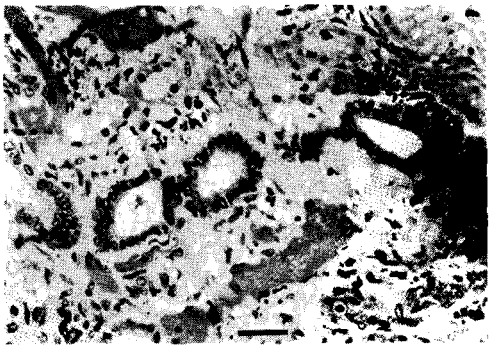

Fig 4—Regenerating tubules among the degenerated and fibrotic background. Bar = 50 μm.

two autopsies and two biopsies had interstitial fibrosis. Vasculitis was not seen. A mild to moderate degree of fibrin deposition in the interstitial vessels and in glomerular tuffs was noted in all autopsies, but none in biopsies.

DISCUSSION

Severe degrees of tubular degeneration and necrosis were seen in autopsies as compared to biopsies of patients who overcame acute renal failure. Also fibrin deposition was noted in all autopsies and none in biopsies. These findings were in accordance with our previous reports of autopsy cases (Than Than *et al*, 1989).

Fibrin deposition was demonstrated in autopsies only. This may be due to efficient fibrinolysis after snake bite since autopsied tissues were received within 14 days after the bites whereas biopsies varied from 2 to 11 months after the bites. Woodham *et al* (1989) also demonstrated increased fibrinolytic activity after Russell's viper bites. Regeneration of the tubules (Fig 4) was noted in 70% of the tissues and in one case who expired one day after snake bite. Thus regenerative capacity of the tubular cells was not defected after Russell's viper envenomation.

As depicted in Tables 2 and 3, in autopsied tissues the significant pathological changes in the glomeruli were endothelial cell swelling, increased cellularity, glomerular necrosis, fibrin deposition, collection of fluid and adhesions in Bowmen's space. Major tubular changes included degeneration

Table 3

Tubular and interstitial changes of ten snake bite cases.

Sr no.	Degeneration of the tubule	Necrosis of the tubule	Casts in the tubule	Regeneration of the tubule	Fibrosis cellular infiltration	Fibrin deposition
1	(+ + +)	(+ +)	(+)	(+)	(−)	(+ +)
2	(+ + +)	(+ +)	(+ +)	(−)	(+) C	(+)
3	(+ + +)	(+)	(−)	(−)	(+) C	(+)
4	(+ +)	(+)	(+ +)	(+)	(−)	(+)
5	(+ + +)	(+)	(+ +)	(+)	(−)	(−)
6	(+ +)	(−)	(−)	(+)	(−)	(−)
7	(+)	(−)	(+)	(+ +)	(+) F	(−)
8	(−)	(−)	(+)	(+ +)	(+) F	(−)
9	(+ +)	(−)	(+ +)	(+ +)	(−)	(−)
10	(+ + +)	(−)	(+ +)	(−)	(−)	(−)

(−) no, (+) mild, (+ +) moderate, (+ + +) severe
C = Cellular infiltration, F = Fibrosis

and necrosis. In biopsy cases ballooning of the capillary loops, thickening of basement membrane and necrosis of glomeruli were more prominent.

The study clearly shows different pathological patterns and severity between postmortem and biopsy cases. It also draws attention to the changes in the glomeruli. Mittal *et al*, (1986) also described glomerular changes in their large collections of viper bite cases.

From the above histological observation it can be concluded that the initial renal insult in viper envenomation is insufficient filtration due to swollen and cellular glomeruli with added fibrin deposition. Tubular reabsorption is also defective due to tubular degeneration and necrosis. However, in the later stages the important renal defects are dilated capillary loops, with thickened basement membranes, necrosis of glomeruli and tubular degeneration. Thus there will be increased filtration through the porous glomeruli compounded with inadequate reabsorption due to residual degenerative tubular epithelium. These findings are of importance in the management of the snake bite patient.

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