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ACUTE ANURIC GLOMERULONEPHRITIS[1]

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With Plate 11

The great majority of patients suffering from acute glomerulonephritis survive the acute attack (Volhard, 1931; Murphy and Peters, 1942; Ellis, 1942) but a few become virtually anuric and die in uraemia within a few weeks of the onset of the disease. The frequency of anuria in acute glomerulonephritis is fortunately low, Volhard (1931) mentioning eight cases from his clinic; Richter (1936), in Boston, found three fatal cases of anuria in 100 patients with acute glomerulonephritis; Ellis (1942) had three deaths from oliguric uraemia in 225 cases of Type I nephritis; Earle and Jennings (1961) had one case of anuria in 36 patients with acute glomerulonephritis. The management of these patients is discussed by Alwall, Erlanson, Tornberg, Fajers, and Moell (1958) who treated two patients with repeated dialyses; more recently Milne, Shackman, Struthers, and Loughridge (1960), and Harrison, Loughridge, Milne, and Shackman (1963), have described their experience with dialysis in treating similar patients with fatal oliguric acute nephritis. In this paper we record our experience in the management of eight patients suffering from acute anuric glomerulonephritis who were admitted to the Manchester Royal Infirmary from 1961 to 1963, discuss the role of haemodialysis, corticosteroids, and renal biopsy in the management of the disease, and describe the pathological appearances at renal biopsy and autopsy.

Clinical Features

The case histories are given in detail in the appendix, and the findings summarized in Table I. The ages of our patients ranged from 10 to 56 years; five of the patients were below the age of 35. Equal numbers of males and females were affected. A history of sore throat shortly before the onset of the disease was elicited in only three of the eight cases. Of five patients who had serum antistreptolysin O (ASO) titres estimated, three had titres suggesting recent streptococcal infection; two of them recalled a previous sore throat. The other two patients had ASO titres which were below 200 Todd units and did not

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rigorously exclude a recent streptococcal infection. Nausea, vomiting, and anorexia were prominent in the first week of the illness in six out of the eight patients. Peripheral oedema was found in six patients. The early onset of oliguria was noticed in all patients, urine volumes rapidly decreasing to below 250 ml. per day in all patients before death. Immediately after dialysis there was absolute anuria, and in four patients this persisted until the death of the patient. (The term 'anuria' has been used in this paper in preference to the cumbersome, less euphonic, but more accurate term 'oligoanuria' to indicate daily urine volumes of less than 250 ml.)

TABLE I

Clinical Findings

No.	Age	Sex	Sore throat	Nausea and vomiting	Oedema	Duration of illness (days)	Max. B.P.	A.S.O. titre	Steroids	Blood urea mg./100 ml. Initial	Blood urea mg./100 ml. Daily rise	No. of dialyses	Daily urinary vol. (ml.)	Urine cultures
1	10	F	0	0	+	56	160/100	200	+	40	25	2	0 to 150	..
2	55	F	0	+	0	42	115/60	Not done	0	525	10·1	1	0	..
3	24	M	0	0	+	40	140/90	200	+	185	33·0	3	0	..
4	34	M	+	+	+	16	120/75	300	+	185	45·0	2	0 to 200	..
5	10	F	0	+	+	63	100/80	Not done	+	515	34·3	1	135	Proteus and coliform
6	56	M	+	+	+	31	165/80	Not done	+	325	22·9	2	200	..
7	17	F	0	+	0	52	140/80	200	+	700	34·3	1	200	..
8	40	M	+	+	+	15	195/105	250	+	500	25·0	1	200	Proteus

The diastolic blood-pressure exceeded 90 in only two patients, in whom it was 100 and 105 mm. of mercury. On admission to the Manchester Royal Infirmary all the patients were uraemic; in four the blood urea was 500 mg./100 ml. or higher. These patients were dialysed immediately in order to gain time so that renal biopsy could be carried out and the cause of the anuria determined. In seven of the eight patients corticosteroid therapy in the form of prednisone or hydrocortisone was administered in total daily doses of up to 300 mg. of prednisone or its equivalent in hydrocortisone. The effect of corticosteroid administration on the pathological picture was determined by comparison of the biopsy and subsequent autopsy in six patients, and is referred to in the section on pathology below.

The anuric patients were maintained on a modified Bull regimen, receiving 500 ml. of 50 per cent. dextrose or 20 per cent. lactose with the addition of appropriate amounts of saline to replace vomitus or diarrhoea; resins in the form of Resonium A were administered orally to hyperkalaemic patients, resulting in hypokalaemia in patient 2 which persisted after the resin was stopped, and ultimately contributed to the patient's death. Anabolic steroids in the form of nandralone or norethandrolone were administered to all patients. Urinary

infection was present in two patients in whom the urine was cultured. No patients were subjected to the added hazard of an indwelling catheter.

Haemodialysis was carried out in all patients, using a Kolff disposable twin coil artificial kidney for about six hours' duration on each occasion. Dialysis was performed three times on one patient, twice on three patients, and once on the remaining patients. In all cases haemodialysis was extremely efficient in

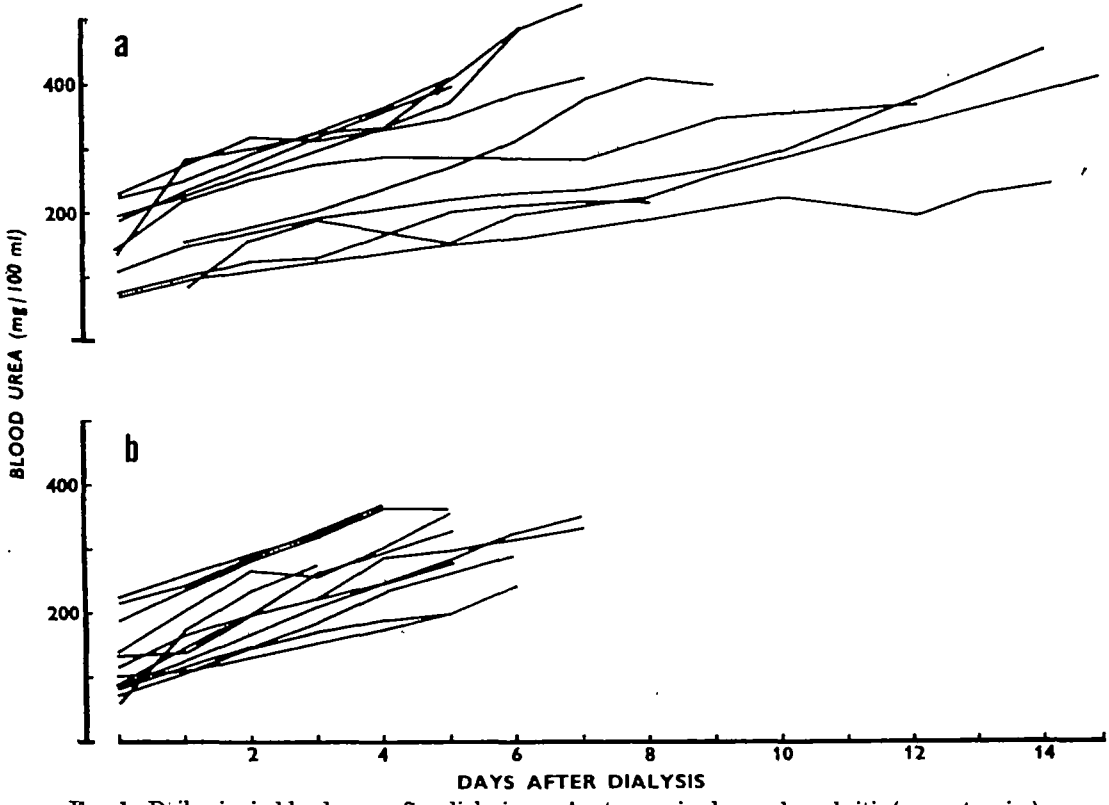

FIG. 1. Daily rise in blood urea after dialysis. *a*. Acute anuric glomerulonephritis (present series). *b*. Acute tubular necrosis (Roscoe, 1963).

partially restoring biochemical normality; usually clinical improvement was striking, leading to a remarkable conversion from the typical picture of advanced uraemia with its confusion, nausea, vomiting, and hiccough to a comfortable lucid state. In one case, however, dialysis apparently precipitated fatal left ventricular failure, although an earlier dialysis had been well tolerated.

Blood Urea Rise after Dialysis

The mean daily rise in blood urea after dialysis in this series of patients was 28·2 mg./100 ml. (range 10–45 mg.). This is shown in Fig. 1*a*; it can be compared to the results in a series of seven patients with acute tubular necrosis (Roscoe, 1963) who had a mean daily rise of blood urea of 28·4 mg./100 ml. after dialysis (Fig. 1*b*). The rise in blood urea is variable and unpredictable during the first 48 hours after dialysis during re-equilibration of intracellular and extracellular urea pools. After this period the rise was less variable but was

not absolutely constant for any patients, depending on fever, infection, blood transfusion, &c. There is no significant difference between the daily blood urea rise in acute tubular necrosis and acute anuric glomerulonephritis ($t = 0{\cdot}2$, $p = 0{\cdot}85$).

Prognosis

The disease was fatal in all eight patients, although the complications of treatment were the immediate cause of death in three of them; two died of possible side effects of steroids (infection and gastro intestinal bleeding) and one died from left ventricular failure coming on during haemodialysis. The duration of the disease from the first symptoms to death varied from 15 to 63 days with a mean of 29 days. There is no doubt that survival was prolonged by dialysis.

TABLE II

Histological Findings on Biopsy and Autopsy

Case no.		Days after onset	Destruction of glomeruli	Polymorphs within the glomerulus	Breaks in capsular B.M.	Necrosis	Glomerular fibrosis	Tubular damage	Red cell casts	Interstitial infiltrate	Oedema
1	Biopsy	24	7/7	+	++	−	++	+−	+	+	+
	No autopsy										
2	Biopsy	32	7/7	−	++	−	+	+−	+	+(f)*	−
	Autopsy	42	100%	−	++	−	++	−	+	+−	−
3	Biopsy	22	14/15	+++	++	++	−	+	+	+(f)	+
	Autopsy	40	100%	−	+++	−	+++	+	+	+−(f)	++
4	No biopsy										
	Autopsy	16	96%	−	+	−	+	+	+++	+(f)	++
5	Biopsy	48	27/29	+	++	−	++	++	++	++(f)	+
	Autopsy	63	98%	−	+++	−	+++	++	−	+	+
6	Biopsy	21	41/43	+	+	+	+−	+	−	++	+
	Autopsy	31	100%		++	−	+++	−	+	+	−
7	Biopsy	42	20/20	+	+++	−	++	+	++	++(f)	+
	Autopsy	52	100%	−	+++	+	+++	+	++	+++	+
8	Biopsy	10	20/22	+	+	+	+	−	+	+−	+
	Autopsy	16	100%	−	+	+	+	+	+	+−	++

* (f) indicates a focal distribution.

Pathological Findings

Histological findings at biopsy and autopsy are summarized in Table II. Percutaneous renal biopsy was performed in seven patients between eight and 30 days before death and was not associated with significant morbidity. The striking histological finding is the massive overgrowth of the glomerulus by large epithelial cells. In some glomeruli this takes the form of the well-known crescent, but in most the whole circumference of the capsule is involved (Plate 11, Fig. 2) with complete obliteration of Bowman's space. The cellular proliferation involves primarily the parietal epithelium which, judging from the mitotic figures, proliferates rapidly, compressing the vascular tuft and occasionally growing down the tubular lumen (Plate 11, Fig. 3).

In most glomeruli there is some necrosis of parietal epithelial cells with karyorrhexis of nuclei (Plate 11, Fig. 3). When the necrosis is extensive there is an infiltration with polymorphonuclear leucocytes but it is important to stress that the infiltration is not an invariable accompaniment of the cellular proliferation and disappears with the subsequent fibrosis. As the disease progresses the large epithelial cells filling the urinary space shrink; the nuclei become denser and

spindle-shaped. Extra-cellular material indistinguishable from collagen on light microscopy appears between the cells until the whole of the glomerulus forms a hyaline knot in which the vascular tuft is represented by a compressed, weakly PAS positive, fibrillary mass containing a few nuclei (Plate 11, Fig. 4). In most cases concentrations of polymorphs appear to be localized along the site of the capsular or tubular basement membranes (Plate 11, Fig. 5); these basement membranes apparently disappear under these conditions—a phenomenon which is confirmed by finding definite gaps in the membrane when the inflammation has subsided (Plate 11, Fig. 4). The tuft shows compression of the capillaries with wrinkling of the basement membranes. In a few tufts one sees hyaline areas some of which are due to fibrinoid necrosis but others on careful examination seem to be collections of visceral epithelial cells containing condensed hyaline droplets.

The tubules show foci of necrosis with occasional regeneration, thinning of the epithelium, and dilatation of the lumen. Many tubules can be found packed with red cells. Some epithelial cells contain abundant hyaline droplets suggesting that there is some persistent glomerular filtration of protein. The tubules are separated by oedema of the interstitial tissue which is infiltrated by inflammatory cells, usually plasma cells and lymphocytes with a few eosinophils. There appears to be no definite localization of inflammation to any particular level in the cortex or to any particular segment of the tubules but the medulla is not involved. The vessels appear normal.

The necropsy material (seven patients) shows progression of the lesions to complete fibrosis of most of the glomeruli. The interstitial inflammatory cell infiltration appears to have decreased in all cases whether treated with corticosteroids or not. Careful search was made through numerous sections of other organs for lesions, particularly of the vessels, which might indicate a polyarteritis nodosa of the group described by Davson, Ball, and Platt (1948); no lesions of this type were found.

At autopsy in Case 3 the lungs were brown in colour; histological examination showed bronchopneumonia with fungal overgrowth; there were large numbers of iron-laden macrophages but no evidence of vasculitis.

Discussion

The immediate prognosis in acute glomerulonephritis is good (Volhard, 1931) although the long-term prognosis is more dubious and the subject of conflicting reports which are well summarized by Murphy and Peters (1942); the death-rate in the acute phase of acute glomerulonephritis varies from 13 per cent. in Murphy and Peter's series to 4 per cent. in that published by Ellis in the same year; Longcope (1936) had four deaths in 36 patients; Earle and Jennings (1961) had one death in their 36 patients but their general opinion was that the mortality rate was about 5 per cent. in the acute attack. Wilson (1962) has pointed out that hospital statistics for the complications of acute nephritis are misleading in that mainly the severely ill patients find their way to hospital, so

presumably the disease is more benign than the literature on the subject would indicate. The deaths in the acute phase are due either to uraemia with severe oliguria or to the complications of hypertension, such as left ventricular failure or hypertensive encephalopathy, or to a combination of the two (Volhard, 1931; Ellis, 1942). In our patients uraemia was the major cause of death, all being severely oliguric or totally anuric. An early complete clinical and pathological description of this type of fatal acute anuric nephritis is that of Koch (1930) working in Volhard's clinic who described seven patients, the first three of whom are strictly comparable on both clinical and pathological grounds to the series described in this paper. Fahr (1925) in describing primarily the pathological changes in '*subacute nephritis*' included a rapidly fatal case (No. 23) of severe oliguria in acute nephritis with a similar pathological picture. With the advent of haemodialysis, interest in this disease was rekindled, Alwall and his colleagues (1958) reporting two cases treated for several weeks by this means with an ultimately fatal outcome. The literature does not make an absolutely clear distinction between the maximum duration of acute glomerulonephritis and the minimum duration of rapidly progressive Type I nephritis ('subacute' glomerulonephritis according to Volhard's classification), but it seems reasonable to accept three months as an arbitrary dividing line, our patients differing from rapidly progressive Type I nephritis not only in the duration of illness, but also in the absence of hypertension and of hypoproteinaemia. In our series the minimum length of duration of the disease from health to death was 16 days, the maximum being 63 days. In all cases death was certainly delayed by therapy such as haemodialysis, resins, modified Bull regimen, and anabolic steroids; the possibility that corticosteroids also helped was not proven; on the contrary there was evidence that in two of our patients death was hastened by the complications of steroid treatment. Comparison of renal histology at the time of biopsy and again at autopsy showed no consistent changes which could be attributed to steroids. However, the dose of steroids given was variable, the time of commencement was likewise variable from case to case, and so strictly no conclusion can be drawn with certainty from our results as to the efficacy or otherwise of corticosteroids in this disease. Had no therapy of any sort been given it is probable that all our patients would have died within seven weeks of the onset of symptoms, and the majority within five weeks. We have preferred to call the disease *acute* glomerulonephritis therefore, rather than *subacute* glomerulonephritis.

There was convincing evidence of preceding streptococcal infection in only three of our patients, although the number may really be higher. Longcope (1936) showed that some post-streptococcal acute glomerulonephritic patients had a delayed rise in ASO titre, and as our patients did not have serial determinations of the ASO titre, a simple negative result could not be interpreted as excluding streptococcal infection. Moreover, the effect of large doses of steroids may have been to reduce the antibody response. The history of a sore throat is, by itself, no criterion of a streptococcal throat infection; Bates, Jennings, and Earle (1957) have described a series of 10 patients suffering from non-streptococcal

glomerulonephritis in whom a sore throat was a prominent feature. They ascribed this to a viral infection. Our cases did not occur in an epidemic, unlike those of Milne and his group (1960), and to our knowledge there was no epidemic of streptococcal infection in the north of England during the years 1960 to 1963 when our case material was collected.

The pathology of typical acute post-streptococcal glomerulonephritis has been described by Jennings and Earle (1961) and by Lawrence, Pollak, Pirani, and Kark (1963) and consists of a lesion confined to the glomerular tufts. The pathogenesis of the disease is not understood but the most widely held belief is that the lesion is due to an antigen-antibody reaction taking place in or near the basement membrane of the capillary loops, with consequent damage. In contrast to this 'typical' lesion our cases appear to show no primary affection of the tuft; even in the most florid cases it is possible to find glomerular tufts containing no polymorphs. The lesions seem rather to be primarily involving Bowman's capsule (capsulitis) with concentration of inflammatory cells in this region and obvious damage to the capsular basement membrane. Fahr (1925) has drawn attention to this type of lesion by naming it 'extracapillary glomerulonephritis'. It seems likely that at least three of our cases were secondary to streptococcal infection and one case ('Cal') described by Jennings and Earle (1961), showing a similar histological picture, has well-documented evidence of a similar infection. Streptococcal infection has also been incriminated as an aetiological agent in acute interstitial nephritis, a disease which was common in the nineteenth century and associated with scarlet fever (Councilman, 1898). In their series of post-streptococcal nephritides Earle and Jennings (1961) have two cases of acute interstitial nephritis. In acute interstitial nephritis complicating drug sensitivity, it appears that the tubular basement membrane is damaged (Baker and Williams, 1963) while the glomeruli escape. Thus it seems that nephritis due to hypersensitivity can affect the basement membrane of any part of the nephron—the vascular tuft, the capsular membrane, or the tubular membrane. It is not surprising to find commonly a combination of the lesions. In the cases described here every one showed some evidence of tubular damage. The combination of a tuft lesion and capsulitis is, of course, common in the form of a rapidly progressive Type I nephritis (Ellis). Thus hypersensitivity nephritis presents a spectrum, the cases we have described here being only one carefully selected facet of the disease. There is no evidence as to what determines which part of the nephron is mainly affected, but this is important as it determines the outcome of the disease. Capsulitis seems to be the most lethal, being capable of killing the patient, in its severest form, in a matter of weeks. Attempts at repair of this lesion result in fibrosis of the entire glomerulus. The pure capillary lesion on the other hand heals, leaving at the most isolated scars in the tuft but usually good renal function. The tubular lesions leave behind some interstitial fibrosis, but even in fairly severe cases adequate function (Earle and Jennings, 1961; Baker and Williams, 1963). In our series of patients there was no evidence of vascular disease, neither an arteritis nor malignant hypertensive changes (Koch, 1930).

The rise in blood urea after dialysis was similar in our experience in acute tubular necrosis and acute anuric glomerulonephritis, although Milne and his colleagues (1960) found that in the latter disease there was a significantly lower daily blood urea rise than in acute tabular necrosis. This difference between their results and our own is real and not accounted for by different statistical methods; it may well be due to their series having a higher proportion of cases in advanced pregnancy at the time of the onset of anuria, whereas the patients in our series were all in fairly early pregnancy with a smaller uterus to involute. The presence of infection, blood transfusion, and corticosteroid administration would influence the blood urea rise and might also contribute to the discrepancy between the two results. In the initial 48 hours after dialysis the blood urea rise was variable, presumably due to the variation in re-establishing an equilibrium between extra- and intra-cellular urea pools.

Renal biopsy has been of great value in determining the nature of the kidney lesion in all our patients with anuria, and consequently in determining the prognosis. We have rarely had a death from uncomplicated acute tubular necrosis and believe that repeated dialysis, if need be for many weeks, is indicated in this disorder; on the other hand, in acute glomerulonephritis a fatal outcome is at present inevitable and repeated dialysis is not justified. Possibly corticosteroids might be useful if they could be given before the capsular epithelial proliferation had become considerable, but this would necessitate treating all cases of acute nephritis with high corticosteroid dosage as early as possible in the disease; the considerable mortality from the side effects of large doses of corticosteroids does not justify this at present in what is generally a fairly benign disease. Once capsular epithelial proliferation has reached a certain stage, healing by fibrosis results in irreversible damage to the glomerulus; functionally an aglomerular kidney is produced and further treatment by dialysis is futile.

We wish to thank the various consultants who referred these cases to the Manchester Royal Infirmary; Professor D. A. K. Black and Professor A. C. P. Campbell for their help, and Mr. Thomas Moore and his colleagues who carried out the haemodialyses.

APPENDIX

Case Reports

Case 1. H. M. Female. 10 years old. In the last week in March 1962 she developed a puffy face and oedema of the lips; she was admitted to Wythenshawe Hospital under the care of Dr. Sylvia Guthrie. At that time she had heavy albuminuria and a blood urea of 40 mg./100 ml. Over the next few days she became severely oliguric and uraemic, and was transferred to the care of Professor D. A. K. Black at Manchester Royal Infirmary on 11.4.62. Her blood-pressure was then 175/95–100; she had oedema of face, arms, and legs. Her blood urea was 430 mg./100 ml., serum potassium was 7·5 m-equiv./l. A percutaneous left renal biopsy was performed on 19.4.62. She had a series of fits and tetany following this, her blood-pressure being 160/100. She responded

to paraldehyde i.m. and oral phenytoin. She was dialysed twice, her urine volume remaining below 150 ml./24 hours. She received prednisone 40 mg./day for two and a half weeks following which the dose was reduced to 30 mg. and then 20 mg., ceasing on 11.5.62. She died on the 56th day of her illness.

Case 2. N. B. Female. 55 years old. At the beginning of July 1962 she lost her appetite, had abdominal pain, nausea, and dark urine, and was thought to be jaundiced. After two weeks these symptoms subsided but she developed vomiting and recurrent diarrhoea. The diarrhoea settled but she continued to vomit, becoming anuric four days before being admitted to Park Hospital, Davyhulme. She was transferred to the care of Professor D. A. K. Black at Manchester Royal Infirmary on 28.7.62, her blood urea being 525 mg./100 ml. at this time. She was dialysed immediately and on 2.8.62 a right percutaneous renal biopsy was performed. She developed hypokalaemia (serum potassium 2·2 m-equiv./l.) on 11.8.62 which continued in spite of stopping resin administration; she died three days later. She received no corticosteroids during her illness.

Case 3. J. D. Male. 24 years old. He developed intermittent haemoptysis in April 1962 which continued until June 1962, when he developed bilateral mid-zone opacities on chest X-ray. He developed anaemia, haemoglobin 58 per cent. in July, and was discharged from Monsall Hospital on 29.8.62. His urine was albumen-free at this stage. Three weeks later he developed haematuria for seven days, followed by increasingly severe oliguria for three days. His blood urea was 185 mg./100 ml. and he was transferred to Manchester Royal Infirmary under the care of Professor D. A. K. Black on 8.10.62. The clinical diagnosis of Goodpasture's syndrome was made. His blood-pressure was 140/90 and he had frequent haemoptyses. He was anuric, with a blood urea of 315 mg./100 ml. He was dialysed three times and given prednisone 200 mg./day. The haemoptysis ceased dramatically, but he died of bronchopneumonia on 30.10.62.

Case 4. A. S. Male. 34 years old. Four days' history of bloody urine followed by severe dyspnoea and pyrexia. He was admitted to Manchester Royal Infirmary under the care of Professor Sir Robert Platt on 3.4.63; he was found to have 'uraemic lung' with a bilateral bat's wing appearance on chest X-ray and a blood urea of 185 mg./100 ml. on admission. His urine output gradually fell over the next week, his blood urea rising to 480 mg./100 ml. on 9.4.63. He was dialysed and the pulmonary oedema cleared dramatically, but during the second dialysis he developed some melaena. He died of gastrointestinal bleeding from a duodenal ulcer on 16.4.63, having received both hydrocortisone and latterly prednisone in a dosage of 100 mg./day.

Case 5. E. T. Female. 10 years old. She presented at Park Hospital, Davyhulme, on 27.5.61 with one week's history of blood-stained urine. An I.V.P. was found to show no excretion and one month later she started to vomit and commenced to bleed per vagina. She was found to have a purpuric rash over her arms, stomatitis, and an excoriated natal cleft. Her blood-pressure was 100/80 and blood urea was 515 mg./100 ml. Her haemoglobin was 39 per cent. and she passed very bloody urine varying in volume from 170 ml. to 45 ml./24 hours. She was transferred to the care of Mr. T. Moore on 28.6.61 at Manchester Royal Infirmary and was dialysed the same day. Percutaneous renal biopsy was performed on 7.7.61. She remained very severely oliguric, dying on 22.7.61. She received prednisone in a dosage of 200 mg./day from 6.7.61 until her death.

Case 6. D. G. Male. 56 years old. At the end of July 1961 he developed a sore throat. This was treated with penicillin. Two weeks later he had nausea and

vomiting, passed some red-brown urine, and became severely oliguric, passing up to 150 ml./day. He was admitted to Crumpsall Hospital under the care of Dr. S. Oleesky, and was transferred to the care of Professor D. A. K. Black on 15.8.61 at Manchester Royal Infirmary. His blood-pressure was 165/70 and he had extensive sacral oedema; he was dialysed on admission because of a serum potassium level of 9·5 m-equiv./l., and had a percutaneous renal biopsy on 21.8.61. He was treated with prednisone in a dose of 300 mg./day, but passed less than 200 ml. of urine a day, dying in left ventricular failure which developed while he was being dialysed on 31.8.61.

Case 7. P. L. Female. 17 years old. At the beginning of March 1962 she developed backache and after one week, nausea and vomiting and anorexia followed by oliguria; she was found to have red blood cells and pus cells in her urine and was given a sulphonamide, developing severe oliguria and an erythematous rash. She was admitted under the care of Dr. Ward at Victoria Hospital, Accrington, and was transferred for haemodialysis to the care of Professor D. A. K. Black at Manchester Royal Infirmary on 9.4.62. On admission she was found to have a blood urea of 700 mg./100 ml. Her blood-pressure was 140/80. She was immediately dialysed and a percutaneous renal biopsy was carried out on 12.4.62. She was maintained on a dosage of prednisone of 20 mg./day initially and was prepared for haemodialysis on 18.4.63, when she began to bleed profusely per vagina and per rectum and died on 22.4.63.

Case 8. W. D'A. Male. 40 years old. Cotton worker. Six months before death he had suffered a myocardial infarction and was on long-term anticoagulant therapy with phenylindanedione. He was readmitted to Bury General Hospital on 16.12.62 with pleuritic pain, and discharged after two weeks. Following this he felt ill, had a sore throat, and developed general malaise, chest pain, and vomiting. On 19.1.63 he developed haematuria with haematemesis and melaena. He was transferred on 24.1.63 from the care of Dr. Davies to that of Professor D. A. K. Black at Manchester Royal Infirmary, with a blood urea of 500 mg./100 ml., a blood-pressure of 190/105 mm. of Hg. He was dialysed the following day, passing subsequently 0 to 150 ml. of blood-stained urine each day, some of the urine clotted spontaneously. Percutaneous renal biopsy was carried out on 28.1.63. He was given prednisone in a dosage of up to 180 mg./day; further dialysis was thought to be unjustifiable in view of the histological picture of the renal biopsy, and he died on 3.2.63.

Summary

The clinical and pathological features of eight cases of acute glomerulonephritis with anuria are presented. All the patients were treated by haemodialysis and seven of them with corticosteroids. The outcome was uniformly fatal. Histological material was obtained from all patients; in seven by percutaneous renal biopsy and in seven at autopsy. In six the biopsy and autopsy appearances were compared. The main histological finding was obliteration of almost all the glomeruli by proliferating capsular epithelial cells progressing to total and irreversible fibrosis of the glomeruli.

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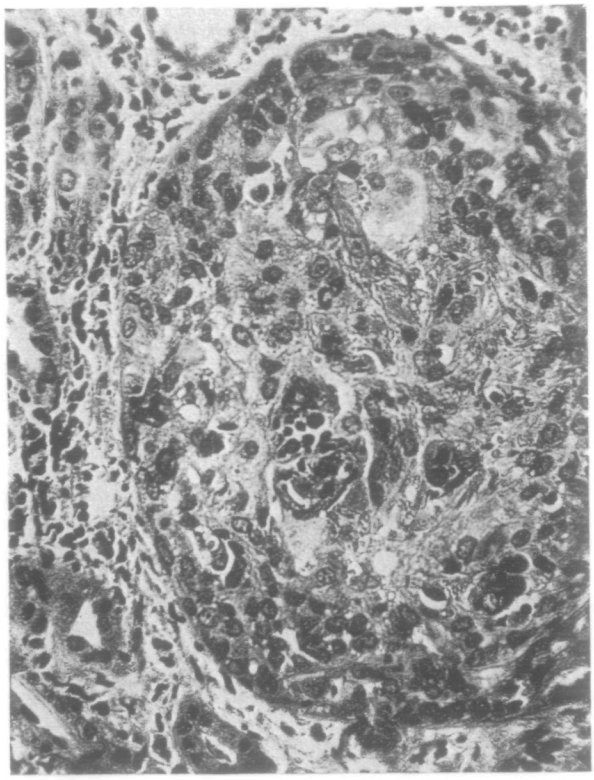

FIG. 2. Glomerulus showing proliferation of the capsular epithelial cells. There are very few inflammatory cells within the glomerulus. Case 7. (Haematoxylin and eosin × 430.)

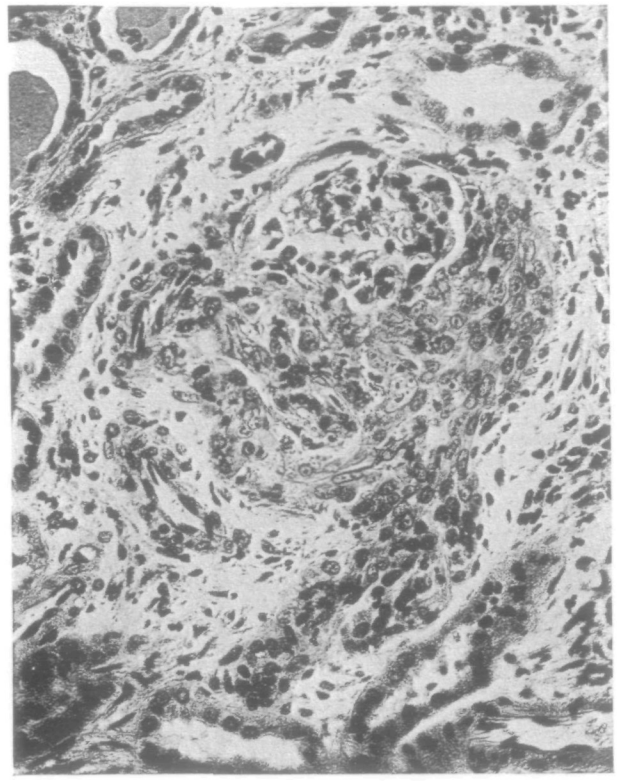

FIG. 3. Glomerulus showing epithelial proliferation compressing the tuft to one side and growing down the tubule. There is a marked periglomerular oedema. Case 8. (Haematoxylin and eosin × 280.)

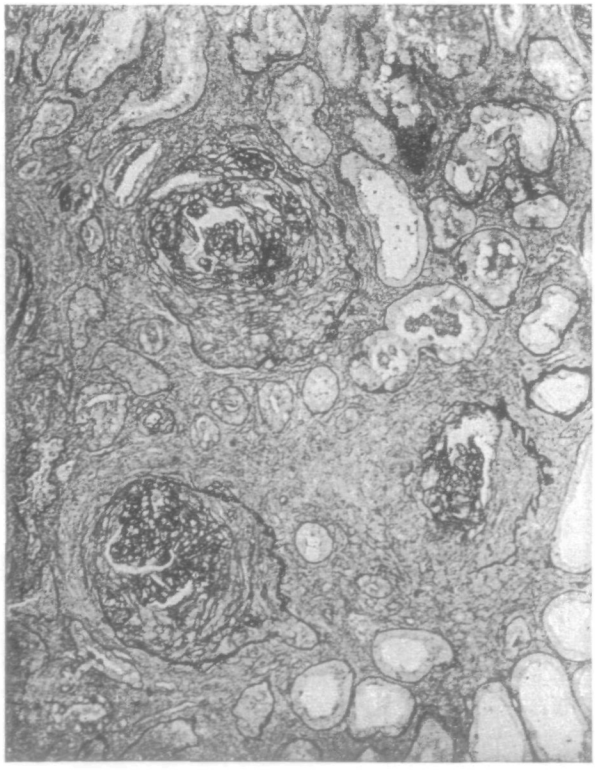

FIG. 4. The glomerular capsular basement membranes contain large gaps. The remnants of the vascular tuft can be seen as a dark knot. Case 2. (Periodic acid Schiff × 150.)

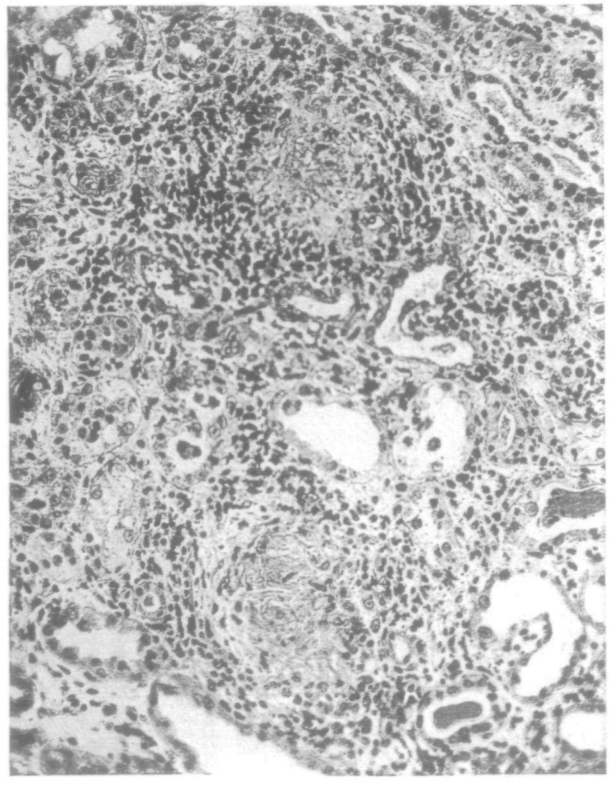

FIG. 5. Two glomeruli showing a marked pericapsular inflammatory cell infiltration. The tubules are dilated and the epithelium thinned. Case 6. (Haematoxylin and eosin × 220.)

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