DEXTRAN NEPHROSION
Renal Changes in 3 Patients with Acute Tubular Necrosis given Low Molecular Weight Dextran (LMWB) during the Oliguric Phase

R. WILKINSON, T. J. MUCKLE and D. N. S. KERR
Royal Victoria Infirmary, Newcastle upon Tyne, Great Britain

Low molecular weight dextran (LMWD) infusion has been shown to reduce the incidence of renal damage following incompatible blood transfusion in animals (Atik et al., 1961) presumably by its antisludging effect. It also produces a diuresis in normal subjects, the mechanism of which is thought to be the inhibition of A. D. H. by increased plasma volume (Matheson et al., 1964). Its use in acute renal failure in man has been advocated (Bergentz et al., 1961), because of these antisludging and diuretic properties, although it has been shown not to protect the animal kidney exposed to prolonged ischaemia, and its diuretic effect is unlikely to work in the post-operative or post-traumatic state when A. D. H. secretion is high.

Whereas there is no convincing evidence for the value of LMWD therapy in acute renal failure, there is evidence that it can threaten renal function in 2 ways:

1. In dehydration states, LMWD which exerts a small osmotic pressure because of its large molecular weight, (mean 40,000), is concentrated in the tubules producing a very viscid fluid which causes tubular obstruction and oliguria (Bergentz et al., 1965)

2. Swedish American dextran (mean m. wt 75,000) has been shown to be deposited in the epithelium of the proximal convoluted tubule and the loop of Henle in normal animals (Friberg et al., 1951), and this deposition is increased when there is renal damage (Mowry et al., 1951). Similar changes have been described in man in war casualties (Vicery et al., 1956) and deaths from burns (Johnston et al., 1953) treated with Swedish-American dextran.

Case summaries

During 1964 three patients with acute renal failure received LMWD therapy, for another condition, during the oliguric phase. All three were found at autopsy to have large pale kidneys with swelling and vacuolation of the cells of the proximal tubule and loop of Henle as shown in Fig. 1 and Fig. 2.

The treatment and course of two of the cases are summarised in the Figs. 3 and 4. The third case was a woman aged 45 who developed septicaemia with hypotension 6 days after combined excision of rectum for carcinoma, and LMWD was given in resuscitation. She was oliguric from the time of her hypotensive episode until her death 5 days later. She had been dehydrated when first given LMWD.

Discussion

We have at present only circumstantial evidence that the histological changes observed are due to LMWD.
1. Of eight autopsies performed on patients dying from acute renal failure in 1964 in the R. V. I. Newcastle only three showed the changes described above, and these were the only ones who had received dextran.

Fig. 1. L.M.W.D. Nephrosis. Renal cortex $\times$ 90 showing vacuolation and granular enlargement of proximal tubular epithelial cells. (Reduced for reproduction 25%).

Fig. 2. L.M.W.D. Nephrosis. Higher power of same ($\times$ 350) to show detail of cellular changes. (Reduced 25%).
2. In 1959–60 when LMWD was not in use, review of renal histology in patients dying of acute renal failure did not reveal any case with comparable histology.

3. The histological lesions resemble those seen in animals given Swedish-American dextran (Friberg et al., 1952) and a similar ‘hydropic degeneration’ in the kidneys of dogs given LMWD before or after a period of renal artery occlusion has been described (Hendren et al., 1964).

We are at present trying to obtain more direct evidence that the histological changes are due to dextran by the following methods:

(a) By specific staining for dextran with a modified P.A.S. stain on a frozen section (dextran being eluted in the usual fixation processes).

(b) By the estimation of the dextran content of an affected kidney taken at autopsy.

Conclusions

We cannot say whether LMWD accelerated the onset or prolonged the course of acute renal failure in these three cases, as in all three there was an adequate alternative explanation for renal failure; however we think that the changes observed, even if potentially reversible, are likely to reduce renal blood flow by increasing tension within the renal capsule, and that these changes are almost certainly due to LMWD.

---

Fig. 3. Acute renal failure following crush injury of right leg. Case 1. P.H.
DEXTRAN NEPHTROIS

Fig. 4. Acute renal failure following lumbar sympathectomy for peripheral vascular disease. Case 2
I.M.T.W.

REFERENCES

122, 343.