THE NEPHRON IN NEPHRITIS.¹

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The classification of nephritis, whether on a histological or clinical basis, leads to complexities which tend to baffle the student. The following attempt to unify the disease by considering it in terms of the function of the individual nephron and its blood supply, explains many of the clinical manifestations. It is realized that this presents a very simplified account of a most intricate disease and that much is based on hypothesis that is difficult of proof, but it has been found helpful as an introduction to the disease.

It is now believed that acute nephritis is not primarily a disease of the kidneys, but that it is a general disease of the capillaries throughout the body. This is suggested by the high protein content and distribution of the edema, by the occasional occurrence of hypertension and even edema before the appearance of albuminuria, and by direct observation of capillaries in the nail bed. Nevertheless the brunt of the attack is borne by the renal vascular system.

The main renal lesion in acute nephritis lies in the afferent arteriole and capillaries of the glomerulus, leading first to increased capillary permeability, with the appearance of albumen and red cells in the glomerular fluid, and later to the gradual obliteration of the vessels. The majority of cases of acute nephritis proceed to "clinical cure"—that is to say the edema disappears, the hypertension and blood urea return to normal and the urine ceases to contain albumen and red cells. There are two possible explanations of this cure, the glomerular capillaries may have recovered, so that the nephrons are again functioning normally, or the damaged units may have ceased to function altogether, so that the apparent cure represents the survival of the healthy nephrons. If the glomerular capillaries become impermeable, blood flow to that glomerulus will cease, no filtration can occur and no contribution from the damaged nephron will appear in the pelvic urine. If the blood supply to the damaged glomerulus is not completely occluded, albumen and red cells will persist in the urine.

The blood supply to the tubules comes mainly through the efferent arteriole from the glomerulus, the plasma, denuded of its solutes by filtration in the glomerulus, reconstituting itself by selective reabsorption from the tubules. A diminished blood supply to parenchymatous tissue often results in its fatty

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degeneration. The damaged, but still patent, glomerular circulation must result in a diminished blood supply to the tubules, so that fatty degeneration of the tubular cells might be expected to follow the glomerular lesions of acute nephritis, and such is, of course, often the case.

The second stage of nephritis, parenchymatous, hydæmic or nephrotic nephritis, is characterized histologically by fatty degeneration of the tubular cells. Degeneration of these cells, be it fatty as in nephritis or the rare primary nephrosis, or syphilitic or due to deposition of amyloid, leads to a characteristic syndrome with massive albuminuria, lower blood proteins with œdema and a marked rise in blood cholesterol. The source of the albumen in these conditions is undecided, but the massive loss of protein is a characteristic of tubular degeneration.

Not all cases of acute nephritis proceed to this nephrotic stage; many pass insidiously to the stage of chronic nephritis without ever having excessive albuminuria and low protein œdema. If the blood supply to the nephron is completely occluded, no urine is formed, the tubular blood supply is greatly diminished and rapid tubular degeneration must occur, but, again, the results of this degeneration cannot be reflected in the pelvic urine.

It is suggested, therefore, that the occurrence of the nephrotic stage in a case of acute nephritis is evidence of the continued survival of the damaged nephrons. If this is so, one might expect that eventually the blood supply to these damaged glomeruli would fail, with resultant complete degeneration of the nephron and cessation of albuminuria. This fits clinical experience for some cases of nephrotic nephritis do improve with apparent though usually temporary cure, while in others the nephrotic syndrome subsides as the patient passes on to the third stage of nephritis.

With a large number of nephrons destroyed, the future of the patient depends upon two factors, the function of the surviving units and the effect of the fibrosis that replaces the damaged tissue. The nephrons that have ceased to function can play no further role in the disease, but there is always the risk that a recurrence of acute nephritis may damage more healthy units, with the reappearance of red cells and albumen in the urine and occasionally the dramatic onset of renal failure. This recurrence of acute attacks, occurring at any stage of the disease, complicates not only the clinical but also the histological picture of nephritis.

The fibrosis is slow and insidious and the patient may remain fit with normal renal function for many years. But ultimately this fibrosis leads to ischaemia of the surviving renal tissue with resultant hypertension, and, at the same time, impairs the function of the surviving nephrons. The patient then passes on to the clinical stage of chronic nephritis with hypertension and his normal renal function gives way first to compensated and finally to uncompensated renal failure and death.

When reviewing a case of nephritis, there are certain questions which one should be able to answer:—

(1) How long a history of renal disease is present?
(2) Is there any evidence of acute glomerular damage as shown by the presence of red cells in the urine?

(3) Is there any evidence of tubular degeneration as shown by the massive albuminuria, lowered blood proteins and raised cholesterol?

(4) Is there any evidence of renal ischaemia as shown by persistent hypertension?

(5) What is the renal function? Is it normal, is there compensated renal failure as shown by renal function tests, or is there decompensated renal failure and uræmia?

Armed with answers to these questions, the clinical state can be more surely judged and the prognosis more accurately assessed than is the case when the diagnosis is made in terms of a complex classification.

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