Immunofluorescent localization of Tamm-Horsfall mucoprotein in human kidney


A fluorescein-labelled antiserum to human Tamm-Horsfall mucoprotein has been used to demonstrate the localization of Tamm-Horsfall mucoprotein within the kidney. The nature of tubules specifically fluorescing with the labelled antiserum was indicated by staining alternate sections for alkaline phosphatase and succinic dehydrogenase as well as with haematoxylin and eosin. Specificity of antibody staining was confirmed by showing that it could be blocked by prior application of unconjugated antibody. In human kidney sections from operation specimens bright fluorescence was present intracellularly particularly in certain tubules in the outer medulla and in medullary rays. In the cortex there were occasional fluorescent tubules usually closely related to glomeruli. The glomeruli themselves were not fluorescent. There appeared to be little or no fluorescence in proximal tubules, the identity of which was indicated by their positive staining for alkaline phosphatase in adjacent serial sections. Heavy staining for succinic dehydrogenase in adjacent serial sections indicated that fluorescence in the medulla and medullary rays was situated in the ascending limbs of the loop of Henle. Intracellular fluorescence was never found in collecting tubules.

The presence of high concentrations of Tamm-Horsfall mucoprotein in the ascending limb cells, suggests that it may be secreted into the tubular lumen in this region. Aggregation into casts could then occur with increasing solute and hydrogen ion concentration in the distal convolutions and particularly in the collecting tubules. The occasional fluorescent areas in the cortex appeared to be related to the macula densa region and may provide a method for histological identification of this region.

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Renal biopsy study of juxtaglomerular cells in renal failure. Analysis of 1906 biopsies performed in 1450 cases


A study of juxtaglomerular cell counts in patients with renal diseases has been performed (1609 Biopsies in 1450 Cases). The quantitative analysis was carried out by counting the cells of 6 J.G.B. (afferent arterioles and Polkissen), using a microscope with a videoscope and drawing attached. The J.G.B. was projected onto a graph paper and a line corresponding to J.G. area (planimetrically evaluated) was drawn, counted and grouped in granulated and degranulated.

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The ‘normal’ J.G.C.C. was 33,6 cells ± 9,06 σ. In acute renal failure either ‘glomerular’ (acute cortical necrosis, acute glomerulonephritis) or ‘tubular’ (acute tubular necrosis, acute papillary necrosis) an increase of J.G.C.C. was found which did not correlate with blood pressure, duration of anuria, degree of BUN and electrolytes abnormalities. A correlation of clinical significance...
was the higher percentage of granulated J.G. cells in acute hypertensive with respect to normotensive renal failure. After extracorporeal haemodialysis serial renal biopsies demonstrated a normalisation in the J.G. cytology either in ‘glomerular’ or in ‘tubular’ acute renal failure even in those cases in which the arterial hypertension persisted. In them a correlation between B.P. and the degree of acutely induced and persisting arteriolar lesions was found. In chronic renal failure a significant increase in J.G.C.C. was rarely found. In chronic decompensated forms (G.F.R. < 30 ml/min.) such increase was never found. In compensated forms (G.F.R. > 30 ml/min.) a marked increase was observed in the malignant phase of essential hypertension, and in reno-vascular hypertension. This was the only clinical situation showing an increase in the cells three-fold the normal, with a percentage of granulated cells above 25%.

Serial renal biopsies carried out in cases of reno-vascular hypertension before and after surgery (aorto-renal artery by-pass) characteristically showed a morphological normalisation in J.G. cells.

Renal biopsy have been finally performed in 2 cases who underwent kidney transplantation. In one of them an increase in J.G.C.C. with a moderate hyper-granulation was found in the graft 2 days after the transplant during an early hypertensive rejection crisis. 1 month later, a third renal biopsy demonstrated a normal J.G.C.C. The B.P. also returned to normal. In the other case no demonstrable J.G.C.C. changes in the graft during a similar early rejection crisis have been found. The first case presented an excellent histocompatibility (donor and recipient) and the vessels anastomosis was rather difficult during surgery.

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Primary familial amyloidosis

The first in Soviet literature description of primary familial amyloidosis is given. 11 of 13 members in three generations of Russian family (from North of the European part of USSR) had different allergic disorders (Quincke’s oedema of the face, skin eruptions, myalgias, arthralgias, abdominal pain with fever and diarrhea), with the dominant type of inheritance. Six patients had a nephropathy. Five patients died, three of them from uremia. In two patients renal biopsy revealed amyloid deposits in mesangial cells of glomeruli, in the blood vessels and in the perivascular connective tissue. Amyloid was found also in the tonsils (two cases) and in one case at autopsy – in the blood vessels of kidneys, liver, spleen and lungs. Histochemical properties of amyloid and its localisation were characteristic of primary type.

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Summaries – Resumes

Plasma renin activity in patients with coarctation of the aorta. A comment on the pathogenesis of the prestenotic hypertension

In 10 patients with coarctation of the aorta, plasma renin activity (PRA) in peripheral venous blood was measured after recumbency and orthostasis as well as in some cases in renal venous blood. In 9 of these patients, basic PRA values and those under stimulatory conditions lay within normal range and in 1 patient only were the values raised. However, since this patient displayed
the lowest gradient of the mean arterial blood pressure above and below the stenosis, it can be regarded as improbable that the elevation in PRA was caused by the coarctation. PRA in renal venous blood of both kidneys, which was measured in 3 patients showed no elevation.

On the basis of observations by other authors and our own studies, it can be deduced that the kidneys can contribute to the development of prestenotic hypertension in acute constriction of the aorta in experimental animals, but that in chronic coarctation in animals or in man the humoral or renal theory has no essential importance as to the pathogenesis of hypertension.

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