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Metaplasia of the parietal layer of Bowman's capsule in the human kidney: Incidence in alcoholic liver disease and hypertension

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Summary. This report is the second of two surveys to determine the incidence of metaplasia of Bowman's parietal epithelium in the human kidney. Human kidney sections obtained at autopsy at the Department of Pathology, University of Texas Medical Branch, Galveston, Texas, were examined with the light microscope. The kidneys were fixed in neutral formalin, sectioned at 6 μm and stained with hematoxylin and eosin. Autopsy records were consulted after kidney section examination to determine if there was any correlation between clinical disease, histopathological changes in organ systems and metaplasia of Bowman's capsule. The kidney sections represented both sexes in 8 age groups, from less than one year to 80 years. A total of 174 kidneys, representing 174 individuals, were evaluated. One hundred renal corpuscles were counted per section and the parietal layer of Bowman's capsule was classified as normal (squamous) or metaplastic (cuboidal). Of the 174 kidneys examined, 137 (79%) - 79 male and 58 female - had metaplasia of Bowman's capsule. On the average, in the kidneys with the lesion, 6% of the renal corpuscles had metaplasia of Bowman's parietal layer. The lesion was present in both sexes in all age groups. The autopsy records revealed that metaplasia of Bowman's parietal epithelium was usually present with hepatic fatty changes and/or congestion. Alcoholic liver disease and hypertension represented the most frequent clinical diseases in the sample; these conditions had the highest incidence of metaplasia. Twenty-six of the 174 kidney samples were from individuals with alcoholic liver disease, all of whom had metaplasia of Bowman's capsule. In the latter kidneys the mean percentage of glomerular capsules with metaplasia was 14%, with a range of 1 to 46%. Thirty-eight of the 174 kidney samples were from individuals with hypertension, 29 (76%) of whom had metaplasia of Bowman's capsule. In these kidneys the mean percentage of glomerular capsules with metaplasia was 6% with a range of 1 to 16%. These observations suggest that metaplasia of Bowman's parietal epithelium is a common occurrence in the human kidney under different pathological conditions, and is especially prominent in alcoholic liver disease and hypertension.

Key words: Human kidney, Glomerular capsule, Metaplasia, Alcoholic liver disease, Hypertension

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Introduction

The normal histomorphology of the parietal layer of Bowman's capsule (pars externa) in the kidney is a layer of simple squamous epithelium (Fawcett, 1986). Under certain circumstances, however, this layer has a structural appearance of a cuboidal to low-columnar epithelium. The cause of this transformation is unknown, and different terms have been used to describe the condition (Haensly and Lee, 1986). For the sake of consistency in this report the terms *metaplasia* and *lesion* will be used with reference to this modified tissue.

The literature dealing with metaplasia of the parietal layer of Bowman's capsule was reviewed in a recent report (Haensly and Lee, 1986). In brief, the lesion was first reported to occur in the mouse kidney, appearing to be age and sex dependent (Crabtree, 1941). Since then, the lesion has been observed in the rat and human kidney under varying circumstances. Metaplasia of Bowman's capsule in the rat kidney has been associated with the aging process (Haley and Bulger, 1983), unilateral nephrectomy (Andrew, 1981), and hypertension (Haensly et al., 1982). Eisen (1946) was one of the first investigators to describe metaplasia of the parietal layer of Bowman's capsule in the human kidney, present in a 54-year-old woman with carcinoma of the gallbladder. Since that time the renal lesion has been reported in conjunction with other malignancies; a 16-year-old male

with an adrenal tumor (Chappell and Phillips, 1950); a 6-month-old male with a primary carcinoma of the liver (Nachman, 1962); a 63-year-old female with cholangiocellular liver cancer (Eulderink, 1964); carcinoma of the ovary in a 40-year-old woman (MacPherson, 1963); and squamous cell carcinoma of the esophagus in a 63-year-old female (Reidbord, 1968). In most of these cases there was a metastasis to the liver. The possibility that liver cancer might be involved in the etiology of the lesion was suggested from these studies (Von Scheele, 1967). To test this hypothesis, an estimate was made on the frequency of the renal lesion in 100 cases of primary liver cancer (Von Scheele, 1967). In none of these cases was metaplasia of the parietal layer of Bowman's capsule present. The author concluded that the association of renal metaplasia with liver tumors was a coincidence.

In an attempt to obtain more definitive information that may lead to an understanding of the etiology and significance of metaplasia of the parietal layer of the glomerular capsule, two histopathological surveys were conducted on the human kidney. The results of the first survey were recently reported by Haensly and Lee (1986), and demonstrated the following: 1) The lesion was present in both sexes in nine age groups from 2 to 87 years. 2) Of the 129 kidney sections examined, representing 129 individuals, 69 (53%) had metaplasia of Bowman's capsule. 3) On the average, in the kidneys with metaplasia, 4% of the renal corpuscles had metaplasia of Bowman's parietal layer. 4) Metaplasia of Bowman's parietal layer was usually present with hepatic fatty changes and congestion. Further, there was no common clinical condition associated with the lesion and none of the kidney samples in this first survey were from individuals with alcoholic liver disease. The observations obtained in the second survey of the human kidney are presented in this report.

Materials and methods

Human kidney sections, taken at autopsy at the Department of Pathology, University of Texas Medical Branch, Galveston, Texas, were examined with the light microscope. The kidneys were fixed in neutral formalin, sectioned at 6 μm, and stained with hematoxylin and eosin. Excluded from this study were young developing kidneys and sections showing extensive histopathologic conditions and/or autolysis such that the histology of the parietal epithelium of the glomerular capsule could not be determined. One section per kidney was evaluated and when right and left kidney sections were available, the data were pooled to represent information from one individual.

For each renal corpuscle the epithelium of the parietal layer was classified as squamous (normal) or cuboidal (metaplastic). One hundred renal corpuscles were counted per section and the percentage of capsules with metaplasia was determined. Kidneys from 174 individuals were evaluated for the presence of the lesion and its frequency.

As in the previous survey (Haensly and Lee, 1986), the autopsy records were consulted after the kidney sections were examined. Disease processes at the time of death and histopathologic conditions recorded at autopsy in the kidney and other organs system were compared with the observations made on the renal corpuscle parietal epithelium.

Results

Metaplasia of Bowman's parietal epithelium was readily identifiable in the kidney samples examined in this survey. The lesion involved varying degrees of the parietal epithelium, ranging from a few cells to capsules where the entire surface was involved. An example of the metaplastic tissue is shown in Fig. 1.

Table 1 summarizes the overall observations obtained in the second survey which contained eight 10-year age groups with ages from 0.5 to 80 years. Metaplasia of the glomerular capsule was again observed in both sexes and in all age groups. The lesion was present in 137 (79%) of the 174 kidneys (individuals) examined, with a mean percentage of glomerular capsules with metaplasia of 6%. The histopathologic parts of the autopsy reports again showed hepatic congestion and fatty changes as a condition usually present when there was metaplasia of Bowman's parietal epithelium in the kidney. Seventy-eight (57%) of the 137 individuals with metaplasia of the glomerular capsule also had hepatic congestion, and 87 (64%) had hepatic fatty changes.

While the mean percentages of glomerular capsules with metaplasia was relatively small (6%), the ranges of the percentages were much larger than observed in the first survey. These higher ranges were present in two clinical conditions which were also the conditions most frequently represented, alcoholic liver disease and hypertension. These data were separated from the main body and are presented below in Tables 2 and 3. The data for the hypertensive group is summarized in Table 2. The hypertensive individuals were in the older age groups, and 29 (76%) of the 38 had metaplasia of the parietal layer of Bowman's capsule. The mean percentage of glomerular capsules with metaplasia was 6% with a range of 1 to 16%. Hepatic congestion and fatty changes were common histopathological conditions present in all 29 cases associated with hypertension and metaplasia of Bowman's parietal layer. Three of the individuals with hypertension also had alcoholic liver disease.

Twenty-six of the 174 individuals (Table 1) had alcoholic liver disease and a history of alcohol abuse. The data for this group is summarized in Table 3. All of the kidneys from individuals with alcoholic liver disease had metaplasia of the parietal layer of Bowman's capsule. The mean percentage of glomerular capsules with metaplasia was 14%, considerably larger than occurred for the overall sample (Table 1), for the hypertensive group (Table 2), and for the sample of kidneys examined in the first survey, 4% (Haensly and Lee, 1986). These individuals, as described in the autopsy reports, had the clinical conditions of alcoholic liver disease.

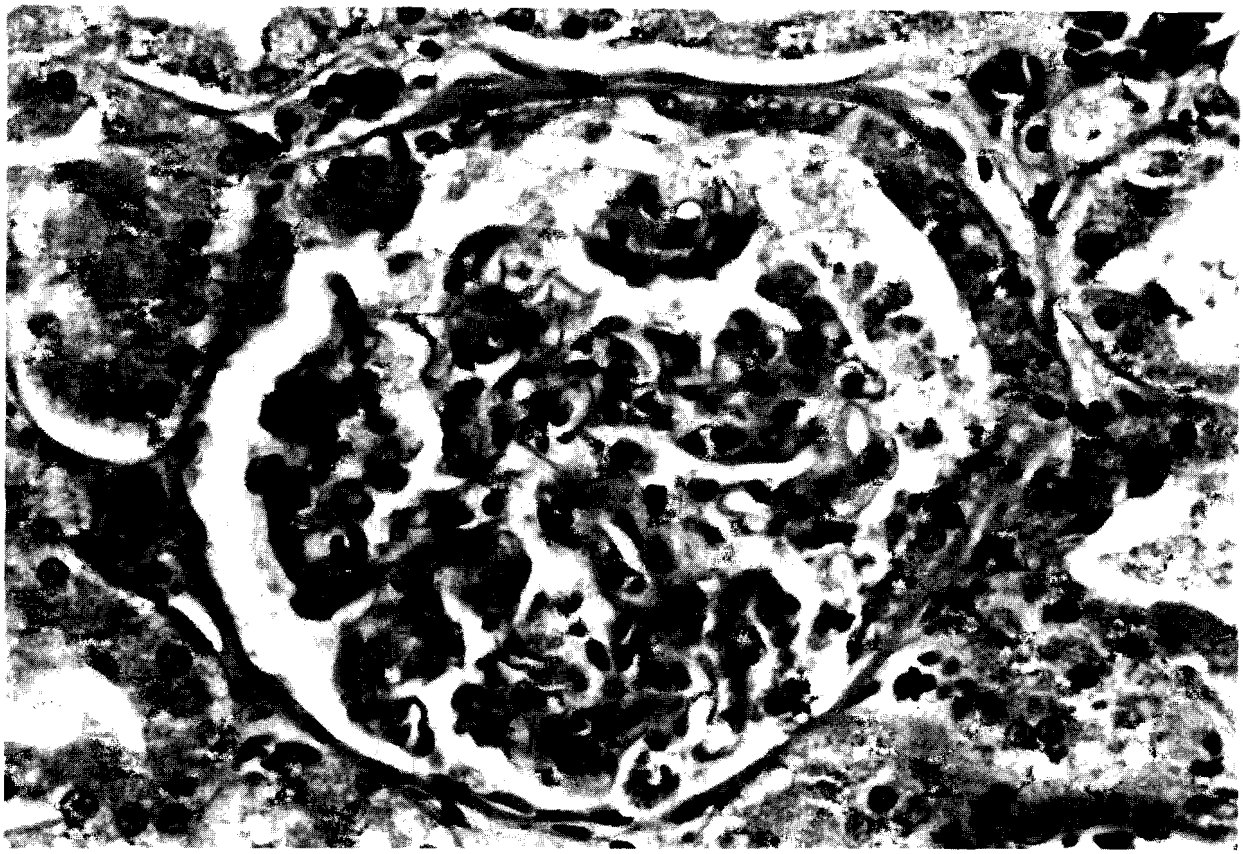

Fig. 1. Renal corpuscle, 32-year-old male; alcoholic liver disease. Metaplastic (cuboidal) layer of Bowman's parietal epithelium (H&E); (× 525).

Table 1. Human kidneys with metaplasia of the glomerular capsule, mean percentages of glomerular capsules with metaplasia, and livers (from the same individuals) with congestion and fatty changes.

Age (years)	Sex	N[a]	Individuals with metaplasia	Mean percentages of glomerular capsules with metaplasia	Livers with[b] Congestion	Livers with[b] Fatty changes
0.5-10	M	9	6	4	4	4
	F	9	6	4	4	1
11-20	M	7	4	6	2	3
	F	4	3	4	1	1
21-30	M	12	10	4	3	8
	F	6	5	11	2	5
31-40	M	17	15	9	6	11
	F	6	6	10	4	2
41-50	M	14	9	5	3	6
	F	11	10	7	6	7
51-60	M	19	18	5	15	13
	F	13	8	7	5	3
61-70	M	15	12	4	8	7
	F	16	14	8	7	10
71-80	M	9	5	5	3	2
	F	7	6	2	5	4
0.5-80	M	102	79	5	44	54
	F	72	58	7	34	33
Total		174	137	6	78	87

[a]Number of individuals.
[b]Obtained from autopsy reports.

Table 2. Kidneys from hypertensive individuals with metaplasia of the glomerular capsule, and mean percentage of glomerular capsules with metaplasia.

Age (years)	Sex	N[a]	Number of kidneys with metaplasia	Mean percentages of glomerular capsules with metaplasia
31-80	M	18	13	5 (1-16%)[b]
	F	20	16	6 (1-13%)[c]
Total	–	38	29	6 (1-16%)

[a] Number of kidneys from individuals with hypertensión. Data for right and left kidney sections, when available, were pooled.
[b] Includes one male with alcoholic liver disease.
[c] Includes two females with alcoholic liver disease.

Table 3. Kidneys from individuals with alcoholic liver disease and with metaplasia of the glomerular capsule, and mean percentage of glomerular capsules with metaplasia.

Age (years)	Sex	N[a]	Kidneys with metaplasia	Mean percentages of glomerular capsules with metaplasia
30-71	M	16	16	14 (1-46%)
26-68	F	10	10	15 (1-37%)
Total	–	26	26	14 (1-46%)

[a] Number of kidneys from individuals with alcoholic liver disease. Data for right and left kidney sections, when available, were pooled.

Discussion

The data from this and the previous survey (Haensly and Lee, 1986) indicate that metaplasia of Bowman's parietal epithelium in the human kidney may be a lesion common in both sexes at any age. Two other observations of the lesion may be quite important: 1) it was usually present in hepatic pathology, specifically congestion and fatty changes, and 2) the incidence may be higher when there is either alcoholic liver disease or hypertension than in other disease conditions.

In alcoholic liver disease, the liver appears to be the main target organ for the toxic effects of alcohol (ethanol) when there is excessive and long-term consumption of this substance. The first pathological condition usually observed is an accumulation of lipid in hepatocytes. The progressive toxic effects on the liver are hepatitis and the deposition of collagen (cirrhosis) (Desmet, 1985; Hall, 1985; Khanna et al., 1975; Lieber, 1975, 1976, 1984a,b; 1985). The fact that metaplasia of Bowman's parietal layer was also present in nonalcoholic disease conditions indicates that alcohol *per se* might not be involved directly in the etiology of the lesion. On the other hand, it appears that the hepatic pathology in alcoholic liver disease is associated with a higher incidence of the renal lesion. It remains to be determined why metaplasia of Bowman's parietal layer is so extensive in alcoholic liver disease; it might be an exaggeration of what is taking place in the liver in other disease conditions.

It is well known that the liver participates in several ways with lipid metabolism, and derangement of the metabolic regulatory pathways could interfere with the secretion of lipoprotein, which in turn could lead to disturbances in hepatic function referred to as «fatty liver», «fatty degeneration», or «fatty metamorphosis» (Alpers and Sabesin, 1982). Such fatty changes occur in a variety of conditions: alcohol abuse, diabetes mellitus, starvation, morbid obesity, adrenocorticoid therapy, and drug toxicity (Bynum, 1984). Renal dysfunction, in turn, is itself a consequence of liver disease. One of the first manifestations of renal dysfunction is a disturbance in the management of sodium and water excretion (Flamenbaum and Schmitt, 1976).

The major function of the renal proximal tubule is the reabsorption of about three-fourths of the glomerular filtrate, the primary constituents being sodium and water (Weinstein and Windhagen, 1985). Sodium retention is a major pathophysiological disturbance in certain hepatic diseases that show fatty changes in the liver (Epstein, 1982). One of the major clinical conditions in alcoholic liver disease is sodium retention which may be associated with ascites. In the course of this disease there is progressive renal failure with no apparent clinical cause, often referred to as hepatoneral syndrome (Levy, 1980). At its extremes, the syndrome is characterized by almost complete sodium retention and oliguria. Even in compensated liver cirrhosis there is sodium retention when patients are challenged with a salt load (Caregaro et al., 1985). The mechanism for sodium retention in these pathological situations remains unknown.

As would be expected, the role of aldosterone in the pathological retention of sodium has been investigated. It has been concluded by some workers that hyperaldosteronism is of mayor importance in the pathogenesis of sodium retention (Arroyo et al., 1979; Pérez-Ayuso et al., 1983). In other studies sodium retention was found to persist following the lowering of aldosterone to normal level (Epstein et al., 1977; Rosoff et al., 1975). Aldosterone levels have also been found to be normal in cirrhotic patients (Wernze et al., 1978). Other workers have suggested that while aldosterone may have an important role in the pathogenesis of ascites in cirrhosis, it may not be the main factor (Jiménez et al., 1985). If sodium reabsorption from the proximal tubule is a major factor in alcohol liver disease, then the role that aldosterone might have in this segment of the nephron is unknown. As yet there is no evidence that aldosterone has any effect on sodium transport in the proximal tubule.

Human clinical and epidemiological studies have shown that blood pressure may be abnormally elevated in alcohol abuse (Arkwright et al., 1981; Beevers, 1977; Celentano et al., 1981; Ibsen, 1981; Ireland et al., 1984; Klatsky et al., 1977; Potter and Beevers, 1984; Saunders et al., 1979). Those working to find a link between alcohol and hypertension have often been confronted with sorting out confounding factors such as smoking and obesity, as well as the part played by nutrition and genetic predilection (Friedman et al., 1982). A direct hypertensive effect of alcohol has not yet been clarified because of this.

Numerous hypotheses have been proposed to explain the possible pressor effects of alcohol. These include increased sensitivity of vascular smooth muscle to vasoconstriction (Knochel, 1983), increased circulating levels of adrenalin (Ireland et al., 1984; Saunders et al., 1979) and sodium retention with plasma volume expansion (Chan and Sutter, 1983). Others have suggested that changes in renal structure and function in alcoholism may cause hypertension (De Marchi and Cecchin, 1985; Linkola et al., 1979). The problem of identifying a mechanism linking alcoholism and hypertension has been further complicated by the fact that during the treatment of alcoholic patients there is a withdrawal period when blood pressure may become abnormally elevated (Bannon et al., 1984; Carlsson and Haggendal, 1967; Friedman et al., 1982; Saunders et al., 1979; Wallace et al., 1981).

Attempts have been made to identify a cause and effect relationship between blood pressure and alcohol cosumption by using experimental animals, especially the rat. The observations from these studies have been variable when the spontaneously hypertensive rat (SHR) has been used. In alcohol-fed female SHRs there was a significant elevation of arterial pressure (Mankes et al., 1985), while in another group of SHRs alcohol appeared to have no effect on blood pressure and the development of hypertension (Kheterpal and Volicer, 1979). In a third experiment with SHRs there was actually a decrease in blood pressure following long-term treatment with alcohol (Sanderson et al., 1983). The cause of this variation in blood pressure in alcohol-treated SHRs is not known, but an earlier report demonstrated an age difference in the rate of alcohol metabolism; it is more rapidly metabolized in young SHRs (Israel et al., 1977). More consistent results have been obtained in normal, non-hypertensive rats. Wistar Kyoto rats (WKY) treated with alcohol showed a dramatic elevation of blood pressure (Mankes et al., 1985), as did Wistar rats (Chan and Sutter, 1983). As in human studies, however, the animal investigations have not identified a mechanism in the development of hypertension in alcoholism.

We first reported that metaplasia of Bowman's parietal epithelium was a prominent kidney lesion in SHRs (Haensly et al., 1982), a strain that develops hypertension with advancing age. It may be more than a coincidence that, as presented in this report, the lesion was also observed with a high frequency in human alcoholic liver disease, a condition associated with hypertension. The kidney lesion might be an important component in the biological mechanism leading to high blood pressure and the pathological disturbances seen in chronic alcoholism.

Our current experimental studies are directed toward determining if, in fact, metaplasia of the Bowman's parietal epithelium does occur during chronic ethanol consumption and the simultaneous development of a high blood pressure. Such a model would be a valuable tool in the study of the relationship between liver disease and renal failure.

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