Case Report

Adenomatous Metaplasia of Renal Glomerular Capsular Epithelium Associated with Carcinoma of the Liver

Tsuneaki SUGIMOTO, M. D., Takao IKEDA, M.D., Eiichi
UCHIDA, M. D., *and* Hideo UEDA, M. D.

THE adenomatoid transformation of the epithelial cells of Bowman's capsule is presented. Four cases of similar findings were reported previously.

CASE REPORT

A 34-year-old man was admitted to University of Tokyo Hospital on April 1, 1961, complaining of shortness of breath and enlargement of abdomen. According to the patient, the swelling of the liver had been present for the past 5 years. In December 1960, suffering from fever and general malaise, he was admitted to a hospital and diagnosed to have a malignant tumor of the liver. There was no previous history of hepatitis or nephritis.

In the family of this patient, many members were said to have cirrhosis or carcinoma of the liver. His mother died of cirrhosis of the liver at the age of 48. Two of his 3 siblings were disclosed to have cirrhosis of the liver, one of whom died of carcinoma of the liver at the age of 28.*

Physical examination on admission disclosed a temperature of 36.6°C, pulse of 100 and blood pressure of 132 systolic and 60 diastolic. The abdomen was symmetrically enlarged. The veins of the abdominal wall were slightly distended. The liver edge was 8 cm. below the right costal margin, and the spleen extended 4 cm. below the left costal margin. The liver was felt firm and its surface was nodular. There were gynecomastias and many spider angiomas on the upper chest.

Laboratory findings on admission revealed hemoglobin of 12.5Gm. per 100ml.,

From the Second Department of Internal Medicine, Faculty of Medicine, University of Tokyo.

* The familial occurrence of cancer or cirrhosis of the liver of this patient was reported in J. Jap. Soc. Int. Med. **50**: 1307, 1962 (In Japanese).

red-cell count of 4,120,000 and white-cell count of 11,300, with a slight shift to the left. The urine was normal. The icterus index was 10, the total protein 7.0 Gm., with an albumin-globulin ratio of 0.69, cholesterol 273 mg., N.P.N. 29 mg., alkaline phosphatase 10.4 units per 100 ml., and serum sodium was 142 m Eq., potassium 4.9 m Eq., and chlorine 107 m Eq. per liter. The B. S. P. retention was 25% at 45 minutes. Serologic tests for syphilis were negative. The X-ray film of the chest revealed multiple metastases in the lungs.

A diagnosis of carcinoma of the liver was made and the liver-protecting therapy and the administration of vitamins and antibiotics were performed. However, a recurring fever was occasionally noted and anorexia, abdominal pain and nausea continued. In spite of the use of diuretics such as chlorothiazide or aldactone, the ascites and edema progressively increased. Nutritional impairment and jaundice developed. Although the urine was normal and a P. S. P. test revealed 68 per cent excretion of dye in 2 hours, the serum N.P.N. gradually increased to 154 mg. per 100 ml. The serum potassium also became elevated to 6.5 m Eq. per liter. There gradually appeared hemorrhagic tendency, such as subcutaneous petechial hemorrhages, retinal hemorrhages, epistaxis and melena. Anemia became marked. Finally, on May 16, the patient died of hematemesis and shock.

At necropsy the body was deeply jaundiced with a moderate amount of edema and subcutaneous petechial hemorrhages. About 3,000 ml. of thin, turbid, yellow fluid was present in the peritoneal cavity. The heart weighed 320 Gm. The right atrium was slightly dilated. The lungs weighed 1,140 Gm. There were numerous tumor cell nodules. The spleen weighed 250 Gm. The liver weighed 2,800 Gm. Its external and sectioned surfaces were diffusely nodular, the nodules varying from 5 to 10 mm. in diameter. Fine, retracted fibrous septums separated the nodules. In the right lobe of the liver, multiple hepatomas, the largest of which was of fist-size, were observed. There was extramedullary hematopoiesis in the midst of the nodule of the hepatoma. The hepatic vein and the portal vein were occluded with the thrombi of the tumor cells. The secondary carcinomas were found in the adrenal glands, the omentum and the peritoneum of the small pelvis. In the stomach, there was bloody content of 700 ml. and an acute ulcer was found.

Each kidney weighed 220 mG. The capsule stripped readily. On section the renal tissue was moderately jaundiced. Corticomedullary differentiation was distinct and all markings appeared normal. The pelves, calyces and ureters were normal. Histologic examination of the kidneys disclosed notable findings. Almost all of the glomeruli in both kidneys showed a marked metaplasia or adenomatoid transformation of the cells lining the parietal portion of Bowman's capsule. The changes consisted of a replacement of the normal flattened epithelium of Bowman's capsule by a layer of tall columnar cells. This layer was usually one cell in thickness; but in some areas, particularly near the exit of the proximal convoluted tubules, it was 2 or 3 cells in thickness. Semilunar accumulations of stratified cells sometimes covered the pole of the glomerulus opposite to the hilum. Occasionally the metaplastic changes extended a short distance into the proximal convoluted tubules. The glomerular tufts and their visceral epithelial cells were generally unchanged.

COMMENT

Eisen¹⁾ first observed such a metaplastic change of Bowman's capsule in a 54-year-old woman with carcinoma of the gall-bladder which widely involved the liver. Chappel and Phillips²⁾ noted an identical lesion in a 16-year-old boy with carcinoma of the adrenal gland and multiple metastases in the liver. Recently Nachman³⁾ reported such a lesion found in a 16-month-old female infant with carcinoma of the liver. In the description of Kanisawa and Isono,⁴⁾ they observed the same change in a 66-year-old man with carcinoma of the esophagus and metastases in the liver, and considered that the metaplasia of Bowman's capsule was due to chronic toxication of narcotics.

The possibility exists that those changes of Bowman's capsule are in fact metastatic foci. However, it is difficult to explain those changes on a metastatic basis. No tumor cells were found in the glomerular tufts, as would be expected if these cells were blood-borne to the parietal capsular surface. There was no evidence of invasion beyond the basement membrane into the surrounding parenchyma. No mitoses were observed. The benign characteristics of those changes are identical to the previously reported cases and suggest that the changes are of metaplastic nature.

Although we cannot make its pathogenesis clear, the finding that this case was also associated with carcinoma accompanied by liver damage like the previously reported cases suggests a possible relationship with carcinoma or with liver disease.

The azotemia may indicate impairment of renal function, but there was no evidence of prerenal or renal basis for the retention of urea. The

Fig. 1. Kidney, showing adenomatoid transformation of capsular epithelium with occasional extension into convoluted tubules. Hematoxylin and eosin stain. $\times 60$.

Fig. 2. $\times 240$.

blood pressure and urine output did not show significant changes. The urine was normal and the P.S.P. test performed a few days before the death revealed normal renal function. Chlorothiazide may cause increased concentration of blood urea and aldactone tends to encourage retention of potassium. Moreover, it has been known that in the end stadium of cirrhosis of the liver slight or sometimes marked azotemia may be observed not infrequently even without oliguria. Therefore, it may be impossible to decide whether the azotemia and hyperpotassemia are due to the metaplastic changes of Bowman's capsule or not.

SUMMARY

In a 34-year-old man with carcinoma of the liver, adenomatous metaplastic changes of the parietal portion of Bowman's capsule were observed. From the fact that this case as well as the previously reported 4 cases were associated with carcinoma with liver damage, carcinoma or liver disease may be considered to concern with the pathogenesis of such a lesion.

REFERENCES

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