MULTIPLE MYELOMA PRESENTING CLINICALLY AS OBSTRUCTIVE JAUNDICE

A Case Report

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Multiple myeloma may present initially in many different ways, the most usual being bone pains, spontaneous fractures, loss of weight, anaemia, vertebral collapse (which may give rise to neurological signs), recurrent pneumonia and renal failure.

The liver is commonly infiltrated with myeloma cells and the patient may subsequently become jaundiced due to this hepatic damage, but we have, however, been unable to find any case in the literature in which the disease first presented clinically with the symptoms and signs of obstructive jaundice.

Case History

W. N., aged 78, was admitted to hospital in November 1958 with a history of increasing jaundice for the previous two weeks, dark urine and pale stools. In addition he complained of central chest pain radiating to the epigastrium and right hypochondrium. Since the jaundice began he had become anorexic and had lost almost ten pounds in weight.

On questioning it was found that there had been no dyspnoea, cardiac pain or oedema and digestion, micturition and bowels were normal. There was no history of any previous illness and the patient neither drank nor smoked, having given up the latter some thirty years previously.

On examination, the patient was fully conscious and co-operative, thin, with obvious jaundice of the skin and sclera, and with scattered skin scratch marks. The cardiovascular system, except for a strongly positive Hess's test, and the respiratory system were normal. The liver was enlarged for some ten centimetres below the right costal margin, its surface being hard and the edge sharp, slightly irregular and tender. No other signs of hepatic disease were found, there being no spider naevi, no ascites, and no distended abdominal veins and the palms were normal in colour.

On admission, the haemoglobin was 10.5 gr. per cent. and the white cell count 6,300 per cmm.,

FIG. 1.—Liver on sectioning. Note the numerous haemorrhagic areas in the parenchyma.

FIG. 2.—Liver. Haematoxylin and eosin. $\times 93$

whilst an examination of the film revealed no abnormalities. The fall of red blood cells in one hour was 132 mm. (Westergren's method.) Total serum protein was 7.8 gr./100 ml. with albumin 3.4 g./100 ml. and globulin 4.4 g./100 ml. The total bilirubin was 24.5 mg./100 ml. while the direct reacting bilirubin was 18 mg./100 ml. The serum alkaline phosphatase was 24 units/100 ml. and the thymol turbidity 1 unit while the thymol flocculation and colloidal gold tests were negative. The urine, on examination, was noticed to be very dark and contained an excess of bilirubin, a normal amount of urobilinogen and only a trace of urobilin.

A week later the total serum bilirubin had risen to 35 mg./100 ml., whilst the direct reacting bilirubin was now 27 mg./100 ml. The serum alkaline phosphatase was 41 units/100 ml., the thymol turbidity was 1 unit and the thymol flocculation and colloidal gold tests were still negative while the blood urea had risen to 78 mg./100 ml.

No opaque gall stones were visible on a straight X-ray of the abdomen and the cause of the obstructive jaundice could not be clinically established. Electrophoresis of the serum proteins revealed a dense band in the β globulin region which was thought to be that of multiple myeloma. There

FIG. 3.—Liver. Haematoxylin and eosin. $\times 504$

was a proteinuria of 0.2 gr. per cent. and, although a Bence-Jones protein could not be demonstrated on heating, electrophoresis demonstrated a dense band of an abnormal globulin in the γ globulin region. X-rays of the skull, ribs, spine and long bones showed no abnormality. A sternal marrow aspiration was then done (Dr. W. J. D. Fleming) and revealed the presence of atypical cells which had the appearance of plasma cells and the diagnosis of multiple myeloma was then considered to have been established.

The patient's condition deteriorated rapidly after admission. The anorexia became absolute, and with the deepening jaundice he became lethargic, then drowsy, and ten days after his admission he developed a petechial rash over the shoulders, vomited a small amount of fresh blood and then lapsed into coma with a flaccid paralysis and a right extensor plantar response. The coma was thought to be due to liver failure and the haematemesis and skin bleeding to the reduced prothrombin concentration—50 per cent. of normal—and a low platelet count of 80,000 per cmm. Treatment with oral neomycin and intravenous 20 per cent. dextrose was started but had no effect and the patient died 18 hours after the onset of the coma.

At autopsy (P.M. 810/59), the body was seen to

be severely jaundiced and showed multiple cutaneous petechial haemorrhages, most notably on the flexor surfaces of the limbs. The cause of death was found to be a left-sided sub-arachnoid haemorrhage with multiple small cerebral haemorrhages. There was also severe pulmonary congestion and oedema, a right-sided plural effusion and multiple haemorrhages into all the tissues of the body.

The liver was enlarged, weighed 2,545 gr. and had numerous subcapsular haemorrhages scattered over its surface. On section numerous haemorrhagic areas, both large and small, were seen to be scattered throughout the hepatic parenchyma (Figure 1). No extra-hepatic obstruction of the biliary system could be demonstrated despite a careful search. The gall bladder and bile ducts were normal in appearance, contained no calculi and showed no evidence of inflammation, strictures or neoplasm while there was no enlargement of lymph glands in the porta hepatis. The pancreas contained no neoplastic, inflammatory or fibrotic changes which could have caused obstruction to the common bile ducts.

Histological examination of the liver showed a gross degree of infiltration with myeloma cells and some necrosis of liver cells, together with fatty infiltration of many of the remainder. (Figures 2 and 3.) In addition, there were numerous haemorrhagic areas filled with red blood cells and some polymorphs and lymphocytes were scattered through the liver parenchyma. There was no portal or biliary cirrhosis and the presence of amyloid could not be demonstrated by methyl-violet or congo-red stains.

Discussion

Visceral involvement has been noted, to some extent, in many cases of multiple myeloma, either by evidence of hepatomegaly or splenomegaly as in the series of Snapper *et al.* (1953)¹, who found palpable hepatomegaly in 40 per cent. of their cases and hepatosplenomegaly in a further

23 per cent., or at autopsy by histological evidence of infiltration of these organs with myeloma cells; Churg and Gordon (1942)² found that 73 per cent. of their series of thirty cases had some infiltration of the liver with plasma cells.

However, diffuse visceral infiltration may occur without invasion of the peripheral blood by plasma cells, as in the patient reported by Stark and Amidon (1948)³, where the clinical picture was very similar to that presented here. In their case the man was admitted for investigation of loss of weight and chest pain and was found to be jaundiced, with an enlarged liver; widespread skeletal involvement was found on radiological examination. Sternal marrow biopsy established the presence of myeloma. At autopsy no cause for the obstructive jaundice could be found but the liver was extensively infiltrated with myeloma cells.

It is difficult to understand how infiltration of the liver by plasma cells can give rise to an obstructive jaundice but the probable explanation is that the cellular infiltration disrupts most of the bile canaliculi and those which are not disrupted have their lumen occluded by pressure from plasma cells on the surrounding hepatic cells.

Summary

A case of multiple myeloma which presented clinically as obstructive jaundice is described. A search of the literature has revealed no previous report of a similar case.

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