

RADIOLOGICAL PRESSURE DEFORMITY OF THE SUPERIOR ASPECT OF THE DUODENAL BULB WITH SPECIAL REFERENCE TO ITS SIGNIFICANCE IN PANCREATIC LESIONS

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The radiological demonstration of malignant or inflammatory lesions of the pancreas is frequently difficult or impossible.^{1,2} A suggestive or positive diagnosis of such lesions is dependent on the interpretation of distortion, displacement, invasion, or obstruction of adjacent viscera, usually the duodenal loop.³⁻⁵ Scant attention has been paid to a particular deformity affecting the superior border of the duodenal bulb, which, in our experience, may provide the only radiological clue to a pancreatic mass.

The purpose of this paper is to draw attention to the radiological technique which facilitates the demonstration of this deformity,⁶ to correlate the radiological findings with the underlying pathology and, in addition, to consider lesions other than pancreatic masses that may be responsible for this particular deformity.

RADIOLOGICAL TECHNIQUE

The technique is similar to that described by Hampton⁷ for the examination of the duodenal bulb after recent haematemesis from presumed peptic ulceration. Demonstration of the deformity is attempted in the concluding phase of the barium-meal examination of the oesophagus, stomach and duodenum, when a substantial residue of the half-pint of flocculation-resistant barium given to the patient is still present in the stomach. The patient is rotated, under screen control, from the supine to the supine right anterior oblique position, with the right side elevated approximately 30° anteriorly. This rotates away overlying shadows and opens out the duodenal loop. Since the loop is now uppermost, swallowed air passes from the stomach and distends the duodenal cap and loop as in the double-contrast method. The thin coating of barium is an essential step in revealing minor deformities of outline. Gentle rotation of the patient from side to side through an arc of 60° may help to demonstrate the deformity by further distending the duodenal bulb.

The deformity we wish to emphasize in the present paper affects the superior border of the duodenal bulb and may extend to involve the superior border of the first part of the duodenum beyond the bulb as well. The contour of the deformity (Fig. 1) is semilunar, with the convexity directed inferiorly; if the deformity is large enough, the proximal portion of the duodenal loop may appear constricted (Fig. 2). Digitate impressions of the duodenal bulb or of the concave inner border of the duodenal loop may also be present in some cases.

CLINICAL MATERIAL

The clinical material was derived from the data of 10 consecutive patients with the above-mentioned deformity

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of the duodenal cap. The diagnosis and the cause of the radiological abnormality was established by means of laparotomy in all 10 patients. The series comprised 7 patients with pancreatic disease, 1 with a carcinoma of the common bile duct, 1 with cholangiohepatitis, and 1 with a carcinoma of the stomach. The group with pancreatic disease consisted of 4 patients with carcinoma, 2 with pseudocysts, and 1 with an inflammatory mass.

Case 1

A 72-year-old female was admitted with a 2-month history of anorexia and epigastric pain radiating through to the back, and a more recent history of progressive jaundice, dark urine and pale stools. The liver and gallbladder were enlarged, and there was evidence of free fluid in the peritoneal cavity. The haemoglobin level was 14.3 G. per 100 ml., and the erythrocyte sedimentation rate (ESR) was 49 mm. in the 1st hour (Westergren). The urine contained bile, but no urobilin. The serum-bilirubin level was 17.7 mg. per 100 ml., the alkaline-phosphatase level was 65 King-Armstrong (K-A) units, and the cephalin-cholesterol flocculation test (CCFT) was negative. The barium-meal examination (Fig. 2) showed depression from above of the duodenal cap, and the remainder of the first part and the upper portion of the second part of the duodenum; this gave an impression of complete flattening of the area. Laparotomy revealed a large, hard, neoplastic pancreatic mass with a distended gallbladder and ascites.

Comment: This elderly lady, with a clinical history suggestive of obstructive jaundice caused by malignancy, showed radiological evidence of pressure deformity of the superior aspect of the duodenal cap. This was due to carcinoma of the pancreas.

Case 2

A 54-year-old labourer presented with a 5-month history of epigastric pain, anorexia, nausea and weight loss, and a more recent history of jaundice, dark urine and pale stools. A firm 4-finger hepatomegaly and an enlarged gallbladder were present on examination, and the ESR was 85 mm. in the first hour (Westergren). The urine contained bile, but no urobilin. The serum-bilirubin level was 21 mg. per 100 ml., and the alkaline-phosphatase level was 20.6 Bodansky units. The barium-meal examination gave evidence of extrinsic pressure on the duodenal cap. The first part and the upper portion of the second part of the duodenum were dilated, and the posterior aspect of the lower half of the second part was deformed and partially obstructed in certain postures. Laparotomy revealed a neoplastic mass, 4 cm. in diameter, in the head of the pancreas. The gallbladder and the common bile duct were distended; neither appeared to be compressing the duodenal cap. There was no evidence of liver or lymph-gland metastases.

Comment: Radiological evidence of an extrinsic pressure deformity of the duodenal cap in this patient with obstructive jaundice was due to a carcinoma in the head of the pancreas.

Case 3

A 61-year-old labourer presented with a 6-week history of anorexia, lassitude, progressive jaundice, generalized pruritis and the passage of dark urine and pale stools. A smooth 4-finger hepatomegaly was found on examination. The ESR was 97 mm. in the 1st hour (Westergren), and the urine contained bile, but no urobilin. The serum-bilirubin level was 10.2 mg.

per 100 ml., the alkaline-phosphatase level was 55.8 Bodansky units, and the thymol turbidity was 1 unit. The barium-meal examination (Fig. 3) showed an intrinsically normal cap, which was compressed from above and posteriorly by a mass. The second part of the duodenum showed no abnormality. Laparotomy revealed a carcinoma of the head of the pancreas. The gallbladder and the common bile duct were distended. There were no enlarged lymph nodes.

Comment: The radiological findings in this patient, with a recent history of obstructive jaundice, were confined to the superior aspect of the duodenal cap, and were due to pressure from a carcinoma of the pancreas.

Case 4

A 71-year-old housewife was admitted with a 3-week history of progressively severe jaundice and weight loss. Nausea was present at the onset and generalized pruritus later. The jaundice was obstructive in type and unassociated with pain. The gallbladder was palpably enlarged, and the stools were positive for occult blood. The barium-meal examination (Fig. 1) showed extrinsic pressure deformities of the duodenal cap and the upper part of the second part of the duodenum. Glucose tolerance was abnormal and pancreatic function grossly impaired. Laparotomy confirmed the pre-operative diagnosis of a carcinoma of the pancreas. The head of the pancreas was involved particularly. The gallbladder and common bile duct were distended. There were no metastases or enlarged lymph nodes; the liver was not enlarged.

Comment: Radiological deformity of the superior border of the duodenal cap in this elderly patient with obstructive jaundice was due to pressure on the cap by neoplastic enlargement of the head of the pancreas.

Case 5

A 45-year-old barman was admitted with a 9-year history of recurrent pancreatitis, complicated by the staggered onset of diabetes mellitus, obstructive jaundice and diarrhoea during the last 2 years. Examination showed obvious weight loss, deep jaundice and an epigastric mass. The serum-bilirubin level was 13.5 mg. per 100 ml., the alkaline-phosphatase level was 75.6 K-A units, and the CCFT was + 1. A straight X-ray of the abdomen showed extensive calcification of the pancreas, and a barium-meal examination (Fig. 4) revealed gross deformity of the duodenal cap and loop. The superior border of the duodenal cap showed a pressure deformity, and the upper portion of the second part was narrowed. The calcification of the soft tissue in the region of the head *extended lateral to the upper portion of the second part of the duodenum*. The pancreatic-function test, utilizing secretin and pancreozymin stimulation, confirmed gross pancreatic insufficiency, and fat absorption was markedly impaired. Laparotomy revealed an enlarged gallbladder, and a grossly dilated common bile duct coursing over a large pancreatic cyst presenting in the lesser omentum and displacing the duodenum downwards. A cholangiogram showed that the common bile duct was *compressed and obstructed in its supraduodenal portion by the pancreatic cyst*. About two pints of fluid were aspirated from the cyst through the posterior stomach wall, and the cyst was then drained into the stomach. A transduodenal sphincterotomy was also carried out.

Comment: This case illustrated 2 important points which help to clarify the nature of the radiological changes in the duodenum from pancreatic disease. Firstly, calcification in the head of the pancreas, extending lateral to the upper portion of the second part of the duodenum, offers convincing proof of a pancreatic basis of pressure deformities so frequently encountered in patients with disease of the head of the pancreas. Secondly, the operative finding of compression by the pancreatic cyst of the supraduodenal portion of the common bile duct provides evidence for our belief that pressure deformity of the superior aspect of the duodenal cap may be due to upward enlargement of a mass arising in the head of the pancreas.

Case 6

A 32-year-old alcoholic male was admitted with a 5-day history of vomiting, constipation and increasingly severe abdominal pain radiating through to the back and eased by leaning forward. He had suffered a similar attack a few weeks previously. On examination he was obviously ill with epigastric distension and tenderness. The serum-amylase level was 80 Wohlgemuth units per ml., and the ESR was 43 mm. in the 1st hour (Westergren); he had a leucocytosis of 21,600 cells per c.mm. The serum-bilirubin level was 2.1 mg. per 100 ml., the alkaline-phosphatase level was 20.8 K-A units, and the CCFT was negative. The barium-meal examination showed an intrinsically normal duodenal cap, but there was a slight pressure deformity of its superior margin. The second and third parts of the duodenum also showed some pressure deformity without obstruction, but the contour was not altogether fixed, and varied with the degree of filling with barium. Pancreatic calcification was not seen. Laparotomy revealed the presence of a large, bulging pancreatic cyst, containing about 3 pints of brownish fluid, and a smaller bilocular pancreatic cyst. The cysts were drained externally.

Comment: The pressure deformity of the cap and the second and third parts of the duodenum in this patient with relapsing alcoholic pancreatitis suggested enlargement of the head of the pancreas. This was found to be due to pancreatic cysts.

Case 7

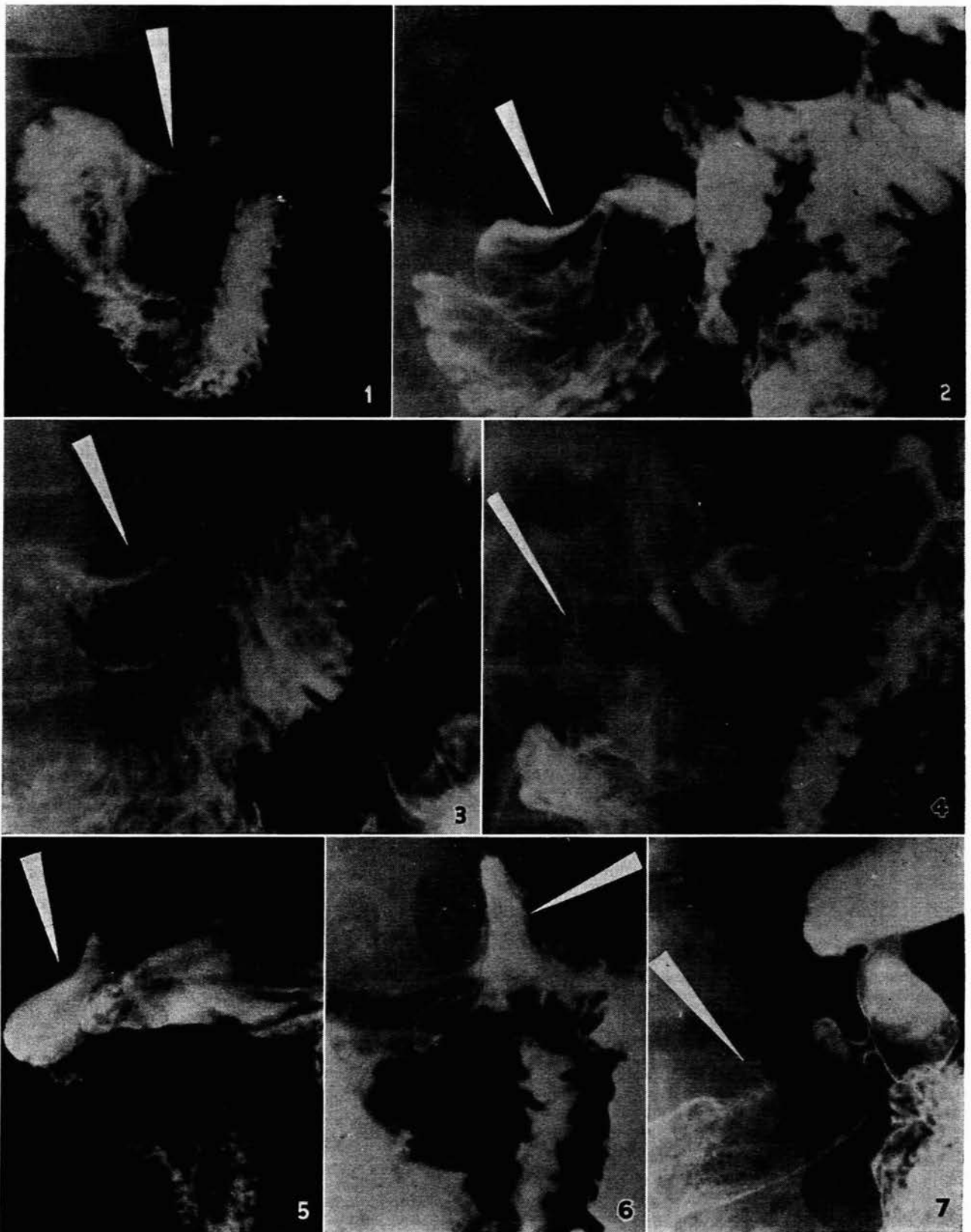
A 41-year-old farmer was admitted with a 1-month history of progressively severe right upper-quadrant pain aggravated by exertion and radiating through to the back. This was associated with anorexia, loss of weight and the passage of dark urine. Waterbrash developed shortly afterwards, and the patient passed several melaena stools in the 10 days before admission. There was no history of alcohol, but he had had an unexplained swelling of the submandibular gland eight years previously. On examination the patient was anaemic, dyspnoeic and slightly febrile. Tenderness and guarding was maximal in the right upper quadrant. The haemoglobin level was 6.5 G. per 100 ml. The serum-bilirubin level was 3.9 mg. per 100 ml., the alkaline-phosphatase level was 21.8 K-A units, and the CCFT was negative.

The barium-meal examination showed an intrinsically normal duodenal cap, but a pressure deformity of the junction of the cap and the first part of the duodenum was evident in all films taken with the patient recumbent. Another pressure deformity was noted in the second part of the duodenum. There was no erosion of mucosal folds. Laparotomy revealed a hard, craggy head of pancreas and an apparently normal body and tail; the gallbladder was distended and the common bile duct dilated. These findings supported the clinical diagnosis of carcinoma of the head of the pancreas, and a Whipple's operation was carried out. On histologic examination of the surgical specimen no evidence of carcinoma was found. The head of the pancreas showed an unusual chronic inflammatory lesion, characterized by a marked degree of productive fibrosis and an unusual type of duct dilatation; there was no evidence of duodenal ulceration.

Comment: Pressure deformity of the cap and the second part of the duodenum in this patient, with a clinical picture suggestive of carcinoma of the head of the pancreas, was due to an unusual chronic inflammatory lesion simulating malignancy in the head of the pancreas.

Case 8

An 82-year-old man was admitted with a 6-week history of painless jaundice associated with the passage of dark urine and, more recently, with pruritus and weight loss. The gallbladder was palpable on examination. The urine contained bile, but no urobilin. The haemoglobin level was 15 G. per 100 ml. and the ESR was 15 mm. in the 1st hour (Westergren). The serum-bilirubin level was 14.3 mg. per 100 ml., the alkaline-phosphatase level was 29.1 K-A units, the CCFT was negative and the prothrombin index was 83%. Glucose tolerance was normal and a pancreatic-function test, utilizing secretin and pancreozymin stimulation, yielded normal results.



The barium-meal examination showed a concave depression on the superior border of the duodenal bulb; the duodenal loop appeared normal (Fig. 5). Laparotomy revealed a very localized neoplastic stricture of the lower end of the common bile duct. The gallbladder was distended, and the common bile duct dilated. Several glands were present in the porta hepatis, but there were no obvious metastases in the liver. A Whipple's operation was carried out. Histology showed an adenocarcinoma of the bile duct with variable differentiation; the lymph glands were reactive and showed no evidence of neoplastic infiltration.

Comment: The radiological findings in this patient with a painless obstructive jaundice were compatible with the diagnosis of a carcinoma of the pancreas or a carcinoma of the common bile duct with involvement of the surrounding lymph glands. A small carcinoma of the common bile duct was found at laparotomy. The radiological distortion of the duodenum, however, was due to glands in the vicinity of the primary lesion; these were reactive and not malignant.

Case 9

A 45-year-old housewife presented with a 7-week history of obstructive jaundice. Fever, vomiting and dull upper-abdominal pain were present at the onset, but the subsequent course was characterized only by painless jaundice, weight loss and pruritus. The liver was smooth and firm, and enlarged to 3 fingers below the costal margin; the gallbladder was not palpable. The white blood count was 12,000 per c.mm., and the ESR was 34 mm. in the 1st hour (Westergren). The serum-bilirubin level was 11.3 mg. per 100 ml., and the alkaline-phosphatase level was 20.9 K-A units. The serum-protein level, CCFT, prothrombin index and serum glutamic oxaloacetic acid transaminase (SGOT) were normal. Glucose tolerance was normal and a pancreatic-function test, utilizing secretin and pancreozymin stimulation, showed no abnormality. The duodenal aspirate did not contain cholesterol crystals or pigments, and no malignant cells were found. Needle biopsy of the liver showed marked plugging of the intralobular bile canaliculi with no evidence of cirrhosis or malignancy. The barium-meal examination showed a concavity of the superior border of the bulb, separated from a second and deeper pressure deformity of the first part of the duodenal loop; the post-apical portion was telescoped between these two concavities (Fig. 6).

A therapeutic trial of steroids for a 2-week period did not influence the degree of jaundice, and the alkaline-phosphatase level actually increased slightly. Laparotomy was carried out 3 weeks after admission to hospital. The operative findings suggested a cholangio-hepatitis. The common bile duct and the hepatic ducts appeared narrow, and an operative cholangiogram showed a narrowing of the intrahepatic ducts as well. Numerous glands were present in the region of the lower end of the common bile duct. The pancreas appeared normal and the gallbladder was not enlarged. The liver was studded with small, pale areas, shown on liver biopsy to be localized areas of marked fatty change. There was active regeneration of liver cells in the central parts of the lobule, distension of the intralobular bile canaliculi, and increased cellularity of the portal tracts. The findings suggested intrahepatic obstruction following a hepatitis, rather than an extrahepatic obstruction.

Comment: Radiological distortion of the duodenum in a

Fig. 1. Barium-meal film showing pressure deformity of duodenal bulb in case 4.

Fig. 2. Barium-meal film showing pressure deformity of duodenal bulb in case 1.

Fig. 3. Barium-meal film showing pressure deformity of duodenal bulb in case 3.

Fig. 4. Barium-meal film showing calcification of pancreas extending lateral to the second part of the duodenum in case 5. Note the shallow depression of duodenal bulb.

Fig. 5. Barium-meal film showing pressure deformity of duodenal bulb in case 8.

Fig. 6. Barium-meal film showing pressure deformities of duodenal cap and upper portion of second part of duodenum in case 9.

Fig. 7. Barium-meal film showing pressure deformity of duodenal cap in case 10.

45-year-old woman was clearly due to reactive glands associated with cholangio-hepatitis.

Case 10

A 59-year-old labourer presented with a 2-month history of anorexia, weight loss and the passage of melaena stools. A mass was palpable in the epigastrium on examination. He had a histamine-fast chlorhydria, and exfoliative gastric cytology was positive for malignant cells. The barium-meal examination showed an hour-glass deformity of the stomach with a filling defect of the fundus; in addition a deformity of the superior border of the duodenal bulb was considered to be suggestive of enlarged lymph nodes in the region (Fig. 7). Laparotomy confirmed the presence of an extensive carcinoma of the stomach with enlarged nodes along the superior border of the duodenal bulb. There was no enlargement of the liver or gallbladder. The lymph nodes showed tumour infiltration.

Comment: This patient, with an extensive carcinoma of the stomach, showed a radiological deformity of the superior border of the duodenal bulb, thought to be due to enlarged lymph nodes; this was confirmed at operation.

The pertinent operative data in the 10 patients are given in Table I. It will be seen that the gallbladder was enlarged in all 4 patients with carcinoma of the head of the pancreas, and that hepatomegaly was present in 3 of these 4 patients. The liver was not enlarged in the 3 patients with non-malignant disease of the pancreas. The gallbladder was distended, but not palpable on clinical exami-

TABLE I. OPERATIVE DATA

Case No.	Diagnosis	Enlargement of					Jaundice
		Pancreas	CBD	GB	Glands	Liver	
1	Ca. pancreas	+	+	+	-	+	+
2	Ca. pancreas	+	+	+	-	+	+
3	Ca. pancreas	+	+	±	-	+	+
4	Ca. pancreas	+	+	+	-	-	+
5	Pancreatic cyst	+	+	±	-	-	+
6	Pancreatic cyst	+	±	±	-	-	+
7	Pancreatic mass	+	+	±	-	-	+
8	Ca. CBD	-	+	+	+r	-	+
9	Cholangiohepatitis	-	-	-	+r	+	+
10	Ca. stomach	-	-	-	+c	-	-

Ca.=carcinoma, CBD=common bile duct, GB=gallbladder, r=reactive, c=carcinomatous.

nation, in these 3 patients. None of the 7 patients with pancreatic disease showed enlarged lymph nodes along the lower end of the common bile duct. On the other hand, all 3 patients with radiological abnormality of the duodenal cap unassociated with pancreatic disease, had marked enlargement of the lymph nodes along the lower end of the bile duct. The gallbladder was palpable in only 1 of the 3 patients, and hepatomegaly was present in another.

DISCUSSION

Anatomy

It is pertinent to this discussion to outline briefly the anatomical relations of the first part of the duodenum (Fig. 8). Since the deformity illustrated in the case histories affects the duodenal bulb in particular, it is important to differentiate the relations of this area from that segment of the first part of the duodenum comprising the superior flexure. The mass of the liver and the head of the pancreas form superior and inferior relations, respectively, to the whole of the first part of the duodenum. Brown and

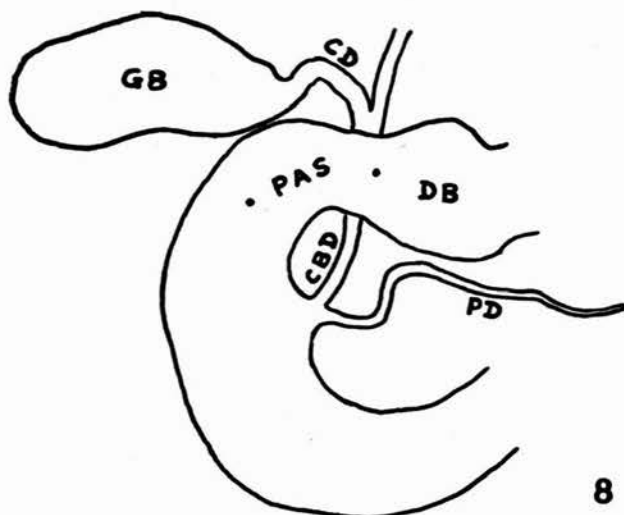


Fig. 8. Diagrammatic representation of relationship of common bile duct (CBD) to duodenal bulb (DB) and postapical segment (PAS). The postapical segment is the area between the 2 dots. PD=pancreatic duct, CD=cystic duct, and GB=gallbladder.

Harper⁸ stated that the superior flexure is surrounded by the neck of the gallbladder on the right side, the cystic and hepatic ducts above and the common duct on the left. These structures therefore encircle this fixed portion of the duodenum and, if distended, will tend to produce pressure deformities in this site.

Although variations in the retroduodenal course of the common duct do occur,⁹ it is evident that it usually assumes a posterior relationship to the apical part of the more flexible duodenal cap. Furthermore, since the common duct tends to enter the substance of the pancreas behind the cap, it is frequently only at the superior aspect of the duodenal cap that these structures lie in direct apposition.

The extensive network of lymphatic channels draining structures in the area terminates in lymph glands closely associated with the common bile duct, cystic duct and the neck of the gallbladder. Thus, any increase in the size of the structures mentioned above may distort or compress the first part of the duodenum; the superior flexure may be deformed by pressure from the neck of the gallbladder or a dilated cystic or common duct, but it is probable that causes other than these are responsible for pressure defects of the superior aspect of the duodenal cap. The operative findings in the cases reported in the present paper would tend to support this concept.

The deformity of the duodenal cap in each of our 10 patients was associated with one or more of the following abnormalities: (1) enlargement of the gallbladder, (2) diffuse hepatomegaly, (3) a dilated common bile duct, (4) enlargement of the head of the pancreas, and (5) adenopathy in the region of the common bile duct.

Gallbladder

The frequency of jaundice in lesions in the region of the head of the pancreas may prompt the inference that pressure by an enlarged gallbladder or cystic duct is responsible for all such deformities of the duodenal bulb. We have attempted to show that this is unlikely on anatomical grounds alone. This is supported by the findings of

Khilnani *et al.*⁹ in gallbladder carcinoma, and Brown and Harper⁸ in cholelithiasis. Both authors stressed that it is the superior border of the postapical portion of the first part and not the duodenal bulb that is affected by these lesions. The fact that the gallbladder was not enlarged in 3 of our patients clearly excludes the gallbladder as the cause of the deformity of the duodenal bulb in these cases, and may be construed as further evidence against the possible view that such enlargement was responsible for the deformity in the remaining 7 patients. At operation, the distended gallbladder appeared to lie lateral to the duodenal cap and was not considered to be the cause of the radiological deformity.

Despite the above, there is little doubt that a normal gallbladder may occasionally compress the superior border of the duodenal bulb rather than the postapical portion of the first part of the duodenum, in the erect position. This is illustrated in Fig. 9. It should be noted, however,



Fig. 9. Radiograph, showing compression of superior border of the duodenal bulb, taken during combined cholecystogram and barium study, in a patient with functional dyspepsia.

that the radiographs of all the cases presented in this paper were carried out in the supine right anterior oblique and not the erect position.

Liver

The close relationship between the liver and the superior border of the duodenal bulb suggests that radiological deformity of the bulb may be caused by enlargement of the liver. Brust and Conlon¹⁰ have recorded a pressure deformity of this type in a patient with a nodular liver caused by a primary hepatoma. It is of interest, however, that this radiological deformity has not been attributed to diffuse hepatomegaly. The liver was not enlarged in 6 of our patients, and the enlargement in the remaining 4 was diffuse and not nodular. These findings suggest that hepatomegaly, where present, did not contribute appreciably

ciably to the deformity of the duodenal cap present in patients with pancreatic or other lesions. Olsen *et al.*¹¹ have recently drawn attention to a shallow compression deformity of the superior aspect of the duodenal bulb caused by a dilated posterior superior pancreatico-duodenal vein in patients with extrahepatic obstruction. This particular deformity was not observed in any of our cases.

Pancreas

Pancreatic masses were present in 7 of the 10 patients in the series. Obstructive jaundice with dilatation of the common bile duct and distension of the gallbladder was present in all 7 patients, 3 of whom showed, in addition, a diffuse hepatomegaly. Pressure deformities of the lesser curvature of the antrum of the stomach are well known¹² in large pancreatic neoplasms of the head and neck where the major portion of the growth extends superiorly, and the elegant schematic drawings of Salik¹³ indicate that the superior border of the duodenal bulb may be distorted by a lesion of the head of the pancreas. With a more localized superior extension the pressure deformity may conceivably involve only the duodenal bulb and spare the antrum or pyloric region.

Increasing awareness of this possibility resulted in a careful inspection of this site in the cases in this series, and in no less than 5 of the 7 cases the surgeon noted that compression of the duodenal bulb could be attributed to the pancreatic mass *per se*. On the other hand, the pancreatic mass did not seem to be responsible for the radiological deformity of the duodenal bulb in 2 of the 7 patients with such lesions. It thus appeared that the dilated common duct or, possibly, the enlarged gallbladder, might have been responsible for the lesion in these 2 patients.

Common Bile Duct

It might be argued that the dilated common duct and not the pancreatic mass caused the concave deformity of the duodenal cap in all the patients with pancreatic masses, since all these patients had a widely dilated common duct. While we believe that the common duct may be held responsible in a number of cases, it is of interest that Brown and Harper⁸ and Salik¹³ emphasized that it is the post-apical or post-bulbar segment of the first part of the duodenum which becomes narrowed, elongated and distorted in intraluminal distension of the common bile duct. Both authors showed radiographs demonstrating a concave duodenal cap deformity attributable to common bile duct pressure, but in these cases the primary pathological change proved to be bile duct carcinoma.

Convincing evidence that the dilated common duct could not be incriminated in all our cases is illustrated in case 5, since a pancreatic cyst, extending superiorly, not only compressed the superior lesser curve of the duodenal cap, but also displaced the common duct posteriorly, away from any possible contact with the duodenal cap. Whether the common duct will produce a deformity of the duodenal cap will depend in part on its anatomical relationship to this area, but it would appear that the post-bulbar portion of the duodenum will bear the brunt of such compression.

Lymph Nodes

That lymph-node enlargement may produce a concave impression of the lesser curve of the bulb is shown by

Poppel.⁴ Indeed, in 1 of the 2 radiographs in his book demonstrating this deformity, the duodenal impression was due to retroperitoneal nodal enlargement at the retropancreatic level, involving the midline pancreatic nodes and extending towards the right; in the other it was due to an ectopic pancreatic nodule.

The radiological deformity in 2 of our patients was clearly due to lymph-node enlargement. It was due to malignant lymphadenopathy in one patient (case 10) with a carcinoma of the stomach, and to benign inflammatory nodes associated with benign cholangiohepatitis in the other (case 9). Reactive glands were considered to be the cause of the deformity in a third patient (case 8) with an extremely small carcinomatous stricture of the retroduodenal portion of the common bile duct, but the widely dilated duct above this stricture may have contributed to the deformity. It is obvious that the lymph nodes would be situated along the superior border of the duodenal cap in order to produce this deformity, and laparotomy has indicated that they do in fact lie parallel and in direct apposition to the retroduodenal segment of the common duct. Our findings suggest that these periductal nodes may be enlarged in pancreatic, common bile duct and, on rare occasions, gastric lesions.

SUMMARY

A concave pressure deformity of the superior part of the duodenal bulb during barium studies of the upper gastrointestinal tract is described, and special reference is made to its causation. The technique for demonstrating the deformity is presented.

We have attempted to show that this radiological sign depends primarily on direct extension of pancreatic masses in a superior direction and to periductal lymph-node enlargement. A dilated common duct or gallbladder may occasionally produce this deformity, but this will depend on the particular anatomical relationship of the common duct to the duodenal bulb in a given individual.

Although pressure by a normal gallbladder on the duodenal bulb may rarely produce this sign in radiographs taken in the erect position, its presence in supine and right anterior oblique views should suggest the possibility of neighbourhood disease. The sign is most frequently due to pancreatic disease, usually pancreatic carcinoma, but it may be found in patients with inflammatory enlargement of the pancreas or regional lymph glands.

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