Case 85 A jaundiced and very ill patient

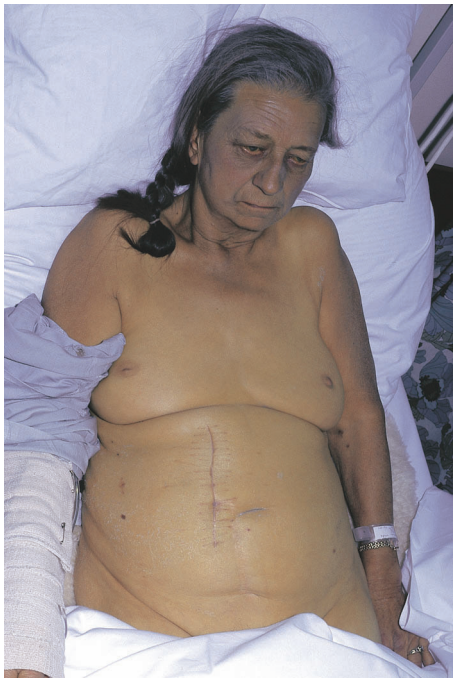

Figure 85.1

A housewife aged 64 years had been admitted to the surgical unit 6 months previously with a few weeks' history of vomiting after meals, anorexia and profound weight loss. The vomit contained pretty well unchanged food that she had just taken. There was almost continuous upper abdominal discomfort, rather than pain, and nothing seemed to relieve this. On her admission at that time, the notes stated that she was pale and looked unwell.

There was a rather ill defined tender mass in the upper abdomen; her haemoglobin was 93 g/L and liver function tests were normal. An abdominal ultrasound showed several small lesions in the right lobe of the liver. An urgent gastroscopy was performed, which demonstrated an ulcerating tumour at the distal end of the stomach, biopsy of which showed a poorly differentiated adenocarcinoma. After a blood transfusion, she underwent a laparotomy. There was an obstructing mass occupying the antrum and pyloric end of the stomach, which was adherent posteriorly to the pancreas. Large firm lymph nodes could be felt along both curvatures of the stomach. Several hard nodules, up to 2 cm in diameter, could be felt in both the right and left lobes of the liver. Frozen section of one of the nodes and of a biopsy of a liver nodule showed adenocarcinoma. A palliative anterior gastro-jejunostomy was carried out to bypass the obstruction.

She had quite a stormy postoperative recovery – pulmonary collapse, treated with vigorous physiotherapy; bilateral deep vein thrombosis, in spite of prophylactic low molecular weight heparin and thromboembolic deterrent (TED) stockings, which required therapeutic subcutaneous heparin; and delayed opening of the gastro-jejunostomy stoma, which necessitated over a week of nasogastric aspiration and intravenous feeding. Eventually, 4 weeks after her admission, she was able to leave hospital and to tolerate a light diet.

She has now been readmitted as an emergency in a very sorry state, as shown in Fig. 85.1. She feels very weak and her husband had noted her colour change a few days before. She has completely lost her appetite and noticed that her stools have gone white and her urine a dark brown colour.

On admission, examination, apart from the obvious jaundice, showed that the surgical scar was well healed but that the abdomen was swollen, there was an obvious hepatomegaly, three to four finger-breadths below the right costal margin, and clinical evidence of marked ascites.

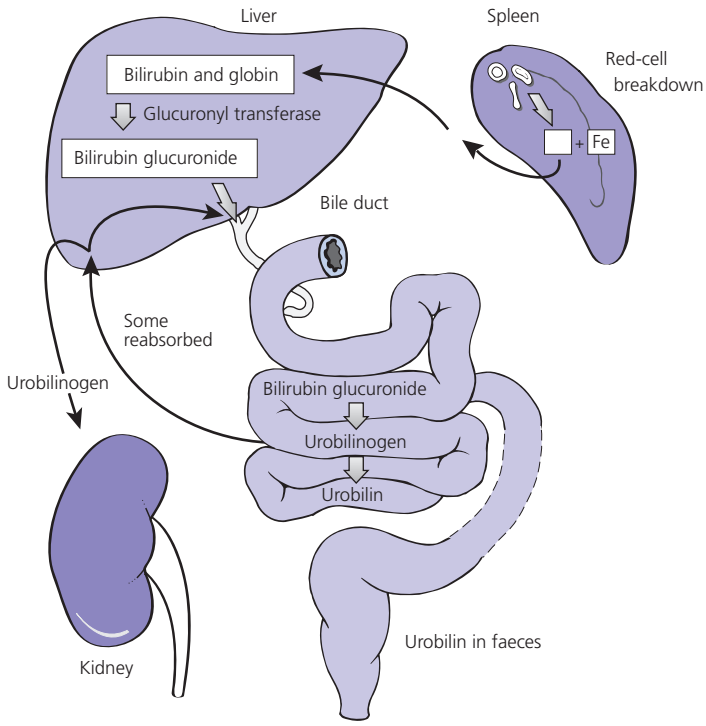

Figure 85.2 The metabolism of bilirubin.

What is the biochemical explanation of this patient's colour change?

Jaundice is due to staining of the tissues of the body with bilirubin (Fig. 85.2). This is detected clinically most easily in the conjunctivae. The normal serum bilirubin is under 17 μmol/L and jaundice becomes clinically detectable when the level rises to over 35 μmol/L.

Can you classify the three large subgroups of the causes of jaundice?

- *Prehepatic*: Due to excessive breakdown of haemoglobin, for example in haemolytic diseases and incompatible blood transfusion.
- *Hepatic*: Due to liver disease, for example hepatitis, cirrhosis or extensive destruction from tumour deposits – as in this case.
- *Posthepatic*: Due to bile duct obstruction. This can be further subdivided into:
  - Obstruction within the lumen of the bile duct, for example from calculi.
- Obstruction in the wall, e.g. congenital atresia, post-traumatic stricture or bile duct tumour.
- External compression of the ducts, e.g. pancreatic tumour or pancreatitis.

How do you make differential diagnoses of the cause of jaundice in a patient?

Of course, as in any diagnosis, this is made from a detailed history, careful clinical examination and then laboratory investigations. Table 85.1 lists the more important of the laboratory findings.

Which of the three aetiological groups usually only produces a tinge of jaundice?

Prehepatic jaundice, where the serum bilirubin is seldom raised above 100 μmol/L, and the colour change may often only be detected by careful inspection of the conjunctivae. An example of this is shown in Fig. 85.3, a woman with haemolytic jaundice. Since the bile ducts are not obstructed, large amounts of excess bilirubin are excreted into the gut.

Table 85.1 Laboratory tests useful in the diagnosis of the cause of jaundice.

Test	Prehepatic	Hepatic	Obstructive
Urine	Urobilinogen	Urobilinogen	No urobilinogen. Bilirubin present
Serum bilirubin	Unconjugated bilirubin	Conjugated and unconjugated	Conjugated bilirubin
ALT and AST	Normal	Raised	Normal or moderately raised
ALP	Normal	Normal or moderately raised	Raised
Blood glucose	Normal	Low if liver failure	Sometimes raised if pancreatic tumour
Reticulocyte count	Raised in haemolysis	Normal	Normal
Haptoglobins	Low due to haemolysis	Normal	Normal
Prothrombin time	Normal	Prolonged due to poor synthetic function	Prolonged due to vitamin K malabsorption; corrects with vitamin K
Ultrasound	Normal	May be abnormal liver texture, e.g. dilated bile ducts, cirrhosis	

ALT, alanine transaminase; AST aspartate transaminase; ALP, alkaline phosphatase.

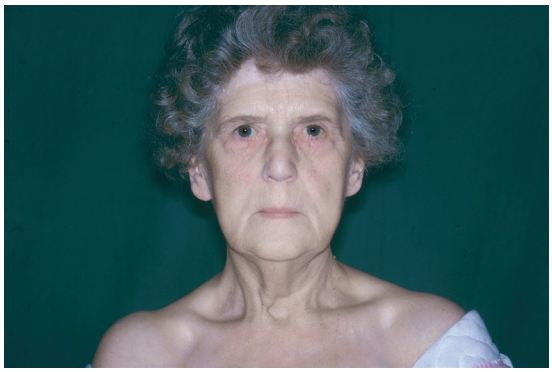

Figure 85.3 Haemolytic jaundice.

When our patient with jaundice and liver secondaries had her blood tests done, she was found to have a prolonged prothrombin time. Why was this?

Vitamin K, necessary for prothrombin synthesis in the liver, is fat-soluble and requires bile salts for its absorption from the gut. In addition, the extensive liver damage from her liver deposits may prevent the hepatic synthesis of prothrombin.

The important message from this is that jaundiced patients who are to undergo surgery require preoperative vitamin K by injection.

> *This poor lady was transferred a week later to the local hospice and died, fortunately peacefully, after 2 weeks. At the request both of the patient and her husband, an autopsy was performed because they both 'wished to help medical science and the doctors'. This forms the subject of Case 86.*