On May 11, 1939, a radical amputation of the right breast was performed by Rodman's method at the Dewsbury and District Infirmary. The patient went home 16 days after the operation, by which time the wound had healed.

Pathological Report (5) (May 21, 1939; Prof. M. J. Stewart).—"The nipple is completely retracted and slightly crusted on the surface. The substance of the breast is almost completely replaced by carcinomatous tissue, the main mass of which, in the zone beneath the nipple, measures 2½ in. (5.7 cm.) from above downwards and about 1½ in. (3.2 cm.) in depth. But the tumour tissue also spreads widely into the more remote portions of the breast and includes a nodule in the adipose tissue close to the pectoralis major muscle. There is evidence of cancerous involvement of axillary glands. Histologically the growth is a spheroidal-cell carcinoma, glandular in places and obviously very invasive. It has extended widely throughout the breast and has invaded the skin of the retracted nipple, with wide extension into the cutis vera and subcutaneous tissues of the areola, including the unstriped muscles of the nipple. Lymphatic permeation and invasion of adipose tissue are notable features. The nodule in the adipose tissue close to the pectoralis major muscle proves to be cancerously infiltrated fibrous tissue. Elsewhere in the depths of the breast there are several small cancerously invaded lymph glands and much lymphatic permeation, though without evidence of involvement of the pectoral muscle itself. The ultimate prognosis must be regarded as extremely bad."

In June, 1939, a course of deep x-ray therapy was given first to the right breast region (200 kV 1 mm. Cu 2000 r in 20 days), and further irradiation was given to the pelvis (200 kV 1 mm. Cu 2000 r in two weeks) and to the left breast (200 kV 1 mm. Cu 2000 r in two weeks). On Oct. 23, 1939, the patient was going out to work again and felt "perfectly well."

Comment

Whilst abroad I received a letter from her sister-in-law telling me that after a short illness my patient had died in July, 1941. She had insisted upon going to work to within a fortnight of her death.

The patient lived just over eight years from the time of removal of the left breast for a frankly malignant type of cancer. She showed remarkable resistance to the disease even after secondaries had appeared. It is also interesting to note that a second cancer, which was considered to be another primary, started in the opposite breast two years after bilateral oophorectomy had been performed.

Metastatic carcinoma from the breast as a cause of perforation of the appendix and general peritonitis must be extremely rare. No record of a similar case has been found in the literature.

Summary

Some of the natural defence mechanisms against malignant disease are discussed. Certain individuals seem to have exceptional powers of resistance to cancer.

A case is described in which a carcinoma of the left breast was removed before the menopause, and a secondary deposit involving the appendix led to perforation and general peritonitis 3 years and 8 months later. The appendix and both ovaries were removed. A year later a recurrence in the original breast scar was excised. The following year a carcinoma, thought to be a second primary, appeared in the right breast, and a radical amputation was performed. The patient died just over eight years after the original operation upon the left breast.

I wish to express my grateful thanks to Prof. M. J. Stewart for his kind help and advice.

REFERENCES

- Atkins, H. J. B. (1939). *British Medical Journal*, **1**, 866.
 Bashford, E. F., Murray, J. A., and Cramer, W. (1907). *Proc. roy. Soc.*, **B**, **79**, 164.
 Bloodgood, J. C. (1932). *Amer. J. Cancer*, **16**, 103.
 Pearce Gould, A. (1910). *Lancet*, **2**, 1665.
 Schmidt, M. B. (1903). *Die Verbreitungswege der Karzinoma*, Jena.
 Teacher, J. H. (1908). *J. Path. Bact.*, **12**, 487.
 Warburg, O. (1925). *Biochem. Z.*, **160**, 307.
 — (1930). *Metabolism of Tumours*, p. 199, London.
 — Wind, F., and Negelein, E. (1925-7). *J. gen. Physiol.*, **8**, 519.

Nurses and midwives of the Commonwealth and Empire have already raised more than £17,000 as a memorial to their colleagues who fell in the war of 1939-45, and the Queen has shown her appreciation of their effort by consenting to become the Patron of the fund to which they are contributing—the British Empire Nurses War Memorial Fund—and the Princess Royal has consented to become Vice-Patron. The address of the fund is Dorset House, Stamford Street, London, S.E.1.

BILIARY PERITONITIS WITHOUT DEMONSTRABLE PERFORATION

BY

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Biliary peritonitis results from the escape of bile into the peritoneal cavity, which excites a marked peritoneal reaction, whether the liberated bile be infected or sterile. Three main varieties of the condition have been described according to the underlying pathology. Injuries to the biliary tract and perforations of the gall-bladder in acute cholecystitis account for the large majority of cases. A number of cases have been recorded in which no perforation could be found. This so-called "perforationless biliary peritonitis" is a rare condition which was first described in 1905 by Maurice Richardson. No general interest in this pathological entity was evoked, however, until Clairmont and von Haberer published a paper on the subject in 1910.

Symptomatology and Pathology

The clinical picture has shown wide variations, and cases have been described simulating perforation of a peptic ulcer (Illingworth and Dick, 1941), with a less sudden development resembling peritonitis resulting from perforation of the appendix (Buchanan, 1918), or of still more gradual nature, with increasing abdominal distension, vomiting, and a rising pulse rate (Rolleston, 1938).

In the cases with a relatively acute onset, which constitute by far the greatest number, remission of symptoms after the first acute attack has been the one almost constant feature (Cope, 1925-6). This can be explained by the fact that sterile human bile, differing from the bile of experimental animals in its small content of the toxic salt sodium taurocholate, is only slightly irritant after the initial concentrated effusion has been diluted by peritoneal exudate. Further, cases are on record where over 30 pints (17 litres) of bile, presumably diluted by ascitic fluid, have been aspirated from the peritoneal cavity over a period of six weeks.

The pathology would appear to be almost as variable as the symptomatology. While a grossly discoloured and almost necrotic common bile duct associated with a tense red gall-bladder has been described (Buchanan, 1918), the biliary system has also been found apparently intact (Rolleston, 1938). Thirteen out of sixteen cases reviewed by Buchanan (1918) showed inflammatory or carcinomatous lesions of the biliary tract or stones. Of the remaining three, one gave a history of trauma and one was thought to be due to typhoid ulceration.

Pohlmann (1939) describes a case with acute inflammation superimposed upon a chronic cholecystitis. Although the cystic duct was blocked and the tense gall-bladder was covered with fibrinous exudate, no perforation could be found to account for the biliary effusion.

McLaughlin (1942) reports eight cases of biliary peritonitis, but all except one of these fall into a different category, since they were a sequel to cholecystectomy. He suggests that in many such cases the leakage of bile is accounted for by accessory hepatic ducts entering the gall-bladder direct from the liver. Others are almost certainly due to duct perforation resulting from increased pressure in the biliary tree caused by overlooked stones in the common duct. The only case that was not post-operative showed at necropsy a gangrenous gall-bladder with stones in both the gall-bladder and the common duct. No perforation could be demonstrated. Four of the post-operative cases were fatal, and in three on which necropsy was performed stones were found in the common duct. Perforations of the cystic duct stump were found in two.

Smith (1926) reviewed 12 fatal cases, all with rupture of intra-hepatic ducts and with a history of calculus, cholecystitis, and intermittent symptoms of common-duct obstruction.

Retroperitoneal extravasation of bile was a characteristic feature in two cases described by Cope (1925-6). In one of these the overlying peritoneum appeared to "weep" bile, but no perforation could be found.

It has been suggested that post-peritoneal rupture or perforation of the common or hepatic ducts results from trauma or ulceration and leads to biliary effusion into the retroperitoneal tissues with subsequent rupture into the peritoneal cavity at a point more or less removed from the site of the perforated bile passage (Buchanan, 1918). This would seem a possible explanation of the two cases with retroperitoneal effusion referred to above.

Glands of Luschka which have penetrated abnormally deeply through the muscular coat of the gall-bladder have been cited as a possible path by which bile may escape, but in view of the very strong safeguard the overlying peritoneum presents against intraperitoneal extravasation this would appear improbable. German authors (Kaufmann, 1929) have blamed percolation and filtration of bile through the wall of the gall-bladder either as a form of diapedesis or as a result of digestion of the wall by pancreatic trypsin.

Ruptures of the intrahepatic bile canal have been found at biopsy, and have all been associated with obstruction to the common duct by stone or carcinoma (Buchanan, 1918). Perforations of a subserous bile duct on the liver surface have also been described.

McLaughlin (1942) remarks that in the majority of recorded cases a small perforation could be demonstrated, though often only by serial sections at the site. Because of their small size these would leak only when the intraperitoneal pressure was increased, closing spontaneously when the pressure was relieved. The natural tendency to spontaneous closure which these organs show is well known, and is attributed in part to the low pressure of bile excretion and the readiness with which adhesions to neighbouring viscera form.

Burckhardt (1923) also believes that there must always be a perforation, either not found or already healed. If this theory be correct at least a temporary obstruction to one of the bile passages must be assumed to promote the escape of considerable quantities of bile through a small opening.

Case Report

A tall well-built man aged 26 was admitted to hospital on May 16, 1945. Five days previously, when walking down the street, he felt a sudden pain in the upper abdomen which lasted probably less than a minute, but caused him to stand still during that time. Two days later he noticed that his urine was dark in colour and he began to suffer from nausea, lack of appetite, and lethargy.

On clinical examination nothing abnormal was detected beyond a yellow tinge in the sclerotics and slight tenderness in the right hypochondrium. Bile pigments were demonstrated in the urine. The leucocyte count was low (5,600), but the differential count showed no abnormality. On the day after admission jaundice was discernible. The van den Bergh reaction was immediate direct. The jaundice continued to deepen, and the patient vomited frequently. He was afebrile throughout and at no time was a raised pulse rate recorded. The patient noticed a change in the colour of his stools, which struck him as being abnormally light in colour, but this information was not obtained till later.

Twelve days after admission, in the early morning, he developed severe pain of very sudden onset, which was first felt between his shoulders posteriorly. He was returning from the lavatory when the pain began, and he found it difficult to straighten his back. A few moments later he felt pain in his abdomen. On examination two hours later he showed the clinical picture of acute general peritonitis, with abolition of abdominal respiration and generalized board-like rigidity. Tenderness was maximal in the right hypochondrium, and the flanks were dull on percussion. Nothing abnormal was detected in the chest.

A diagnosis of acute general peritonitis, possibly due to rupture of the gall-bladder or bile ducts, was made, and it was decided to perform laparotomy immediately. A drip blood transfusion was begun before starting the operation.

Operation.—Pentothal was given through the intravenous tube, supplemented by gas and oxygen. The abdomen was explored through a right upper paramedian incision. The peritoneal cavity was filled with a golden-brown fluid resembling bile, and amounting to about two pints (1.14 litres). In the absence of a sucker this was mopped out with towels. The upper abdominal organs were carefully and systematically examined. The liver appeared slightly congested but showed no gross pathology. The gall-bladder was thickened, suggestive of some chronic inflammation, but the peritoneal covering was healthy and intact, and no stones were palpable. The contained bile was not under tension and emptied easily on pressure. No perforation or other lesion could be found in the

cystic duct, the common bile duct, or the common hepatic duct. Both stomach and duodenum were normal. The pancreas was palpated and visually inspected through the lesser sac, and no lesion was found. The spleen was not enlarged. Inflammation of the bowel surface and other signs of acute infective peritonitis were entirely absent.

Some of the peritoneal fluid was collected in a sterile test-tube and was observed to coagulate almost immediately. A pelvic drain was inserted through a suprapubic stab wound. The peritoneum and rectus sheath were approximated with No. 2 twenty-day catgut. Cotton thread was used for ligatures and silkworm-gut for tension sutures and skin.

The drip blood transfusion was continued until two pints (1.14 l.) was given, and this was followed by intravenous saline to ensure adequate fluid and glucose intake until the patient was able to swallow fluids without vomiting.

After-history.—On the day after operation the intravenous drip was discontinued. There was profuse discharge of bile-stained fluid from the suprapubic tube. The peritoneal fluid was reported on as containing bile pigments. (It was sterile after seven days' culture.) The patient's general condition was poor, and his pulse varied from 110 to 130, and his temperature from 101 to 103° F. (38.3 to 39.4° C.). Jaundice was unchanged and bile pigments were still present in the urine. A bad taste in the mouth was a prominent symptom. The next day, much of the oral intake being vomited, an intravenous drip was restarted. The vomit was deeply bile-stained and was strongly positive to Hay's test. The leucocyte count had risen to 12,400. On the fourth day jaundice was noticeably disappearing, and his condition was much improved. The urine was bile-free. The suprapubic tube was removed. The following day the drip was considered no longer necessary. On the seventh day the sutures, which were cutting through the skin, were removed, and the skin edges appeared firmly united. Shortly after, the patient "cleared his throat" and the wound fell apart and bowel protruded. Under pentothal anaesthesia the prolapsed bowel was replaced and the wound edges approximated with through-and-through sutures of stout silk. At this second operation there was still a small quantity of bile-stained peritoneal effusion. There was no sign of the catgut used to close the wound a week previously, nor was there any evidence of inflammation of the bowel wall. This complication resulted in a deterioration in the general condition, and intravenous glucose-saline was restarted, raising the systolic blood pressure from 80 to 120 mm. in the three hours following operation.

With the exception of a collapse of the base of the right lung the patient made steady progress, though there was considerable inflammatory reaction round the wound. A good formed motion of normal colour was passed on the eleventh day after the first operation. Three days later jaundice had almost disappeared.

Bile staining persisted in the discharge from the abdominal wound, and did not entirely disappear until four weeks after the abdominal crisis. Two weeks later a cholecystogram was done and showed deficient filling of the gall-bladder with poor concentration and delay in emptying. No calculi were seen.

Comments

The peritoneal effusion in this case was proved to be bile, from which no organisms could be cultured. The maximum intensity of jaundice coincided approximately with the abdominal crisis, after which both the jaundice and the bile content of the urine began to diminish. The pale colour of his stools attracted the patient's attention for several days before operation, but it was not possible to ascertain whether they were entirely free of bile pigment. The presence of bile in the post-operative vomit excludes the possibility of common-duct obstruction at this stage. A diagnosis of either infective hepatitis or obstructive jaundice is compatible with the history and findings in this case, at least until the advent of an abdominal crisis. Epigastric pain is apparently a not uncommon symptom in the former condition, which was the accepted pre-operative diagnosis. If this was a case of infective hepatitis, however, it is extremely difficult to postulate how the biliary effusion may have occurred. Obstructive jaundice is therefore considered a more likely explanation, being consistent with the symptomatology while at the same time offering a possible explanation of the biliary effusion, and one which is in keeping with the findings of at least a considerable proportion of the reported cases.

It is suggested that at the first attack of pain, before the appearance of jaundice, a small stone passed down from the gall-bladder, which from operative findings and a cholecystogram is known to be pathological, and lodged at or near the ampulla of Vater. This would result in jaundice and increased obstruction in the biliary system. A small perforation possibly occurred in the lower part of the common bile duct, which does

not lend itself to visual inspection. This may have been temporarily plugged with the stone, permitting the escape of bile as soon as it left its site of impaction.

The effusion would in this case be first of all retroperitoneal, accounting for the fact that the initial pain during the abdominal crisis was clearly located to the back. The intraperitoneal effusion would follow a secondary perforation through the peritoneum, as suggested by Buchanan (1918). It will be remembered that Cope (1925-6) described the peritoneum as appearing to "weep" bile.

The obstruction to the biliary system was possibly not released until after the initial escape of bile. After the relief of tension in the common duct the perforation would tend to close spontaneously.

REFERENCES

- Buchanan, J. J. (1918). *Surg. Gynec. Obstet.*, **26**, 303.
 Burckhardt, H. (1923). *Beitr. klin. Chir.*, **128**, 209.
 Clairmont, P., and von Haberer, H. (1910). *Mitt. Grenzgeb. Med. Chir.*, **22**, 154.
 Cope, Z. (1925-6). *Brit. J. Surg.*, **13**, 120.
 Illingworth, C. F. W., and Dick, B. M. (1941). *Surgical Pathology*, 4th ed., London.
 Kauffmann, E. (1929). *Pathology for Students and Practitioners*, vol. 2, London.
 McLaughlin, C. W. (1942). *Ann. Surg.*, **115**, 240.
 Pohlmann, H. F. (1939). *Amer. J. Surg.*, **48**, 400.
 Richardson, M. H. (1905). *Trans. Amer. surg. Ass.*, **23**, 217.
 Rolleston, Sir Humphry (Ed.) (1938). *Brit. Ency. med. Pract.*, **9**, 540.
 Smith, W. (1926). *Ann. Surg.*, **83**, 55.

EPIDEMIC PLEURITIC PAIN

A REVIEW OF 17 CASES IN A MILITARY CAMP, INDIA COMMAND

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During the period December, 1945, to January, 1946, a series of cases presenting themselves as pleurisy occurred in an Indian military camp for British personnel. The clinical picture led us to believe that the usual textbook accounts of dry pleurisy, which emphasize the tuberculous aspect, are unbalanced.

Symptomatology

The cases occurred in young troops arriving in India from England, and were seen in the space of six weeks. Coryza and pharyngitis were common owing to inhalation of dust and to change in climate. Pain in the chest was present in all cases with the exception of two. The men were not ill, and when asked if they would have continued at work in civilian life the majority answered "Yes." More than half reported sick with both pain and upper respiratory tract infection, while others gave a history of the latter during the previous three weeks. The pain was pleuritic in type, mostly localized to the lower thoracic region in the axillary line, and in two cases was felt in the second left intercostal space. It lasted from two to twenty-one days, and the majority of patients had a cough with slight sputum. In some cases there was local tenderness on pressure over the painful area. A pleural rub was heard in three cases, and six showed signs of a parenchymal lesion in the lung. These signs were fleeting, and for the most part consisted of localized crepitations at the site of the pain. They were heard in late inspiration and early expiration, and in one case disappeared after the first few deep breaths. The crepitations persisted after coughing.

It must be emphasized that the physical signs in the chest were transitory. One case presented with pain in the chest and had no physical signs until the sixth day, when crepitations were heard over the site of the pain. Another case had symptoms of pleuritic pain without physical signs over a period of fourteen days; physical signs suggesting a pleural effusion then occurred, but skiagrams showed a parenchymatous lesion of the right lobe—a pneumonitis.

Only four cases were pyrexial, the temperatures ranging from 100.6° F. (38.1° C.) to 103.8° F. (39.9° C.). Of cases transferred to hospital one developed a lung abscess, while another, de-

scribed above, showed radiological evidence of a pneumonitis. On being screened six others showed no radiological lesion whatsoever. Three patients had the blood sedimentation rate estimated: one was normal, one was 17 mm. (after an hour), and a third showed the series 39-20-9 mm. (after an hour) over a period of three weeks after admission. In no case was the leucocyte count abnormal.

A Typical Case

On Jan. 10, 1946, a youth aged 19 had a brief illness consisting of headache, sore throat, and rhinitis, with a temperature of 103° F. (39.4° C.). Next day there was no pyrexia, and the symptoms resolved. On the fourth day the temperature rose to 102° F. (38.9° C.) without any symptoms other than sweating. Blood slides were negative for malaria parasites. There was no rash or any further pyrexia. This was not a case of dengue fever, but an upper respiratory tract infection.

On Jan. 21 he developed pleuritic pain in the left second intercostal space, with a temperature of 100.6° F. (38.1° C.). There was a slight cough, but no sputum. At the site of the pain crepitations were heard for the first few breaths on deep inspiration. On Jan. 22 the pleuritic pain was less severe, and persistent crepitations were heard in the same position. On Jan. 23 the patient said the pain was felt only in the early morning. Temperature, 99.4° F. (37.4° C.). No signs were present in the chest. There was no further pyrexia and no further pain. The patient is quite well to-day.

Discussion

Access to literature was limited owing to difficulties of travel, and on account of Service conditions observations on the diseases are largely clinical. The cases sent to hospital were followed up there, but once the benign nature of the condition was realized they were treated in bed in the camp hospital for a few days.

The differential diagnosis lies between: (1) a pleurisy of tuberculous origin; (2) epidemic myalgia (Bornholm disease); (3) atypical virus pneumonia; (4) a benign condition such as that described by Williamson (1924) under the name of "epidemic pleurisy."

There are many reasons for not considering the condition a tuberculous process. In no case was there a suggestive personal or family history. With the exception of two, both now well, the condition was benign and recovery rapid; most of these patients were off duty for less than two weeks, and none developed a pleural effusion. Another point against tuberculosis is that a number of cases occurred in a short time among healthy troops—i.e., the condition was epidemic.

Epidemic myalgia, as first described by Daac in 1874, shows points of similarity, but there are differentiating features. Our chief reference is the Cincinnati outbreak of eleven cases. Here a syndrome was described in which the pain in the chest was severe and occurred in paroxysms, sometimes numbering five in all, with periods of quiescence during which the patient was comfortable. The quiescent periods lasted 24 to 30 hours. In our cases the pain was not usually intense, and was of definite pleuritic type; there were no intermissions. Fever of low grade is common to both syndromes. In the Cincinnati outbreak there were no physical signs, and this is our criterion for differentiation; physical signs were present in the majority of our cases, and were fleeting enough to make us think that we should have heard them in more if we had had the opportunity for further observation. We must agree that our cases seem to have taken an epidemic form, since they all occurred in a space of six weeks; and there were other cases in neighbouring camps, all showing the same benign process, migrating to the same hospital. Also, the end of the epidemic was coincident with a considerable fall in the numbers passing through the camp. Local tenderness may occur in both pleurisy and epidemic myalgia. Atypical virus pneumonia is ruled out by the absence of pyrexia in most cases and the fact that the patients were not ill; those in hospital showed no radiological evidence of the affection.

In 1924 Williamson described a condition having an onset resembling "subacute influenza," with pyrexia of short duration followed closely by pain of pleuritic type. In seven of his thirteen cases he detected a pleural rub, but no case developed