16 Abdominal tuberculosis

16.1 Introduction.

Abdominal TB is common and becoming more so, especially with HIV disease. Extra-pulmonary TB will suggest HIV disease in 90% of cases where TB was previously not so common. It is responsible for about 10% of bowel obstruction in some parts of the world, *viz.* India and Nepal. You may see it when you are expecting something else.

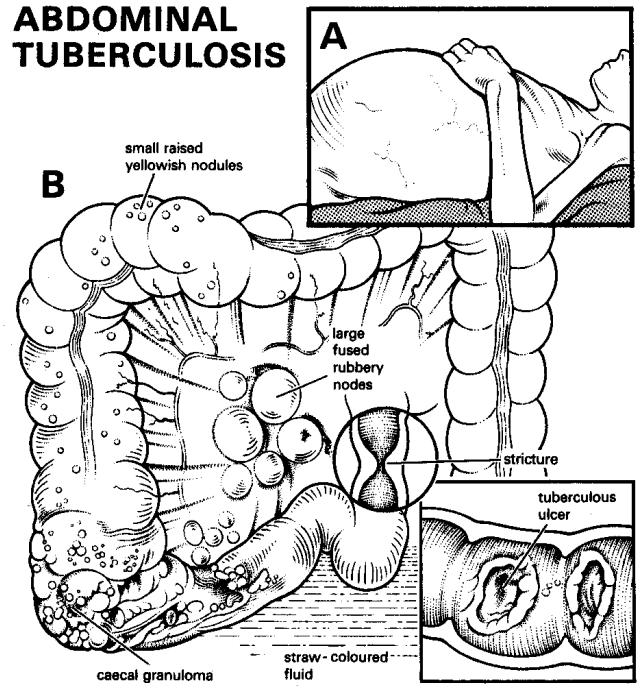

Fig. 16-1 ABDOMINAL TB can present in many ways. Patient A's abdomen is distended with ascitic fluid. You may not be able to diagnose some of the other forms of tuberculous peritonitis until you do a laparotomy. *Kindly contributed by Gerald Hankins.*

There are 4 main types, and several less common ones.
(1) The ascitic type.
(2).The plastic type, which causes intestinal obstruction, and may affect the gynaecological organs (23-3D).
(3) The glandular type, which involves the mesenteric nodes.
(4) Strictures anywhere in the bowel, but usually in the caecum and distal small bowel, where they are caused by contracture of a tuberculous ileocaecal mass to form a fibrous constriction. Strictures may be multiple, and are then highly likely to be due to TB.

Less commonly, you may also find:
(5) Tuberculous ulcers which can occur anywhere in the bowel, but are most often seen in the ileum, caecum, rectum, or sigmoid colon. In the small bowel, a tuberculous ulcer can cause diarrhoea. A tuberculous ulcer may perforate the bowel, or bleed; because this occurs distally, bleeding is usually from the rectum.
(6) Tuberculous sinuses or fistulae.
(7) An isolated tuberculoma, usually in the right iliac fossa, or other masses.
(8) Intussusception affecting the ileocaecal segment.
(9) Tuberculous appendicitis, presenting just as acute appendicitis.
(10) TB of the liver, spleen or pancreas.
(11) Tuberculous gastric or duodenal ulceration.

All this pathology can present in so many ways, and with so few distinguishing signs, that diagnosis is difficult. With all forms of TB a patient loses his appetite, loses weight, and feels ill, just as with TB elsewhere. There is vague abdominal pain and tenderness, and maybe vomiting. Depending on the type of TB there may be symptoms of abdominal swelling (the ascitic type), obstruction (the plastic type or tuberculous strictures), abdominal masses (the glandular type), bleeding or perforation (tuberculous ulcers). There may also be symptoms of chronic PID (gynaecological TB, 23.1). Not infrequently abdominal TB presents after a laparotomy for another condition, *e.g.* a Caesarean Section.

Unless there is obstruction or bleeding, you can treat the TB medically. But operate if the bowel obstructs completely (the plastic or stricturing type) or, rarely, if bleeding from tuberculous ulcers persists.

Start standard TB therapy (5.7) as soon as oral intake resumes.

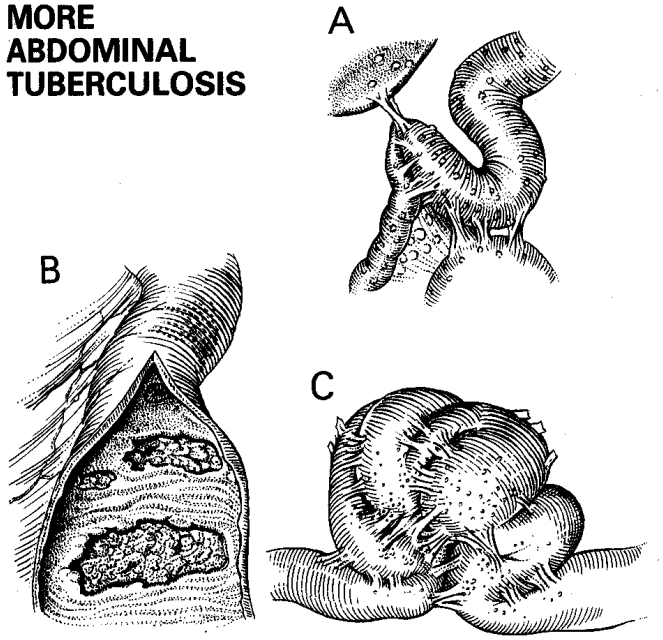

Fig. 16-2 OBSTRUCTING & ULCERATIVE ABDOMINAL TB. A, adhesion causing obstruction. B, tuberculous ulcers. C, coils of bowel matted together. *Adapted from a drawing by Frank Netter, with the kind permission of CIBA-GEIGY Ltd, Basle Switzerland.*

16.2 The ascitic type

Tuberculosis may be responsible for 80% of all your cases of ascites. Presentation is with a swollen abdomen containing many litres of straw-coloured fluid. A child with advanced abdominal TB typically has 'a ballooned abdomen and matchstick legs', but in many children the diagnosis is far from obvious. The fluid accumulates as a result of large numbers of exudative miliary tubercles on the peritoneum. The only certain way to make the diagnosis is to perform a mini-laparotomy, or laparoscopy, which will also enable you to diagnose cirrhosis, periportal fibrosis (due to *Schistosoma mansoni*), carcinomatosis of the peritoneum or hepatoma. You can usually diagnose miliary TB with your naked eyes; but you can be wrong, so take a biopsy of the parietal peritoneum and/or the liver. (The small seedlings may be cryptococcal in HIV+ve patients). Occasionally the ascites may be encysted and be confused for a giant ovarian cyst.

In the Concato syndrome, there is a pleural and pericardial effusion as well; this may be due to polyserositis, but in such cases, you should start anti-TB therapy (and prednisolone 120mg od, tapering off over 4wks) immediately and not intervene surgically: the patients are just too ill for that! *Only stop TB treatment if you can really prove its absence!*

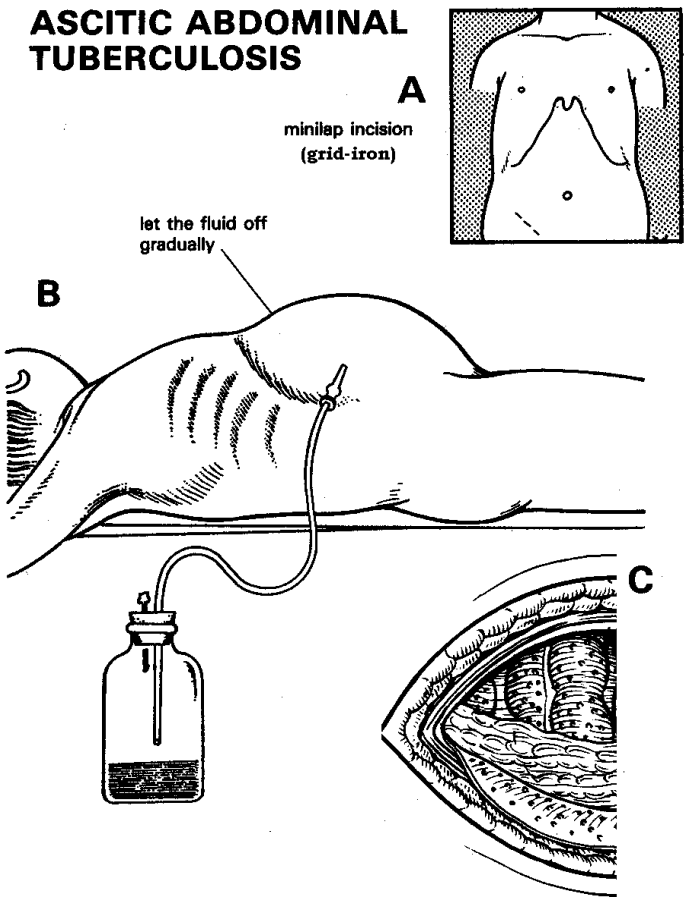

Fig.16-3 ASCITIC ABDOMINAL TB.
A, minilaporotomy (grid-iron) McBurney incision. B, draw off the fluid slowly before you start. C, miliary tubercles of the parietal peritoneum and bowel.

SPECIAL TESTS. Check the Hb, ESR and HIV. Get a chest radiograph and examine sputum for AAFB's. Do an abdominal ultrasound scan to look for lymphadenopathy, and the condition of the liver and kidneys.

Examine the ascitic fluid: if the white cell count is over 25/µl, with at least 70% lymphocytes, tuberculous peritonitis is fairly likely.
If the fluid has fewer lymphocytes than this, the ascites is more likely to be caused by cirrhosis, periportal fibrosis or bacterial sepsis. Your lab will be unlikely to find any AAFB in it, because they are very sparse. Use GeneXpert polymerase chain reaction (PCR) testing if possible (5.7)

Measure the protein in the peritoneal fluid. In tuberculous peritonitis it is usually 4-10g/l, but it may be up to 20g/l, or higher. Most patients with ≥20g/l have carcinomatosis. If it contains >4g/l of protein, it is likely to be an exudate. If it contains <4g/l, it is likely to be a transudate as found in cirrhosis or periportal fibrosis.

A more accurate test is the albumin gradient (*i.e* .the difference between serum and ascitic albumin): this is >1·1g/l in cases of portal hypertension, and significantly less than 1g/l in tuberculosis.

If you can do it, measuring adenosine deaminase is highly specific: levels >33 units/l indicate TB in >95%. Note that in the presence of cirrhosis, the value may be lower. You may be able to get special dipsticks for ascites which, though expensive, are very useful.

Blood in the fluid usually suggests malignancy.

THE DIFFERENTIAL DIAGNOSIS OF ASCITIC TB
Suggesting ascitic TB: miliary nodules on the peritoneum, each about 1-2mm in size, slightly raised and whitish. The nodules of carcinomatosis, which is the main differential diagnosis, are larger, usually >3mm, more vascular, and more irregular. You will soon learn to distinguish them. Illness is not as severe as would be with a malignant effusion of the same size.

Suggesting ascites secondary to liver disease: the liver may be enlarged, hard, and irregular, or small and hard to feel; the spleen is usually large; there are usually <4g/l of protein in the peritoneal fluid.

Suggesting the nephrotic syndrome: the ascites is less marked than the generalized oedema. If there is ascites, there is usually also marked oedema of the abdominal wall. There is usually <4g/l of protein in the peritoneal fluid.

Suggesting nutritional oedema (hypoproteinaemia): other signs of protein deficiency, but these may also be present in TB. There are usually <4g/l of protein in the peritoneal fluid.

Suggesting heart failure leading to cirrhosis and ascites: a raised jugular venous pressure, and other signs of heart failure; <4g/l of protein in the peritoneal fluid.

Suggesting carcinomatosis affecting the peritoneum: hard deposits in the pouch of Douglas, umbilicus or rectovesical pouch; usually >20g/l of protein in the peritoneal fluid.

Suggesting pancreatitis: epigastric and back pain, persisting after an acute episode, often related to alcohol excess or gallstones; the ascitic fluid usually has an amylase >2,000 IU/l.

A MINI-LAPAROTOMY OR LAPAROSCOPY TO DIAGNOSE THE CAUSE OF ASCITES (GRADE 2.3)

INDICATIONS. Ascites of uncertain cause. A patient can have more than one diagnosis, for example; cirrhosis *and* tuberculous peritonitis. Ascites predominating over other signs usually requires a mini-laparotomy. It is seldom indicated when the ascites is not predominant, as in the generalized oedema of heart failure, or renal disease.
Check the blood urea before you proceed.

CAUTION!

(1) *A mini-laparatomy is NOT suitable for exploring the abdomen*.
(2) You can diagnose tuberculous abdominal glands this way, but look for lymph nodes easier to access elsewhere, for example in the axilla. *You should not perform a mini-laparotomy to take a liver biopsy:* it might bleed catastrophically and you will then have inadequate exposure and/or anaesthesia to control it.
(3) Laparoscopy (19.5) is useful if you have the means but the same comment applies as with regard to liver biopsies.

DRAINING THE ASCITES. If there is more than mild ascites, draw off most of the ascitic fluid slowly before you begin. If it all escapes suddenly, as you open the abdomen, the circulation may collapse. So draw off 1l every 2hrs, starting 48hrs preoperatively, to a maximum of 6l. If there is still significant ascites, after you have withdrawn 6l, wait until next day before you draw off more. Use a wide-bore intravenous cannula, a drip set, and a sterile bottle. *Re-examine the abdomen once the ascites is drained away*; you may be able to feel a liver, spleen or other abdominal masses previously obscured.

CAUTION! To avoid possible injury to a large spleen, which may be difficult to feel because of the ascites, drain the fluid from the right lower abdomen.

ANAESTHESIA. Use LA in an adult or ketamine in a child. Avoid GA, because there may be cirrhosis.

INCISION. Make a 5cm right iliac fossa incision as for appendicectomy (14.1). This will allow you to see and examine a tuberculoma, and will be less likely than a midline incision to leak ascitic fluid postoperatively.

Look for miliary tubercles and secondary deposits on the peritoneum. Tubercles are remarkably uniform in size, and fairly uniform in appearance (like salt grains). Biopsy the peritoneum by removing an elliptical piece of the parietal peritoneum 2x0·5cm, from the edge of the abdominal incision. Close in the usual way, but *do not insert a drain*, as it will leak continuously.

16.3 The plastic peritonitic type

This is the result of a tuberculous granuloma, which causes the omentum, and the other structures in the abdomen, particularly loops of the distal small bowel, caecum, and ascending colon, to stick together with many adhesions. The affected coils of bowel are thick and rubbery, with characteristic transverse lesions on the small bowel.
Loops of small bowel may obstruct, and be difficult to separate. Carcinoma, *amoeboma*, and Crohn's disease can all cause a plastic peritonitis, but TB is more common than all these others combined. Amoebiasis makes loops of small bowel stick to the descending colon, without causing a true plastic peritonitis.
The obstruction in the bowel is commonly incomplete, so that symptoms are subacute or chronic, and may have lasted months or years. The adhesions which stick the loops of the bowel together are extensive and difficult to separate, so manage them non-operatively if you can. Start anti-TB treatment, and oral fluids only if tolerated, before proceeding to normal diet. A tuberculous granuloma of the small bowel usually resolves without a stricture; but in the ileocaecal area fibrosis and stenosis often follow.

Occasionally, you may have to operate for persisting complete obstruction. Even then, if you know TB is the cause, you will be wise to try non-operative treatment for a few days first provided there is no strangulation. When you do operate, you may find that there is no stricture in the wall of the bowel, and that you can relieve the obstruction by dividing adhesions only, but *do not divide adhesions which are not causing obstruction*. Try to avoid opening the bowel, because there is always a danger that a fistula may follow. If you have to open bowel, you have a choice between:
(1) A 'stricturoplasty', if there is a narrow stricture in the small bowel (16-4A).
(2) A small-bowel resection.
(3) An ileocaecal resection. *Avoid bypass procedures such as ileo-transverse colostomy or entero-enterostomy*: they result in blind loops, malabsorption and further obstruction; use these only when extensive matting of bowel prevents you from reaching the site of obstruction, but knowing that another laparatomy will be necessary later!

Avoid these common mistakes:
(1) *Do not try to make a diagnosis without understanding the nature of the disease*. Weigh up the signs and symptoms carefully.
(2) *Do not be too eager to start a therapeutic trial without confirming the diagnosis*: there may be some other disease. A mini-laparotomy under LA (16-3) is almost always possible.
(3) If you cannot make a diagnosis, *do not wait too long before exploring the abdomen*.
(4) If there is chronic obstruction, which does not respond to non-operative treatment, surgery is mandatory.
(5) If the patient is desperately ill, *do not make meddlesome and dangerous attempts to resect grossly scarred bowel, nor to free difficult adhesions.*

SYMPTOMS.
Weight loss (all cases), may be excessive.
Weakness, malaise, fatigue, and anorexia (75%) with also nausea and vomiting, fever and night sweats (60%).
Abdominal pain (90%) is usually constant, central, and not severe. If it is in the right lower quadrant, it suggests ileocaecal TB. With ascites pain is often mild, and may be absent.
Alternating constipation and diarrhoea, cramps, and gurglings (30%). Typically, a description of a 'ball of wind' moving in the abdomen.
Rectal bleeding (5%) may be severe. Vaginal discharge and bleeding may be marked.
Steatorrhoea with pale, bulky, and offensive stools (5%).
N.B. A chronic cough, blood-stained sputum, and TB elsewhere may not be present.

SIGNS
Abdominal tenderness (60%) is ill-defined, and is usually maximal in the middle of the abdomen. There is often a peculiar 'doughy' feel (20%) of the abdominal wall.
An abdominal mass (40%) may be present. There may be some well-defined tender rubbery masses, either in the ileocaecal region, at the base of the mesentery, or in the adnexa or pelvis in a female. A mass is unusual in the ascitic type.
Signs of obstruction to the lower small bowel may be acute or subacute.
Anaemia is usually moderate.
Dependent oedema as the result of hypoproteinaemia is common.
Lymphadenopathy: look carefully for enlarged axillary or cervical lymph nodes; if you find one, biopsy it. An inguinal node is less likely to be diagnostic, unless it is very large. (17.4)
Perianal sinuses or fistulae may be present: *do not forget to look at the anorectum!*

SPECIAL TESTS. Check the Hb, ESR and HIV, and examine the stools. Get chest and erect and supine abdominal radiographs, and examine the sputum for AAFB's. *Avoid a barium follow-through in the acute stage: it may make an incomplete obstruction complete. However, you can use water-soluble contrast, and this may also relieve an incomplete obstruction through its osmotic effects.*

Get an abdominal ultrasound to look for lymphadenopathy, thickened bowel, peritoneal nodules, areas of calcification, intestinal stones, a mass or 'cold' abscesses. You may be able to aspirate for AAFB's.

Perform a sigmoidoscopy (26-3) if there is rectal bleeding; if this is unhelpful try to get a barium enema radiograph (38-3). All parts of the colon may be involved with segmental hypertrophic, annular or ulcerative lesions 5-7cm long.

The appearance may look like a carcinoma; TB can indeed be present with a carcinoma, so operation is usually necessary unless you can get a biopsy endoscopically.

DIFFERENTIAL DIAGNOSIS OF PLASTIC ABDOMINAL TB

Suggesting ascaris infection: a child with vague abdominal pain, and subacute obstruction but no weight loss or fever. Tenderness is not constant, and palpable masses of worms are unusual (12.5).

Suggesting an appendix mass: a short history, and an acute onset.

Suggesting amoebiasis: a history of passing blood and mucus rectally, and *trophozoites* in the stools.

Suggesting carcinoma of the colon: an urban life style. It does occur in villagers but is unusual.

Suggesting cirrhosis or a liver tumour: an irregular firm or hard liver, prominent ascites, and a large spleen, a previous attack of hepatitis, or chronic alcohol abuse. A bruit is often present (15.11).

Suggesting Crohn's disease: loss of weight and diarrhoea are the main symptoms. The differential diagnosis may be impossible until tissues are examined histologically. The disease is very rare outside the West.

Suggesting *Oesophagostomiasis*: multiple nodules in the colonic wall, which may become confluent and thickened, associated with *O. bifircum* eggs in the stool, especially found in Northern Ghana & Togo.

NON-OPERATIVE TREATMENT FOR ABDOMINAL TB
INDICATIONS.

(1) You are reasonably certain of the diagnosis, and feel a therapeutic trial is reasonable.
(2) There is incomplete obstruction.
(3) There are no signs of strangulation.
(4) There is tuberculous ulceration without perforation or severe bleeding.

Start standard anti-TB therapy (5.7). In an adult, the abdominal symptoms and masses are unlikely to respond for about 2 months, although a child may respond sooner. However, fever, anorexia and malaise will subside sooner. If there is no response to TB treatment, consider the possibility of another diagnosis.

If there is partial obstruction, pass a nasogastric tube, and resuscitate with IV fluids (12.4). If the obstruction passes off, you may not need to operate.

DIAGNOSTIC LAPAROTOMY FOR PLASTIC ABDOMINAL TB (GRADE 3.3)

A mini-laparotomy for ascitic TB (16-3) is not suitable for the plastic type of TB; a standard laparotomy through an ordinary incision is, however, a more extensive procedure, which may involve you in further surgery. Do this, however, if there is a persistent vague abdominal pain, perhaps some intestinal symptoms, weight loss, and a raised ESR, and especially if there is a mass.

Open the peritoneal cavity through a midline incision, mostly below the umbilicus, and look for the signs of TB. Biopsy a lymph node. If the site of the biopsy bleeds, control it with packs or with a 3/0 figure of 8 suture which runs under the bleeding point on both sides.

If you cannot find peritoneal tubercles or rubbery lymph nodes easily, take a biopsy from the parietal peritoneum.

If you find a firm mass at the ileocaecal junction, perhaps with adhesions to adjacent structures and a normal peritoneum, the diagnosis is more difficult. Cut across an enlarged node. If you see caseous areas, you have confirmed the diagnosis. Even so, take a specimen for histology. *Avoid taking a biopsy from the wall of the bowel:* this may lead to a fistula. If the nature of the ileocaecal mass is uncertain, but is probably tuberculous, leave it if it is not causing obstruction. In this case, perform an ileocaecal resection (12.7), or if this is too difficult, an ileo-transversostomy side-to-side. If it might be neoplastic, and mobile, try to perform a right hemicolectomy (or ileocaecal resection if there are metastases); if immobile, biopsy an enlarged node nearby and await the histological report.

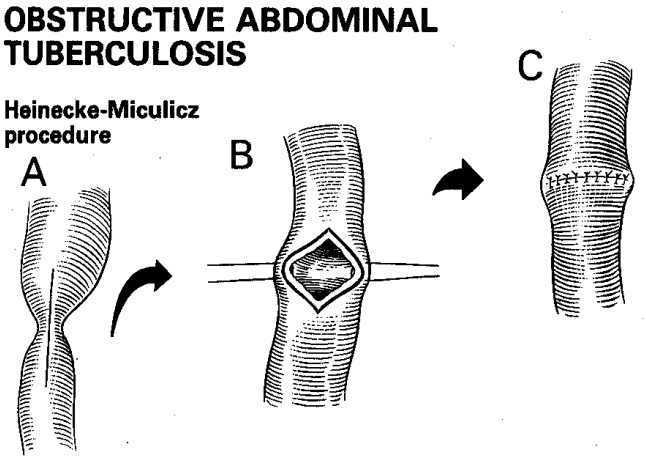

Fig. 16-4 STRICTUROPLASTY FOR OBSTRUCTIVE ABDOMINAL TB. A-C, Heinecke-Mickulicz procedure for a stricture of the small bowel. A, incise the bowel longitudinally. B, insert stay sutures beside the middle of the incision, pull them out, and suture the bowel transversely. C, completed procedure. *Kindly contributed by S. Nandy.*

If you find a large 'cold' abscess, biopsy and drain it.

If you find an inflamed appendix, excise it taking care that the base of the appendix is closed securely.

If you find a thick mobile fibrotic segment of small bowel and the ileocaecal region is normal, resect it and anastomose the ends (11-7). If it is very short, perform a stricturoplasty (16-4A).

If a tuberculous ulcer has perforated the terminal ileum, oversew and patch it, as you would with a typhoid perforation (14.3). Treat it by resection, if the perforation is large and friable.

If a tuberculous ulcer of the colon has fistulated into the bladder or vagina, make a defunctioning colostomy (11.6); *do not attempt resection until treatment has made the patient better.*

If loops of the bowel are severely stuck down by plastic peritonitis, *do not do too much dissection:* the risks of creating fistulae are too high. Instead, if obstruction is definite, perform a simple side to side ileo-transverse colostomy or entero-enterostomy (11-10) without resecting any bowel. This will bypass the diseased segment, and avoid much stressful surgery but creates a 'blind loop'. However, a repeat laparotomy later will probably be needed after anti-TB treatment is completed in order to correct malabsorption problems of the blind loop.

16.4 The glandular type

This presents as irregular lumps in a child's or young adult's abdomen, sometimes with ascites, and with little tendency to obstruct. The mesenteric nodes are large, and not very mobile. They may be so large that you can feel them through the abdominal wall. They are matted together, and firm to hard, with characteristic pale yellow areas of caseation on their cut surfaces. There is often hepatosplenomegaly, moderate anaemia, dry depigmented skin, hypoproteinaemic oedema and low-grade fever.

In gastroduodenal TB, lymph node involvement is common; for stricturing in the duodenum perform a gastrojejunostomy (13-16). Occasionally enlarged lymph nodes in the *porta hepatis* cause obstructive jaundice (15.9) or portal hypertension.

Sinuses or fistulae often develop from deep-seated infected nodes or tuberculous abscesses, especially in HIV+ve patients. You find them in the iliac fossae, in the suprapubic area, or at the umbilicus. Unfortunately biopsy of the track only demonstrates TB in 20%, so look for evidence of TB elsewhere.

Often you can be fairly certain of the diagnosis. If many lymph nodes are involved, biopsy one from the neck, axilla, or groin (17.4). Non-specific adenitis is common in the groin, so only biopsy an unusually large one. You may see enlarged nodes in a chest radiograph. If you cannot establish the diagnosis in any other way, you may need to perform a laparotomy and take a node for biopsy. Lymphoma is the important differential diagnosis (17.6).

With reasonable clinical suspicion, a trial of anti-TB therapy is justified.

16.5 Tuberculosis of liver, spleen or pancreas

Tuberculosis of the solid abdominal organs is uncommon (1%) and often, but not always, found when there is TB elsewhere. Where it affected the adrenal gland in Addison's original case, it resulted only in endocrine disorders. In the liver, there may be multiple granulomas, a single mass with cavitation, or abscess formation; in the spleen an enlarging semi-solid mass with cavitation. In both forms there is enlargement of the organ but otherwise symptoms and signs are non-specific. Diagnosis is easiest with ultrasound and needle aspiration.

Other causes of hepatic granulomas may be leprosy, brucellosis, syphilis, lymphoma, and drug damage!

Surgical intervention is not necessary. You should avoid ethambutol in treatment as this may further damage the liver. TB affecting the pancreas resembles chronic pancreatitis and can only be differentiated by needle aspiration cytology at operation or biopsy of an adjacent lymph node. *Do not try to biopsy the pancreas itself* for fear of causing a pancreatic leak. HIV disease can cause pancreatitis on its own, but if an ultrasound shows a mass, abscess or cyst, especially in the head of the pancreas, you are probably justified in starting anti-TB therapy.

16.6 Small & large bowel tuberculosis.

Any part of the bowel may be involved with TB, but especially the ileocaecal junction. Isolated segments, though, usually 5-7cm long, resulting in hypertrophy of the wall, ulceration or stricturing are not that uncommon.

Perforation, fistula formation and intussusception may result. The appearances are virtually indistinguishable from Crohn's disease, which is very rare outside the Northern hemisphere.

Ileocaecal TB may look like an adnexal mass, an amoeboma, a mass of *ascaris* worms, an intussusception, a caecal carcinoma, a lymphoma, an ileal phytobezoar, a bilharzioma, actinomycosis, or angiostrongyliasis, but unlike malignancy there is no history of weight loss and anorexia.

SPECIAL TESTS.
Barium enema (38.1) is especially useful for ileocaecal or colorectal disease; initially there is hyperirritability of the bowel so there is flocculation of contrast throughout the small bowel with absence of contrast in the diseased segment. The loops lose their mobility and then strictures form with proximal dilation (which may be massive) and filling defects or ring shapes due to enteroliths. The caecum becomes contracted and conical in shape with filling defects, and the ascending colon stenosed and shortened, pulling up the ileocaecal junction. This results in a widened ileocaecal angle (the 'goose-neck' deformity).

In intussusception there is a crescent-ring filling defect, or 'watch-spring spirals' of barium seen as contrast is held up or seeps past the intussuscepting bowel. Ultrasound scan appearance is typical (12.7).

Colonic lesions look like carcinomas with 'apple-core' deformities.

MANAGEMENT.
Ileocaecal resection, stricturoplasty, entero-enterostomy or ileo-transverse colostomy are the options if surgical intervention is necessary (16.3).

Involvement of the appendix (2%) may be primary or as a result of ileocaecal infection; unless you send the appendix for histology, you may miss the diagnosis!
Colorectal involvement usually results in bleeding, which may be severe. The sigmoid and rectum are the commonest sites. Check the stools for *trophozoites* to exclude amoebiasis. You may not be able to distinguish colonic lesions from carcinoma, which may exist simultaneously, so a resection may still be best, if it is possible.

16.7 Urological tuberculosis

If there is persistent cystitis, which fails to respond to antibiotics, with pus cells and red cells in the urine, but no bacteria are cultured from it by routine methods, there may be TB of the urinary tract. There may also be HIV cystitis, or both! You will see this usually in a young adult without signs of TB elsewhere. Treatment of the TB is usually effective if the disease is not too far advanced, and treatment is taken conscientiously. Improvement may be dramatic, especially if you can start early, and even strictures of the ureter have been known to heal. So watch for urological TB, and be prepared to treat on suspicion alone. The surgery needed for late presentation is complex. Unfortunately, the disease starts so insidiously that there may be no complaints till late.

Bacilli reach one of the kidneys (usually only one, but sometimes both) in the blood, after which caseation slowly destroys it. Only when the disease has eroded into its calyces do bacilli spread in the urine down to the ureter and bladder, and cause frequency and pyuria. Eventually, most of the kidney is destroyed, after which the disease may spread beyond, to form a palpable mass in the loin, perhaps with a discharging sinus.

TB inflames the mucosa of the bladder and forms tubercles which may ulcerate, coalesce, and form shallow ulcers, especially round the orifices of the ureters and on the trigone. Ultimately, much of the wall of the bladder is destroyed, so that it ends up scarred, red, and contracted. A ureter which drains a tuberculous kidney is flooded with bacilli, and becomes thick, fibrosed and strictured, usually in its lower third. Above this, the urinary tract dilates to form a hydro- or pyo-nephrosis.

Presentation occurs with:
(1) Symptoms of chronic cystitis: frequency and dysuria. This later progresses to the burning nocturia and strangury (slow and painful discharge of urine, drop by drop) of a small shrunken bladder, which may become secondarily infected. These symptoms make the bladder appear to be the cause of the disease, rather than the kidney.
(2) Painless intermittent microscopic haematuria or rarely obvious bleeding. (By contrast, a renal carcinoma usually presents with macroscopic haematuria).
(3) Dull discomfort in the loin, which gets steadily worse, especially when TB is complicated by a pyogenic infection (20% of cases). The kidneys are not enlarged or tender, until late.
(4) Malaise and the usual general symptoms of TB.

SIGNS. The kidneys may be palpably enlarged. Several parts of the urinary tract and genitalia may be involved at the same time: a non-tender irregular and boggy prostate, *not* usually enlarged; thickened oedematous spermatic cords with vesicles thick and boggy; thick, woody, and craggy epididymes, which may caseate, and form sinuses, or may involve the testis, and cause a secondary hydrocoele.

SPECIAL TESTS. Urine with pus cells and red cells, but no bacteria on standard culture (unless there is secondary infection), is strongly suggestive. However recurrent *E Coli* urinary infection occurs in 20%.

A 24hr urine, or a freshly-voided clean-catch early morning urine specimen (after a period of dehydration), may show AAFB in a stained film. Repeat the examination 3-5 times. This needs little equipment, but it does require considerable skill, and much patience. You will probably have to rely on finding pus cells and red cells only. If possible, culture the urine for AAFB. Fine needle aspiration from the epididymis may be more helpful.
Ultrasound may show an irregular shrunken bladder or deformed kidney, but is not that useful.
Perform a cystoscopy before an intravenous urogram (IVU, 38.1a), which is much more expensive and will only show changes if disease is advanced. This may show 'moth-eaten calyces', and dilatation of the renal pelvis, and ureter. There may be extravasation of contrast. Cavitation in the kidney is highly suggestive of TB. If it is very advanced, the kidney will not be functioning. There may be multiple long ureteric strictures; a corkscrew appearance and beaded ureter strongly suggest TB. The bladder is small and contracted.

DIFFERENTIAL DIANOSIS. In endemic areas *Schistosomiasis* is much the most common cause of pain on micturition with pus cells and red cells in the urine, compared with urinary TB, which is uncommon or rare.

Suggesting *schistosomiasis:* small 3-5mm nodules in the epididymis, nearly always in the tail, and calcification of the bladder wall, as shown by a line in the shape of the bladder, which collapses after micturition.

CYSTOSCOPY (27.3) will confirm the diagnosis, show the degree of involvement of the bladder, and exclude *schistosomiasis*. You may see a 'golf-hole' ureteric orifice.

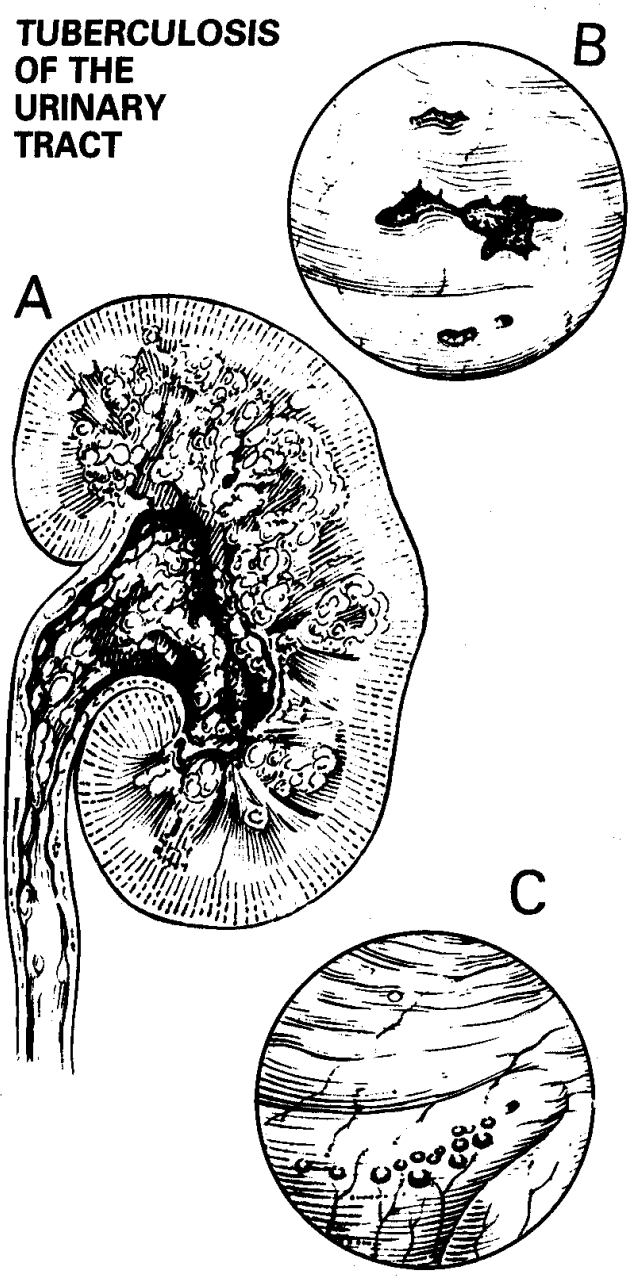

Fig. 16-5 TUBERCULOSIS OF THE URINARY TRACT.
A, TB of the kidney involving the pelvis and the ureter. B, tuberculous ulcers of the bladder. C, tubercles near the orifice of the ureter.
Adapted from a drawing by Frank Netter, with the kind permission of CIBA-EIY Ltd, Basle Switzerland.

TREATMENT OF RENAL TB
Treat on an outpatient basis, with standard TB therapy (5.7). If renal function is impaired, avoid streptomycin, or ethambutol, or use them intermittently. Rifampicin, isoniazid and pyrazinamide are safe. Review every 2 months for regular assessment, including the examination of the urine.

If there is a relapse, and you think that drugs have not been taken faithfully, consider changing to a regime using second-line drugs. Check the HIV status if this has not already been done, or was previously -ve.

If you are in an endemic area and routine examination shows no ova of *Schistosoma haematobium*, examine the deposit from a specimen passed at midday (the time when most ova are passed) on 3 consecutive days.
Check the Hb (low), ESR (raised) and HIV status.

ULTRASOUND may show caseation in the pelvis of the kidneys, hydronephrosis, hydro-ureter, and a shrunken bladder, but will only show advanced pathology.

RADIORAPHS. On plain abdominal films, look for the outline of an enlarged kidney, diffuse calcification, and obliteration of the psoas shadow. et a chest film.

INDICATIONS FOR SURGERY. Operations for renal TB are complicated and need an expert.

If the IVU shows no function, or has a moth-eaten appearance, with flecks of calcium, a nephrectomy is needed. Hypertension is an additional reason. If the patient is toxic and febrile, suggesting a pyonephrosis, or a perinephric abscess, these need urgent drainage (6.15, 27.14).

If there is a ureteric stricture, ideally a ureterogram is needed. If you have drained a pyo- or hydro-nephrosis externally, you can inject contrast through the nephrostomy tube. For upper ureteric strictures, a pyeloplasty is needed; for lower ureteric strictures, a re-implantation of the ureter or bladder flap.

N.B. In endemic areas, Schistosomiasis is a common cause of a lower ureteric stricture.

If there is still extreme frequency and dysuria after 6 months of treatment, suspect that there is a small contracted bladder. Confirm this by ultrasound or cystoscopy and/or a cystogram. Surgery may be possible to augment the size of the bladder.