CHAPTER 21

Surgery in the Tropics

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21.1 INTRODUCTION

The spectrum of surgical care in the tropics varies from excellent to almost non-existent. The majority of the tropical countries, being developing countries, share the common phenomenon of scarce surgical resources both in terms of personnel and medical equipment. The estimated number of surgeons per 100,000 populations is only 7 for Colombia; 1.5 for the Philippines; 0.5 for West Africa and only one surgeon for every 180,000 people in Papua New Guinea[1]

> "In surgery physiology is the king, anatomy the queen: you can be the prince, but only provided you have the judgement" (Mosche Schein, Professor of Surgery, Cornell University).

The principles of surgery in tropical/developing countries are no different from those in the developed countries, but the practice of surgery may need to be modified due to a lack of optimum supporting facilities compounded by late presentations. Outside teaching or capital city hospitals a tropical surgeon is unlikely to have a radiologist, or a pathologist for frozen sections or cytopathology, which leads to difficulty in making an accurate diagnosis of malignancy and institution of treatment. The surgeon in these situations needs to make decisions on the basis of clinical features, often in absence of a back-up surgical specialist in the region.

The profile of surgical patients also differs from those seen in developed and temperate countries. Patients tend to be younger but often present with advanced disease associated with poor nutrition. Surgical workload mainly comprises managing trauma and the sequel of infective processes. The surgeon in the tropics often will be called upon to manage obstetrical emergencies such as obstructed labour (Caesarian section), rupture of the uterus or a ruptured ectopic pregnancy.

Surgical conditions of special note in tropical and developing countries include:

- Trauma
- Infection
- Tropical ulcer
- Mycobacterial ulcer
- Fournier's gangrene
- Cancrum oris
- Enteritis necroticans
- Pyomyositis
- Splenic problems

- Tuberculosis
- Amoebiasis
- Chagas' Disease
- Schistosomiasis
- Hydatid disease
- Ascariasis
- Helminthic appendicitis
- Nodular worms
- Filariasis

21.2 TRAUMA

Road trauma is the major cause of accidental injuries and other causes include farming injuries, falls from trees, tribal fights, political/tribal warfare, and land mines. Major burns both accidental and suicidal are common occurrence, especially in small children.

Delayed presentation of common fractures and dislocations poses significant challenge in treatment and often surgery is performed to improve function rather than restoration of anatomical alignment.

21.3 INFECTION

Abscesses involving every organ from skin to bone are common, with *Staphylococcus aureus* being the most frequent aetiological agent. Of particular interest are pyomyositis (abscesses occurring in the muscles) - a condition rarely, if ever seen, in developed temperate countries[2].

Surgical complications from typhoid such as intestinal perforation, once common, now occur in about 3% of cases[2] Tuberculosis affecting almost every body part is common and often presents with atypical clinical features.

Deformities and ulcers from leprosy still occur and require special surgical skills for treatment.

Fungal infection such as maduramycosis and the similar filamentous bacterial conditions of nocardiosis and actinomycosis is commonly seen in the thorny semi desert zones of the equator and elsewhere in tropical or developing regions. They are all characterised by swelling, multiple discharging sinuses and, in the case of the latter infection, the discharge of yellow sulphur granules,

Parasitic infections dominate the infectious disease scene in the tropics, and malaria causing splenomegaly offers a special surgical problem as will be discussed in more detail later. Schistosomiasis, amoebiasis, filariasis, ascariasis and South American Trypansomiasis (Chagas' disease), all can lead to sequelae and complications for which surgical intervention may be needed.

21.4 TROPICAL ULCER

Acute tropical ulcer is a specific entity caused by fusiform bacilli and spirochaetes and occurs in the tropical and subtropical regions[3]. However, the so-called chronic tropical ulcers, apart from the ones which follow the acute tropical ulcers, can occur anywhere in the world arising from a combination of trauma, poor nutrition and poor hygiene.
The aetiological factors of true tropical ulcers can be best remembered by the mnemonic: flies, friction (trauma), (lack of) food, and fuso-spirillosis infection[4] Ulcers usually occur on the lower leg, initially as a papule, which rapidly ulcerates associated with pain, and malodorous exudates. Without treatment ulcers become chronic and may persist for many years.

Treatment of acute ulcers consists of improving nutrition and hygiene, debridement, and dressings. Penicillin and metronidazole are given for one to two weeks. Once the ulcer bed is covered with clean granulation tissue, a partial thickness skin graft will speed up the healing.

In the case of a chronic ulcer, multiple small biopsies from the margin are necessary to exclude possible squamous cell cancer, which may occur in a chronic ulcer.

21.5 MYCOBACTERIAL ULCER (Buruli ulcer)

Initially reported in Australia as Bairnsdale (or now sometimes, Daintree) ulcer in 1945, the same disease became known as Buruli ulcer in Uganda, and in Papua New Guinea as Kumuzi ("Sick bilong Sepik") ulcer. It is caused by the acid-fast bacillus, *Mycobacterium ulcerans* and is often found in swampy areas[5]. The actual reservoir of the organism has yet to be identified. It is common in children, but people of all ages can be affected. Ulcers occur more often on the leg following minor trauma, often unnoticed. Lesions start as an indurated nodule, which subsequently ulcerates and rapidly progresses subcutaneously with undermined edges. Healing occurs in the centre while ulceration progresses at the periphery with necrosis of subcutaneous tissue containing the acid-fast bacilli. The laboratory diagnosis is based on finding *M.ulcerans* in smear and histology. Currently PCR (polymerase chain reaction) has been developed for rapid confirmatory diagnosis[6]

Treatment is essentially debridement of the ulcer and curetting the undermined edges followed by repeated skin-grafting[7]. It has been noted that keeping the affected limb under a plaster cast promotes healing by raising the local temperature.
Recently a combination of streptomycin and rifampicin for 8 weeks have been reported to be effective in this condition (John Buntine- personal communication).

21.6 FOURNIER'S GANGRENE

Fournier's gangrene is a rapidly progressive necrotizing soft tissue infection of the scrotum or perianal region, associated with severe systemic illness and caused by a synergistic combination of anaerobic and aerobic bacteria. Typically it occurs in male patients, being associated with depressed immune states as in diabetes, malignancy, alcohol and drug abuse. A recent history of urethral dilatation, catheterization, perianal abscess, anal fistula or fissure predisposes to mixed infection by bacteria (*Bacteroides*, coliforms, *Klebsiella*, *Proteus*,

staphylococci, and streptococci). The infection travels along the fascial planes of the perineum and can extend into the anterior abdominal wall.
The disease usually starts with perianal or perineal pain, often disproportionate with physical findings such as swelling or pruritus in the affected area[8]. Apart from local discolouration and pain the patient develops fever, chills, sweats and malaise. Rapid deterioration leading to death may occur if progress remains uncontrolled.

Treatment involves the use of intravenous broad spectrum antibiotics and aggressive, often repeated, surgical debridement. Hyperbaric oxygen therapy, although remains unproven in published studies, can be a valuable adjunct in controlling the infection (Morpurgo[8]).

21.7 CANCRUM ORIS (Noma)

This is an infective gangrene of the cheek or mouth occurring in undernourished individual, usually children, with poor oral hygiene and usually following an acute illness such as measles. Noma is reported to be more common in sub-Saharan Africa, mostly in West Africa. As in tropical ulcers, the organisms responsible are fusiform bacilli and spirochaetes. Noma often starts as an ulcer on the oral mucosa and rapidly develops into a massive necrosis, moving from inside outward, often involving major portions of the face. Without appropriate treatment the patient (usually child) will perish from complications e.g, pneumonia, septicaemia or diarrhea. Those who survive usually suffer from severe facial disfigurement and/or trismus and will need difficult staged surgical reconstructions.

Initial treatment is with penicillin or broad spectrum antibiotics together with rehydration, correction of electrolyte imbalances and supplemental nutrition[9].

21.8 ENTERITIS NECROTICANS (Pig-Bel)

Enteritis necroticans, also known as ‘Pig-Bel’ in the highlands of Papua New Guinea (PNG), and ‘Darmbrand’ (firebowels) in post World War II Germany, is a patchy necrotic process affecting the small bowel caused by the beta-toxin of *Clostridium perfringens* type C. The disease has also been reported from Uganda, Indonesia, Thailand, Malaysia, the Solomon Islands, Bangladesh and China.

In the highlands of PNG, the disease most commonly affects children following a feast of pig meat and sweet potato. The pig meat is contaminated with beta-toxin producing *Cl.perfringens* type C. The exotoxin is normally susceptible to destruction by intestinal proteases, including trypsin. However, the sweet potato diet and concurrent *Ascaris* infection results in the production of trypsin inhibitors and this allows the exotoxin to reduce gut motility, increasing adherence of the clostridia with more production of toxin resulting in necrosis of the mucosa in sharply demarcated zones.

The usual clinical presentation is abdominal pain, distention, vomiting, and passage of bloody diarrhoea. This is associated with variable systemic features of toxaemia and shock depending on the severity of the disease. In severe cases death occurs within 24 hours while mild cases recover and may subsequently present with subacute obstruction or malnutrition.

Initial treatment is restoration of fluid and electrolyte imbalance by intravenous therapy, nasogastric decompression and antibiotics. Intravenous penicillin and chloramphenicol (or a suitable broad spectrum antibiotic) are the antibiotics of choice. In the absence of improvement or the presence of signs of bowel perforation or peritonitis, laparotomy is indicated. At operation resection of the gangrenous segment of bowel is performed.

The introduction of a toxoid vaccine, given to infants at the same time as the triple antigen vaccine, has been found to be highly successful in preventing this disease in PNG[10].

21.9 PYOMYOSITIS (myositis tropicans)

Skeletal muscles normally enjoy immunity to pyogenic infections. However, in tropical and rarely in temperate climates, purulent infection of skeletal muscle occurs without penetrating trauma or spread from an adjacent septic focus. In 95% cases *Staphylococcus aureus* is cultured from the pus, although *Streptococcus pyogenes*, and *Escherichia coli* have also been found.

The mechanism by which bacteria cause infection in the muscle and the reasons for its occurrence mainly in the tropical countries are unknown, but several factors such as chronic ill health and debility, trauma producing muscle haematoma, associated filariasis, *Dracunculus* infection, malaria and viral myositis have all been implicated as predisposing factors. Recent data indicate that this is often seen in immunocompromised patients as in HIV.

The condition is more common in men and affects usually the heavy powerful muscles of the trunk and extremity. The illness starts with fever and muscle pain progressing to suppuration with classical signs of acute inflammation and later septicaemia, if untreated.

A recent history of trauma associated with localized tenderness of the muscle helps in the diagnosis. Ultrasonography is very useful and a guided needle aspiration of pus confirms the diagnosis.

Once the diagnosis is established the treatment is drainage of the abscess under cover of antibiotics such as cloxacillin. If the patient fails to improve and/or culture and sensitivity of pus swabs are not available, treatment with combination of gentamicin, cephalosporins and metronidazole is indicated[11].

21.10 SPLEEN

In the tropics the three main conditions affecting the spleen and for which a surgeon is involved, are splenomegaly (post-malaria, tropical splenomegaly syndrome), splenic abscess and splenic trauma.

21.10.1 Splenomegaly

Chronic splenomegaly in malarious countries, like PNG, Uganda, Zambia and Nigeria, may be seen in up to 80% of population. It is believed that the post-malarial splenomegaly occurs following chronic stimulation of the splenic reticuloendothelial system by circulating antigen-antibody complexes that are triggered by a non-specific mitogen from the malarial parasite.

Chronic schistosomiasis with severe liver fibrosis can also lead to a secondary splenomegaly in areas endemic for bilharziasis.

The massively enlarged spleen in malaria may produce portal hypertension from increased splenic blood flow, pancytopaenia from sequestration of erythrocytes, neutrophils and thrombocytopaenia from sequestration of platelets.

Splenectomy, although it may produce initial benefit, almost always results in higher mortality subsequently due to overwhelming sepsis, malaria or even babesiosis. Hence, splenectomy is no longer recommended and patients with post-malarial splenomegaly should be treated with lifelong antimalarial medication, which has been shown to result in reduction of splenic size.

21.10.2 Splenic abscess

These can be primary or idiopathic and secondary from distant septic focus by haematogenous spread. It is more common in patients with HIV/AIDS[12]. It is the primary type, which is seen mostly in Africa where malaria and sickle cell disease co-exist. In these cases the material found in the abscess cavity is usually sterile and contains cholesterol crystals.

Aspiration or drainage of the abscess with splenic preservation is the preferred course of treatment. However, splenectomy is the definitive treatment when radiological intervention is not possible or failed[12].

21.10.3 Splenic trauma

The enlarged and friable spleen following chronic malarial infection is vulnerable to rupture from even low impact injury. The availability of ultrasonography has been very useful in the diagnosis and monitoring of splenic injury. Conservative treatment of splenic injury is possible even in small hospitals with close observation, and appropriate resuscitation[13]. Successful outcome has been reported with non-operative management of spontaneously ruptured malarial spleen[14]. However, persisting haemorrhage and presence of coexisting hollow visceral injuries necessitate laparotomy, and even in such cases splenic repair or segmental resection should be attempted.

In the event of splenectomy the patient must be immunized with polyvalent vaccines for the pneumococcus, *Haemophilus influenzae* and the meningococcus.

21.11 TUBERCULOSIS (TB)

“If you can’t think of a diagnosis, always remember the possibility of tuberculosis”

21.11.1 Tuberculosis of the breast

Tuberculosis of the breast is known to occur in Africa, India and Hong Kong where tuberculosis is endemic. Its importance lies in the fact that the condition may clinically simulate breast cancer with its presentation such as, breast lump, nipple retraction, Peau

d'orange appearance and axillary lymphadenopathy[15]. It usually affects young women between 20 and 40 years. As in any case of a breast lump, tissue diagnosis by fine needle aspiration or excision biopsy of the breast swelling is essential before instituting appropriate antituberculous chemotherapy for at least 6 months.

21.11.2 Abdominal tuberculosis

The prevalence of abdominal tuberculosis varies between 1% to 10-20% of all cases of TB[16]. Abdominal TB may present as acute intestinal obstruction following stricture of small bowel, as ascites from peritoneal involvement, as enlarged mesenteric lymph glands or with a combination of all these features. Tuberculosis of the peritoneum and gastrointestinal tract can mimic a variety of abdominal diseases[17].

The terminal ileum and ileocaecal junction are the most common sites, but like Crohn's disease any part of the gastro-intestinal tract can be affected and pathologically it may either be an ulcerative form with caseation, perforation and internal fistulae, or a hypertrophic form with fibroblastic reaction leading to intestinal stricture and obstruction.

Patients with chronic abdominal tuberculosis are often malnourished, anaemic with a distended abdomen. Sometimes patients may present with crampy abdominal pain and diarrhoea. Examination of the abdomen may reveal a vague lump - which may be rolled up omentum, enlarged lymph nodes or matted loops of bowel with omentum.

A barium meal follow-through showing localized areas of irregular, distorted mucosal folds and areas of dilated and strictured bowel loops indicate a tuberculous process. Confirmation of diagnosis is made either from culture of ascitic fluid or from tissue obtained following a laparoscopy or colonoscopy or laparotomy depending on the available resources. However, in some patients therapeutic trial with antituberculous medication may be necessary on the basis of strong clinical suspicion.

Antituberculous chemotherapy is the mainstay of treatment for abdominal tuberculosis.

Surgery in the form of resection of the involved bowel or by-pass is performed in acute presentations or in those cases where medical treatment fails[17,18].

21.12 AMOEBIASIS

Amoebiasis caused by *Entamoeba histolytica* has a wide distribution but is particularly prevalent in tropical countries and tends to occur with more severity, partly due to poor sanitation and poor nutritional states. Of all human parasitic diseases, amoebiasis carries one of the highest death rates and it is estimated to be responsible for approximately 100,000 deaths annually worldwide.

The disease is transmitted through water and food contaminated with *E. histolytica* cysts.

Invasive amoebiasis presents in two distinct forms - intestinal affecting colon and rectum and extraintestinal involving liver, lung, brain and skin.

The majority of patients present with symptoms of dysentery and only in severe fulminating colitis does the patient present with features of acute abdomen due to perforation of the amoebic ulcer and peritonitis. Invasive amoebiasis may also present as an amoebic appendicitis and again, perforation can lead to peritonitis. Amoebomas are inflammatory thickenings usually involving the caecum and characterized by firm to hard granulomatous lesion. It is important to be aware of this condition, which although rare, may be mistaken for caecal cancer leading to unnecessary resection[19].

Apart from the typical features of acute dysentery and systemic toxicity, a diagnosis of intestinal amoebiasis is confirmed by finding amoebic trophozoites in the stool specimen.

Amoebic trophozoites from the colon may invade the liver parenchyma via the portal vein and cause multiple micro-abscesses or a single large abscess usually in the right lobe of the liver. The clinical presentation here is less acute than in pyogenic liver abscess, but sometimes they can be difficult to differentiate and amoebic serology, if available, is useful in diagnosis.

Delayed presentation can lead to rupture of the abscess either in the peritoneal cavity or to adjacent viscera such as lung, into pleural or pericardial cavity. Uncomplicated amoebic liver abscess can be managed conservatively with metronidazole and ultrasound guided aspiration. Operative management is required in cases when conservative therapy fails or for complications such as rupture[20].

Cutaneous amoebiasis fortunately rare now, is a rapidly progressing painful ulceration of the skin - often around the tube following the previous practice of open drainage of a liver abscess.

Penile amoebic granuloma, although rare, has been described and like caecal amoeboma need to be distinguished from penile cancer to avoid mutilating surgery. Metronidazole by the oral or intravenous route, depending on the severity of the case, is the treatment of choice and is very effective.

Peritonitis from colon perforation needs laparotomy. Aggressive resection of bowel should be avoided as it is associated with high mortality. Closure of perforation, diversion colostomy or exteriorisation of the affected loop is recommended.

21.13 CHAGAS' DISEASE (megacolon; megaoesophagus)

Chagas' disease is caused by *Trypansoma cruzi* and is restricted to Latin America. Surgical problems occur in the form of megaoesophagus and megacolon in the chronic stage of the disease due to the destruction of submucosal parasympathetic ganglion cells and reduction in nerve fibres. The surgical management of both megaoesophagus and megacolon may be complicated by associated Chagasic myocarditis.

The diagnosis is based on serologic tests- complement fixation, immunofluorescent antibody, haemagglutination, and ELISA. In future use of PCR technology for the diagnosis is expected to replace the serologic tests.

The chief complaints of megaoesophagus (achalasia) are dysphagia, regurgitation leading to aspiration pneumonia, discomfort and weight loss.

The treatment of this condition ranges from forceful dilatation, cardiomyotomy (Heller's operation) to oesophagectomy with oesophagoplasty. Recently treatment with local injection of botulinum toxin has been reported from Brazil[21].

Progressive obstipation is the main presenting feature of the chagasic megacolon, manifested in the form of abdominal distention, meteorism, tenesmus and occasional pseudodiarrhoea. The two main complications of this disease are faecaloma and sigmoid volvulus.

Initial treatment for mild to moderate disease is with high fibre diet, stool softeners and laxatives. Digital evacuation may be required in patients with faecaloma. Severe cases would require resection of the colon with primary or delayed reconstruction[22].

21.14 SCHISTOSOMIASIS

Schistosomiasis, caused by trematodes of the genus *Schistosoma*, is one of the most prevalent helminthic infections found in the tropics and is estimated to affect over 500 million people. Infection takes place when cercariae, shed into the fresh water by the snail intermediate host, penetrate the skin of an individual while in the water. The cercariae mature into adult worms and normally live within the venules of intestine or bladder depending on the species of schistosome. Adult worms may live in the human body as long as 20-30 years and the granulamatous changes (bilharzial pseudotubercles) produced from their eggs in tissues throughout the body are responsible for the human pathology.

21.14.1 Schistosomiasis of the bladder

The principal clinical manifestation of urinary tract schistosomiasis is haematuria due to lesions in the bladder wall from eggs of *S.haematobium*.

The mucosa of the bladder on cystoscopy, may show tubercles, sandy patches, or granuloma.. The fibrotic changes and bladder calcification from chronic infection result in contracted bladder and ureteric stricture, when it affects the ureter. These in turn lead to hydronephrosis.The association between bladder cancer and urinary schistomiasis is based on evidence from the increased rate of bladder cancer in endemic areas of urinary schistosomiasis, and squamous cell cancer – resulting from chronic irritation and perhaps activation of inactive carcinogens by the presence of the worms.

21.14.2 Portal hypertension

Portal hypertension occurs as a result of portal fibrosis following fibrotic reactions to the schistosome eggs in the liver. Adult *S.mansoni* in the inferior mesenteric veins and *S. japonicum* in the superior mesenteric veins, are particularly associated with this clinical picture.

The diagnosis of schistomiasis depends on typical clinical features in an endemic area such as haematuria in infections with *S.haematobium* and haematemesis or ascites in *S.mansoni* or *S. japonicum* infections. Laboratory tests include identification of eggs in the urine, bladder

mucosal biopsy in *S.haematobium* and eggs in the faeces and rectal biopsy in case of *S.mansoni* and *S. japonicum*. An intradermal skin test using schistome antigen has been found to be a fairly sensitive immunological test provided the patient has been infected over 4-8 weeks. The circum-oval precipitation test (formation of a precipitate around isolated schistosome eggs in the presence of sera from infected individuals) has been found to be useful for determining success of treatment , as the test becomes negative after all the eggs in the tissue have been killed[23].

Once the diagnosis is made the patient can be treated with anthelmintics such as praziquantel (all species), oxamniquine (*S.mansoni*), or metrifonate (*S. haematobium*) .

Surgical treatment for urinary schistosomiasis depends on the extent of the pathology. A contracted bladder is treated with either augmentation cystoplasty or in advanced cases with cystectomy and urinary diversion e.g., ileal conduit. Ureteric stricture may be treated by uretero-neocystostomy or replacement of the ureteric segment with a loop of ileum.

Bladder cancer requires a partial or total cystectomy.

21.15 HYDATID DISEASE

The surgical manifestations of hydatid disease occur in cystic forms due to infestation by a canine tapeworm *Echinococcus granulosus* as opposed to the more lethal the alveolar forms due to *E.multilocularis* which found in the Northern Hemisphere.
Cysts occur more often in the liver followed by lungs and spleen. Rarely it may occur in other organs. The onset of symptoms depends on the variable rate of growth of the cyst and its effect on the surrounding organ. However, severe anaphylactic reaction followed by death can occur from rupture of the cyst in the pleural or peritoneal cavity.
A hydatid cyst in the liver usually remains without symptoms unless it becomes large or with complications of rupture either within the biliary tree producing obstructive jaundice or in the peritoneal cavity as mentioned above.

Similarly a lung cyst may only be found during a routine chest x-ray. If the cyst compresses or ruptures within the bronchial tree, symptoms of fever,chest pain, cough, and haemoptysis may occur. Rupture may also lead to expectoration of hydatid contents and may result in a lung abscess from secondary bacterial infection. Rupture into the pleural cavity may cause pneumothorax, empyema and anaphylactic shock as previously mentioned.

While a positive serologic test provides confirmation of the disease (or previous exposure), the diagnosis of lung cyst is made by a chest x-ray. Regarding abdominal hydatid cysts, ultrasonography and computed tomography are the best diagnostic options.

A completely calcified cyst does not require treatment. Albendazole is used in preoperative and postoperative period to prevent or reduce the risk of recurrence.
Percutaneous ultrasound guided aspiration of the liver cyst followed by injection of equal volume of hypertonic saline is the preferred method of treatment as opposed to open surgery[24].

21.16 ASCARIASIS

Ascaris lumbricoides is one of the largest of human parasites and ascariasis has a worldwide distribution affecting over a billion people[25]. It is commoner in areas of poor sanitation and is thus more prevalent in developing regions. The adult worms normally live within the small bowel where they cause minimal damage. However, they can produce such serious problems as acute small bowel obstruction arising from a heavy worm load and intertwining of worms with bolus formation. Adult worms can also migrate into the common bile duct leading to either obstructive jaundice, liver abscess or acute pancreatitis[26]. They may penetrate the small intestinal wall which can lead to peritonitis or they may block the appendix giving rise to a simulated appendicitis or cause tracheal obstruction (especially in small children) which can lead to death by asphyxiation.

The diagnosis of worms causing obstruction is often made at operation, but occasionally straight x-ray of the abdomen may show shadows of the worms.

An initial trial of conservative treatment with nasogastric suction, intravenous fluids and antispasmodics may be successful in early obstruction without clinical features of peritonitis.

Failure of conservative treatment or clinical features of complete obstruction with or without peritonitis require laparotomy. At laparotomy breaking and milking of the bolus of worms into the large bowel is tried first and if successful avoids need of enterotomy and extraction of worms followed by either closure or resection of the affected bowel segment.

All patients should be treated with an anthelmintic such as piperizine, pyrantel, mebendazole or albendazole following recovery from the acute attack.

Conservative treatment is also indicated in patients with jaundice or acute pancreatitis and endoscopic retrograde cholangio-pancreatography is reserved for failure to resolve on conservative therapy.

21.17 HELMINTHIC APPENDICITIS

Ascaris lumbricoides (the large intestinal roundworm), *Enterobius vermicularis* (threadworm/pinworm), *Trichuris trichiura* (whipworm) and proglottids of the Beef Tapeworm (*Taenia saginata*) can all at times be found in the appendix where they may result in a simulated appendicitis – usually diagnosed in the specimen after surgery.

21.18 NODULAR WORMS

Nodular worms of the genus *Oesophagostomum*, a hookworm relative, may cause nodular granulomas and abscesses in the colon, occasionally in stomach and in small intestine. This may result in perforation and peritonitis or may mimic ileocaecal carcinoma or tuberculosis leading to surgical laparotomy[27].

21.19 FILARIASIS

Filariasis, commonly caused by the filarial nematodes *Wuchereria bancrofti*, and *Brugia malayi*, blocks the lymphatic system in its chronic phase. As a result there is lymph stasis with the lower limbs and scrotum being more frequently affected. The condition once started, progresses with further deterioration from fibrosis of the lymphatic vessels due to recurrent lymphangitis. The resulting thickening of the subcutaneous tissue is described as elephantiasis. In many areas, a more common presentation is hydrocoele with or without a history of recurrent attacks of epididymo-orchitis. The passage of milky urine, known as chyluria, can occur from obstruction of the renal lymphatics.

It is important to realise that filariasis is not the only cause of elephantiasis and a diagnosis of filariasis should be confirmed by examining blood films taken between 10pm and 2am to demonstrate the motile, sheathed microfilaria. Serological tests have now been developed and, if available, can be most useful in diagnosis.

Treatment with anthelmintic drugs such as diethylcarbamazine or ivermectin often helps to reduce hydrocoeles, but is ineffective as treatment for elephantiasis of scrotum or legs.

Early elephantiasis of legs can be treated by reduction of lymphoedema with intermittent pneumatic compression and graduated compression stockings exerting pressure of 40mm Hg (class-4).

In advanced cases surgery is indicated. The operative procedures described fall under two categories:

- Excisional procedure in which the thickened skin and subcutaneous tissue are excised down to the deep fascia and epidermal grafts obtained from the excised skin is placed on the raw area,
- Lymphatic reconstruction by direct lymphatic-venous anastomosis or by burying a skin flap deep.

Long-term results of the above procedures are unsatisfactory as recurrence is common.

The treatment of chyluria is also very unsatisfactory due to poor results from operative procedures to disconnect renal lymphatics, although there are reports with successful outcome following surgical ablation[28]. Sclerotherapy with 5% silver nitrate solution instilled through a ureteric catheter in the affected kidney to obliterate the pyelolymphatic channels, has been used.

21.20 CONCLUSION

The surgical workload in the tropical and developing countries is variable and can be demanding at times. Surgical presentations of patients span a range of very common to rare kinds of emergency and elective clinical conditions. Malignancies almost always present in an advanced state.

There are diverging opinions on the question of how to better equip doctors in isolated hospitals to deal with such situations through surgical training[29].

A surgeon working in the tropical region of a developing country must have good basic surgical skills and an innovative mind. He has to be prepared for times of frustration, with failure from late presentation of otherwise curable diseases to the lack of basic and essential medical equipments. The reward to the surgeon is almost celestial when a patient leaves the hospital after a successful surgical procedure.

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