**<u>Acute abdominal pain in the tropics</u>**
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Bruce C. Steffes, MD, MBA, MA, FACS, FWACS, FCS(ECSA), FICS

Description:  The principles of abdominal surgery are not different in Africa, but the presentation, available diagnostic tools and frequency of certain etiologies is significantly different. The approach and therapy of peptic ulcer disease, small bowel obstruction, large bowel obstruction and parasitic disease in the abdomen will be the primary diseases discussed.

Session Problem: For the uninitiated, abdominal crisis due to tropical diseases and conditions can be frightening. Diagnostic, physiological and treatment principles must be applied in a unique fashion and this session is designed to increase awareness and to give principles which will help in the treatment of these patients.

Session Solution: To increase awareness of presentation, diagnosis and treatment of these abdominal conditions which are usually found only (predominantly) in the tropics.

Educational objectives:
Formulate a differential diagnosis for the African presentation of small and large bowel obstruction.
Modify first-world treatment plans for peptic ulcer disease and its complications for the realities of resource-limited areas.

I.      Introduction
   A.   Surgery in the developing world is different
            1.   Different diseases or at least different prevalence
            2.   Advanced pathology
            3.   Fewer care givers
            4.   Limited resources
   B.   Diseases seen less commonly in the developing world
            1.   Diveriticulitis
            2.   Acute and chronic cholecystitis (marked variation)
            3.   Appendicitis
            4.   Small bowel obstruction due to adhesions
   C.   Diseases seen more commonly in the developing world
            1.   Primary peritonitis
            2.   Perforated duodenal ulcers
            3.   Volvulus
            4.   Adult intussusception
            5.   Tuberculous peritonitis
            6.   Pigbel
   D.   What are diagnoses for laparotomy seen in the two-thirds world?
            1.   University Hospital in Ghana[i]

a) Appendicitis
b) Perforated typhoid
2. Bongolo Mission Hospital, Gabon[ii]
a) Incarcerated/strangulated hernias
b) Appendicitis
c) Volvulus
d) Adhesive SBO
e) Perforated typhoid
3. Tenwek Mission Hospital[iii]
a) Volvulus
b) Appendicitis
c) Perforated PUD
d) Trauma
e) Perforated typhoid
f) SBO
E. Differential diagnosis of small bowel obstruction in 2/3 world
1. Hernias
2. Ascariasis (often regional)
3. Abdominal tuberculosis
4. Adhesive SBO
a) Lower incidence than in N. America but Increasing in areas where abdominal surgery is available
b) May be due to pelvic inflammatory disease if STDs are high
5. Volvulus
6. Intussusception
a) Adults are more common in children in series from Nigeria, Uganda and S. Africa (remembering cases in children > 3 years old and adults are more likely associated with surgical pathology)
b) Colocolic intussusception is uncommon but more common in areas with high rate of amebiasis.
c) Hydrostatic barium reduction is often not available; Consider pneumatic reduction[iv] (the recommended pressure of air insufflation should not exceed 120 cm of water) or saline reduction (recommended maximum100 mm Hg). [v]
a) Reduction should be tried only if the patient is stable without peritonitis, shock, small bowel intussusceptions, etc.
F. Differential large bowel obstruction
1. Volvulus – both sigmoid and cecal
a) 'Volvulus belt' which includes countries in the Middle East, Africa, the Indian subcontinent, Turkey and South America where it accounts for almost half of all large bowel obstructions.
2. Lower frequency of colon cancer and diverticulitis

3. Increased prevalence of lymphogranuloma venereum rectal stricture – but still uncommon

II. Abdominal Surgical Emergencies

    A. Perforated Peptic Ulcer

        1. Case Presentation – 42 year old man with a history of occasional smoking and occasional alcohol intake presented with acute abdominal pain, more severe in the upper quadrant. He doesn't present for 16 hours after onset. His examination shows tachycardia, tachypnea and a nearly rigid abdomen.

        2. Surgical treatment

           a) Graham omental patch (open or laparoscopic)

           b) Rarely definitive surgery – only if minimal contamination

           c) Occasionally observation with NG and antibiotics if no peritonitis and no leak by water-soluble study

           d) Surgery >12 hours after perforation has much increased morbidity/mortality

        3. Medical Treatment

           a) Metronidazole and Clarithromycin resistance is increasing. Both protocols are about 80% effective if no resistance

           b) Cost and availability of drugs determine preferred regimen

           c) Triple therapy (for 14 days)

               (1) Bismuth 525 mg qid

               (2) Metronidazole 250 qid

               (3) Tetracycline 500 qid or amoxicillin 500 mg qid

               (4) PPI (omeprazole or lansoprazole) – improves response by 10%

           d) Alternative (for 10 - 14 days)

               (1) Clarithromycin 500 mg bid

               (2) Amoxicillin 500 mg qid

               (3) Omeprazole 20 mg bid

    B. Pigbel (With appreciation to Dr. Jim Radcliffe[vi] and Dr. Jeremy Stone)

        1. Case Presentation – 5 year old boy from from Papua New Guinea. Pig feast 5 days before. Now with severe abdominal pain 4 days during with fever, nausea & diarrhea. Intermittent cramps, especially with eating & drinking. WBC 14,400. Abdomen initially soft. Dx? Tx? Treated with NPO, nasogastric tube and antibiotics. "Dark" NG output, "dark" diarrhea and abdomen became "surgical". Dx? PIGBEL

        2. Pigbel (Enteritis Necroticans, Necrotizing Enteritis) was reported first in medieval Europe and again in Germany after WWII when it was called "darmbrand" (gut-fire). It resurfaced in the early 1960s in Goroka, PNG with culture-positive cases and was the most common cause of death in children >24 months). At one time, Pigbel was the most common cause of acute abdominal pain in the PNG Highlands (prior to vaccination was the most common cause for abdominal laparotomy in that area)

3. Incidence of Pigbel
    a) Male > Female (2.2:1)
    b) Males encouraged to eat more protein for strength
    c) Most commonly in children after the first year of life (70% <10 years old) but can be seen in young adults (25%)
    d) Maternal IgA likely protects infants
    e) More common in dry season (better weather leads to more frequent pig feasts)
4. Bacteriology of Pigbel
    a) Clostridium Perfringens Type C (also known as Clostridium welchii) = Anaerobic, gram +, spore-forming rod
    b) C.P. found in human stool, pig stool and soil
    c) Type A commonly causes food poisoning
    d) Spores are heat stable up to 95° C (Boiling point of water is 95° in the Highlands)
    e) C.P. grows in protein food in the intestine and produces a toxin (B-toxin)
    f) B-toxin is rapidly degraded by intestinal proteases in well-nourished people
    g) The toxin attacks the intestinal lining and causes inflammation and necrosis and may also cause arterial thrombosis
    h) C.P. in bowel can spread to liver and spleen
5. Predisposing Factors
    a) Malnutrition (especially low protein diet), Ascaris worms, Sweet Potato (Kaukau), High Protein Meal such as pig feasts (1/2-4 days before presentation)
    b) Malnutrition causes a decrease in the pancreatic production of all proteases including trypsin, a key enzyme in the digestion of meat and protein and the toxin
    c) Ascaris and kaukau cause high levels of heat stable trypsin inhibitors
    d) Sporadic high protein meals provides growth medium for C.P.
    e) Poorly cooked pork/meat or spillage of pig's bowel contents in mumu preparation
    f) Unwashed hands and feet of food handlers
    g) Malnutrition (decreases trypsin) + Kaukau (anti-trypsin) + Ascaris (anti–trypsin/dysmotility) (up to 75% of the Highlands diet)
    h) High Protein meal
6. The Clinical Course - Four Types of Pigbel:
    a) Mild Diarrheal (type IV)
        - May go undiagnosed or diagnosed as gastroenteritis (GE)

- Usually only diarrhea but can progress to Type 3
- Mortality: Rare
   b) Subacute Surgical (type III)
   - Presents later
   - Complication of Type II (See next category)
   - Mortality: 49%
   c) Acute Surgical (type II)
   - Present with ileus, small bowel obstruction (SBO), strangulation, perforation, peritonitis
   - Mortality: 42%
   d) Acute Toxic (type I)
   - Fulminant toxemia and shock
   - Usually young children
   - Mortality: 85% (Some deaths before presenting to hospital)
7. The Clinical Course
   a) Symptoms usually become apparent 48 hours after a large meat or protein meal
   b) Can present as late as a week later
   c) Present with colicky or constant abdominal pain, vomiting with dark emesis (blood flecks), blood in stool, foul flatus (meteorism), and diarrhea early
   d) Tachycardic, febrile, dehydrated, tender & distended upper abdomen with visible bowel, guarding, rigidity, decreased bowel sounds
   e) Pain > diarrhea (differentiates from gastroenteritis)
   f) Normal bowel peristalsis may force intestinal contents through the diseased neighboring portions of bowel causing colicky pain
   g) May be hungry but eating increases pain
   h) Later they can have SBO, malnutrition, fibrosis, adhesions, malabsorption and strictures. (Type III)
   i) If bowel resection needed, mortality 50%
   j) Mortality due to peritonitis, septicemia, dehydration, electrolyte abnormalities, and shock
8. Diagnostic Approach
   a) Serological test possible, ? availability (immuno-florescence using type C coated silicon beads)
   b) Culture C.P. from stool (anaerobic blood agar)
   c) Gas in bowel wall or SBO on abdominal x-ray
   d) Neutrophilic leukocytosis (>/= 20,000)
   e) Bloody ascites on ultrasound
   f) Bloody NG aspirate or blood in stool
9. Early recognition of pigbel and quick action are of utmost importance. The toxin begins attacking the bowel instantly and constantly. Timely recognition

and treatment may reduce severity or even prevent death of the child! Early fluid resuscitation, decompression of SB, and appropriate antibiotics may preclude need for laparotomy.  If severe, early referral and surgery may prevent death.

10. Treatment of Pigbel
    a) Correction of fluid and electrolyte deficits; hydrate well; correct moderate to severe anemias.
    b) Nasogastric drainage
    c) Intravenous antibiotics - CMP, Crystalline PCN and Metronidazole/Tinidazole (+/- Gentamicin)
    d) Treatment of Ascaris
    e) ?treatment of malaria
    f) Consider hyperalimentation or TPN if course prolonged
    g)  Antiserum +/- (Not readily available or effective)

11. Early Hospital course
    a) If improves (decreased swelling, pain, fever, pulse, white count, NG aspirate, abdominal tenderness and vomiting; increased hunger; normal bowel movements), wait 24 hours then oral rehydration solution (ORS)→ milk → solids
    b) After 48 hours , laparotomy if there if failure to improve:  high NG output, persistent SBO by x-ray, persistent peritonitis, high white count, persistent fever

12. Surgery
    a) Due to the rapid progression of pigbel, the decision for surgery is often a judgment call by the surgeon based on clinical experience
    b) Urgent laparotomy with wide resection of SB to normal margins
    c) End-to-end anastomosis vs. Two ostomies
    d) Usually 50-200 cm (2-4 feet) of jejunum need resected
    e) Questions that arise:
        (1) How much bowel to resect?
        (2) Which patients to do second look?

13. Surgery Findings
    a) Palpable loops of thick bowel
    b) Enlarged mesenteric nodes typical
    c)  "Tiger Striping"
    d) "Skip Lesions"
    e) Mucosal Ulcerations
    f) Perforations/SBO

14. Post Op Care
    a) Strict I & O, Adequate fluid resuscitation and good nursing care.
    b) Attention to the CBC (transfusion is needed) and K+ levels
    c) Nutritional supplement

15. Pathology
    a) Blood and pus in stool (from "sloughing" enteritis of jejunum, ileum and colon)
    b) Transmural infection of the bowel (patchy segmental ulcerative necrosis)
    c) Gas gangrene, separation of the layers of the bowel wall, pseudomembranes
    d) Affects Jejunum > ileum > cecum > colon
16. Prevention of Pigbel
    a) Type C toxoid immunization.  Inactivated toxin: 0.5 cc given at 2, 4 and 6 months of age with the DPT vaccine.  Protects 2-4 years
    b) Was used from 1980- mid 1990's and cases were 1/5 of pre-immunization levels,  When the PNG government felt it was too expensive (and quit paying for it), it became an orphan drug and the manufacturer quit making it.
    c) In one recent study, 6 of 25 non-immunized kids had pre-existing antibodies to C.P. type C indicating that the organism is still common.
17. Prevention of Pigbel
    a) Changes in dietary habits (Less reliance on sweet potatoes and more regular protein)
    b) Changes in cooking methods (higher temperatures) and better preservation of food
    c) Changes in hygiene and food preparation
    d) Education through PHC and CBHC
    e) Eradication of Ascaris
    f) Reinstitution of vaccination program!
C. Typhoid fever
   i. Salmonella enterica serovar typhi (previously known as Salmonella typhi), a pathogen specific only to humans, as well as by certain non-typhoid salmonella (NTS), particularly Paratyphoid strains A, B, C.
   ii. Location and Prevalence:  16-30 million cases per year, almost exclusively in the developing world, with an overall mortality rate of 10%. These waterborne gram negative aerobes are associated with poor sanitation and fecal contamination of water and food supplies.
   iii. Clinical features of typhoid infection
      1. Classically a four week disease
         a. Weeks one and two: fever, headache, abdominal pain
         b. Week three: "typhoidal state" with disordered mentation and toxemia
         c. Week four: Defervescence and improvement
      2. Lab: leucopenia/thrombocytopenia are common
         a. Widal test: very controversial

b. Conclusion of a paper by Tupasi et al (*[Phil J Microbiol Infect Dis 1991, 20(1):23-26]* "Culture isolation of Salmonella typhi from blood and bone marrow should be considered the standard diagnostic test to confirm typhoid fever. A single Widal test in an endemic area is of no diagnostic value. In addition, it should not be used as a screening test in asymptomatic individuals. Neither should a "negative" Widal test rule out the diagnosis of typhoid fever in patients with signs and symptoms of the disease since a "negative" Widal test may be seen early in the course of illness. The Widal test should not also be used as the basis for deciding the duration of antimicrobial therapy."

iv. Indications for surgery in enteric fever

1. Surgery for carrier state is NOT a usual indication. Normally, do only for chronic cholecystitis per se (doesn't always work for carrier state)
2. Hemorrhage (1.5 - 10% of patients, bleeding usually in 3 or $4^{th}$ week, usually UGI in type and may be hard to find if in the small intestine)
3. Perforation (1 - 5% of patients, common in the second and third weeks of illness, but can be much later. Some patients perforate without an obvious prodrome)
   a. Mortality for perforation is as high as 40%, affected by many factors in the austere environment.
   b. Indications for surgery:
      i. Pneumoperitoneum on x-ray (may require left lateral film)
      ii. Persistent palpable mass (especially with erythema of abdominal wall)
      iii. Diffuse peritonitis or positive peritoneal tap
      iv. Persistent sepsis/failure to improve on medical therapy
   c. Suspicious of abdominal catastrophe but negative x-rays? Do frequent examinations (by the same or equally experienced examiner) and x-rays (q. 6 h at first) until improvement or perforation is evident.

v. Surgical options after vigorous resuscitation and appropriate antibiotics

1. Aggressive resuscitation prior to OR with appropriate antibiotic coverage (triple antibiotics to cover GI flora as well as Salmonella)
   a. Ampicillin and chloramphenicol are no longer the drugs of choice. Fluoroquinolones (?decreasing efficacy) and third generation cephalosporins are probably the best at present.
2. Oversew with interrupted vertical mattress sutures if 1 – 3 perforations. Perforations are in the distal ileum (rarely proximally) and on the antimesenteric border. They are usually single (70%) but can be

multiple.  Need of excision of the ulcer edges is debatable.  Look for areas "about to perforate" and consider plicating those.

3. Resection if multiple lesions close to each other or tissue quality is poor
4. Aggressive peritoneal debridement/irrigation of peritoneal cavity
5. Consider use of retention sutures.  Leave skin open.
6. Consider second-look operation
7. A negative laparotomy is rare and better tolerated than a missed perforation.

    vi.   Typhoid cholecystitis

1. Acute cholecystitis – very uncommon
2. Predominance in children?
3. Often advanced (gangrene or perforation) because of low index of suspicion

D. Infectious Bowel obstruction

1. Ascaris – especially in children
   a) Problems arise when they migrate and when they don't
   b) Migratory problems
      (1) Loeffler's syndrome (non-surgical)
      (2) Biliary-pancreatic
      (3) Biliary colic
      (4) Acalculous cholecystitis
      (5) Acute cholangitis
      (6) Chronic cholangiohepatitis (especially when associated with Oriental liver flukes)
      (7) Acute pancreatitis
      (8) Hepatic abscess
      (9) Anastomotic perforation
   c) Other surgical conditions
      (1) Intussusception
      (2) Acute appendicitis
   d) Non-migratory – Bowel obstruction
      (1) Diagnosis –
         i. prevalence varies widely by locale and age
         ii. May have history of recent Antihelminthic treatment
         iii. Physical examination
         iv. Plain x-ray
         v. Contrast studies – especially with Gastrografin ®
         vi. Ultrasound and CT scan
      (2) Treatment
         i. Operative vs. nonoperative management
            1. Does the patient have peritonitis?
            2. Is the patient toxic?

3. How long has the child had symptoms?

3. How long has the child had symptoms?
4. Does X-ray suggest complete SBO?
5. Is the child worsening on nonoperative treatment?

    ii. Medical:
1. decompression and antihelminthics (mebendazole or albendazole versus piperazine). Some prefer no treatment at all.
2. ?Hypertonic saline enemas
3. Effective with about 75%

(3) Surgical –
    i. Milk the worm bolus through into colon if possible.
    ii. If not effective, transverse enterotomy and removal of worm bolus with primary closure. Remember to prevent spills into peritoneal cavity (bacterial infection), close anastomotic line securely (to prevent worm migration) and use antibiotic prophylaxis.
    iii. 1/3 will require resection

(4) Educate family and village

E. Volvulus
1. Sigmoid Volvulus
    a) Facts
        (1) Wide age range
        (2) More common in males (most series 2:1)
        (3) Most common form of GI volvulus
    b) History
        (1) Rapid onset of pain and remarkable distention
        (2) Obstipation
        (3) Prior episodes
        (4) Nausea/vomiting as obstruction persists
    c) Diagnosis
        (1) Massive distention
        (2) Empty rectum
        (3) Characteristic X-ray
            i. Massive distention of colon "bent inner-tube" sign (aka "coffee bean sign," "horse's butt").
            ii. +/- small bowel distention
            iii. Empty left iliac fossa
            iv. No rectal gas
    d) Management
        (1) Resuscitation
        (2) Antibiotics

              (3) Barium enema, colonoscopy or CT scan for diagnosis of uncertain cases

              (4) If no peritonitis, urgent rigid or flexible sigmoidoscopy with rectal tube placement (sutured).  Some studies suggest flexible sigmoidoscopy/colonoscopy has a higher success rate.

              (5) Urgent laparotomy for signs of peritonitis or failed sigmoidoscopy

                    i. Controversy over primary anastomosis and Hartmann's procedure.

              (6) Semi-elective laparotomy after successful endoscopic detorsion with placement of rectal tube (guide wire helps)

                    i. Sigmoidopexy has a high recurrence rat

                    ii. Low morbidity and mortality rate for semi-elective resection and anastomosis

2. Cecal Volvulus
    a) Clinical characteristics
        (1) Similar presentation with sigmoid volvulus
        (2) More common in females
        (3) Preoperative diagnosis much more challenging
        (4) Gangrene of colon is common
    b) Diagnosis
        (1) X-ray: Suggest obstruction. 1 in 5 are diagnostic
        (2) Barium enema: high rate of accuracy but challenging without fluoroscopy.  Bird's beak seen at point of volvulus.
        (3) CT scan
        (4) Colonoscopy: difficult in unprepared colon and there is high risk of perforation
    c) Surgical options
        (1) Decompression alone has 50% recurrence
        (2) Resection and ileostomy with mucus fistula: choice for septic/unstable patients with comorbid conditions
        (3) Resection (partial or complete right colectomy) with anastomosis +/- colopexy
        (4) Detorsion and cecopexy: At times an option if gangrene is not present
        (5) Detorsion and cecostomy: most authors feel this procedure should be abandoned as complication rates can be 50%

3. Small bowel volvulus with or without sigmoid volvulus ("double volvulus" or "compound volvulus")
    a) Often with necrotic gut.  In that case, detorsion is difficult, confusing and potentially dangerous.  Resection in situ with staplers (if possible) and then sorting out the ends has been my usual approach

b) Ileostomy should be avoided if at all possible

c) Small – bowel volvulus: Short gut syndrome is one possible result but not usually. Second look laparotomy should always be considered for questionable bowel in setting of possible short gut syndrome

d) In ability to achieve detorsion or the lack of immediate improvement after endoscopic detorsion of presumed sigmoid volvulus should lead the surgeon to the OR so as not to miss small bowel volvulus

F. Primary peritonitis in previously health children

1. Clinical features

a) Very rare problem in the West

b) Disease of children, especially girls, ages 6-10

c) Females much more common than males

d) Cause is not understood

2. Presentation

a) Rapid onset (usually <48 hours)

b) Fever and leukocytosis with severe abdominal pain

c) Tenderness often most severe in RLQ

d) Very difficult to distinguish from acute appendicitis

3. Treatment

a) Exploratory laparotomy with appendectomy and washout (nosurgeon wants to sit on a case strongly suggesting acute appendicitis)

b) Laparoscopy with washout (if equipment available)

c) Broad spectrum antibiotics until culture results available

d) Some authors have suggested a paracentesis with strep species or Pneumococcus on Gram stain would obviate the need for laparotomy.

4. Bacteriology

a) Strep species (especially pneumococcus) are most common in most reports

b) E. coli

c) Mixed aerobe/anaerobe

5. Summary: Primary peritonitis

a) A disease causing a rapid onset of an acute abdomen with vomiting and diffuse peritonitis, especially in girls, which usually resolves quickly after surgery for diagnosis and antibiotic therapy.

**Contact e-mail: bruce.steffes@paacs.net**

**Endnotes:**

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[vi] Radcliff, J; J Stone "Pigbel" PowerPoint presentation loaned by Dr. Radcliff.