CASE 5- Toy & Liu CASE FILES: Surgery

A 63-year-old male complains of a 6-month history of difficulty voiding and feeling as though he cannot empty his bladder completely. After voiding, he often feels as though he needs to urinate again. He denies a urethral discharge. He has mild hypertension and takes a thiazide diuretic. His only other medication is ampicillin prescribed for two urinary tract infections during the past year. On examination, his blood pressure is 130/84 and his pulse rate 80/min; he is afebrile. Findings from examinations of the heart and lungs are normal, and the abdomen reveals no masses.

What is the most likely diagnosis?

What is the best therapy for this patient?
ANSWERS TO CASE 5: Benign Prostatic Hypertrophy

Summary: A 63-year-old hypertensive male complains of a 6-month history of difficulty voiding and feeling as though he cannot empty his bladder completely. He has experienced two episodes of cystitis. He denies dysuria or urgency and does not have a urethral discharge.

♦ Most likely diagnosis: Benign prostatic hypertrophy (BPH).

♦ Best therapy: Transurethral prostatectomy (TURP).

Analysis

Objectives

1. Know the clinical presentation of BPH.
2. Know the differential diagnosis for urinary outlet obstruction in males and when a biopsy is appropriate.

Considerations

The prostate gland is the male reproductive organ that is positioned at the base of the bladder and completely encircles the urethra as it exits the bladder and before it becomes part of the penile urethra. The physiologic function of the prostate is to produce the ejaculate, which serves as a vehicle for spermatozoa. As the male ages, the prostate increases in size. This increase in size can have consequences, as the human prostate is the only mammalian prostate with a capsule. The capsule restricts expansion of the prostate gland as BPH progresses. The bladder neck and prostatic urethra become compromised in their function, leading to a condition known as bladder outlet obstruction.

Symptoms of BPH, known as prostatism, include irritative and obstructive symptoms. They can include frequent urination of small amounts, a feeling of incomplete voiding with subsequent attempts to urinate to achieve the feeling of bladder emptying, slow urinary flow, voiding at night after sleep (nocturia), hesitancy at the beginning of
urinary flow, and in its extreme form, complete urinary retention. Several conditions that produce similar symptoms mimic BPH. Urethral stricture disease (a narrowing of the urethra with scarring), urinary tract infection, including infection of the prostate (prostatitis), prostate cancer, and neurologic conditions affecting the control and strength of bladder contraction all mimic and may be indistinguishable from BPH. When there is nodularity or an elevation in the prostate-specific antigen (PSA), biopsy of the prostate is generally indicated.

APPROACH TO URINARY OUTLET OBSTRUCTION

Definitions

Micturition: The physiologic act of voiding. This involves contraction of the detrusor (bladder muscle) followed by relaxation of the bladder neck and other urinary sphincters to allow unrestricted, complete emptying of the bladder in a single setting.

Digital rectal examination (DRE): The prostate is palpated with a gloved examining finger inserted into the rectum. The normal prostate has the “feel” of the thenar eminence of the thumb (Figure 5–1).

Prostate-specific antigen: A blood protein normally produced by the prostate. PSA is specific to the prostate but not to a particular condition of the prostate because age, size, infection, and cancer are among the several reasons why PSA values can be elevated.

Urodynamics: Testing performed on the function of the bladder in both its filling and emptying phases, which may be as simple as voiding into a specially developed toilet to measure the voiding flow rate to as complicated as the placement of a catheter into the urinary bladder to measure pressures and volumes during filling and emptying.

Clinical Approach

When faced with the vague symptomatology of prostatism, the initial duty of the physician is to exclude other etiologies because the treatment would differ. This exclusion process begins with obtaining a history and looking for associated signs and symptoms of other disease
processes. A review of systems should entail a search for neurologic abnormalities. A urinalysis is the cornerstone of laboratory testing to exclude the presence of a urinary tract infection or microscopic hematuria that might indicate a bladder tumor. PSA blood testing should be performed as well as determination of the serum creatinine level to rule out severe prostatism with renal compromise. A DRE not only characterizes the size of the prostate but also is performed to exclude the presence of a palpable nodule suggestive of prostate cancer (Figure 5–1). Even the best history and physical and laboratory testing may not discriminate between bladder outlet obstruction secondary to BPH and a urethral stricture because both of these pathologic entities are secondary to restriction of the urethra. If a patient requires urodynamic testing in cases where the diagnosis is not clear, consultation with a urologist is generally helpful. Making matters more difficult, prostatism may coexist with a urinary tract infection and/or a neu-
rologic disease such as Parkinson disease. Evidence of renal compromise, an elevated serum creatinine value and/or urinary retention, multiple small voids with incomplete emptying, and/or a palpable bladder on physical examination call for urgent urologic intervention.

Once the correct diagnosis of BPH has been made, initial treatment is often medical. Two classes of medication are available for the management of prostatism. The first class are alpha agonist agents, which cause relaxation of the prostate smooth muscle, thereby increasing the functional diameter of the urethra. Another class of medication used in the management of prostatism causes a reduction in prostate size by blocking a metabolite of testosterone, thus leading to the involution of prostate glandular tissue and shrinkage of the overall prostate size. When medical therapy fails, surgical intervention, which serves to destroy prostate obstructing tissue, is used. The standard operative procedure is known as transurethral resection of the prostate, or TURP. This procedure is carried out transurethrally using a specially developed scope that has attached to it a cutting element with water irrigation. “Chips” of the prostate are carved out from within the prostate urethra and removed via the scope. Alternative methods to destroy prostate tissue include the use of a laser, radiofrequency waves, or microwaves. Rarely, the prostate enlarges to such a size that open surgical removal known as a suprapubic prostatectomy is required. Regardless of the method of therapy chosen to manage BPH, the patient needs to be monitored thereafter for response to therapy as residual glandular tissue will continue to grow.

Comprehension Questions

[5.1] A 57-year-old asymptomatic male is noted to have a prostate that is normal in shape and size on rectal examination. His PSA level is 18 ng/mL (normal < 2.5 ng/mL). What is the best next step for this patient?

A. Observation
B. Transrectal ultrasound examination with a prostate biopsy
C. Repeated PSA testing in 6 months
D. Initiate finasteride therapy
[5.2] A 72-year-old man has a lower abdominal mass and constantly dribbles urine. Which of the following is the best next step?

A. Computed tomography (CT) scan of the pelvis
B. Enema
C. Placement of a Foley catheter
D. Referral to a general surgeon and a neurologist

[5.3] A 58-year-old commercial airline pilot has confirmed prostatism. He is being treated by a doctor but seeks emergency room treatment for dizziness, which precludes his flying. What is the most likely problem?

A. Unrecognized Parkinson disease
B. Undiagnosed metastatic prostate cancer
C. Drug side effect
D. Silent renal failure

[5.4] A 42-year-old male requests prostate “testing” because his father has recently been given a diagnosis of prostate cancer. You perform a digital rectal examination, which reveals a normal-sized, smooth prostate gland. A PSA test is then performed and is run stat because the patient insists on knowing the results before leaving the office. The PSA result is 3.2 ng/mL (normal < 2.5 ng/mL). Which of the following is the best next step?

A. CT scan of the abdomen and pelvis for a workup for prostatic cancer
B. Sonographically directed prostate biopsy
C. Repeated PSA test
D. Prostatectomy with possible lymphadenectomy

Answers

[5.1] B. A substantially elevated PSA value in this patient generally requires a prostate biopsy to assess for prostate cancer. Transrectal sonography is performed to help determine the location of the biopsy.
Overflow incontinence occurs when the urinary bladder is filled to capacity. As the pressure rises, with standing and coughing, a small amount of urine leaks out of the bladder through the restricted bladder outlet in a dribbling fashion. A small amount of urine is seen to squirt from the penis as the Valsalva maneuver pushes on the massively distended bladder. Immediate urinary drainage and hospitalization are in order.

The alpha agonist class of medications, originally developed for blood pressure control, relax the smooth muscle within the arterial wall, leading to a decrease in blood pressure that may result in dizziness and/or syncope (fainting). Patients must be warned of this side effect. Titration and nighttime dosing are often required.

Mild elevations of the PSA value may be seen immediately after a DRE. The best course in this case is to repeat the PSA test several days to 1 week later.

**CLINICAL PEARLS**

- Patients with symptoms suggestive of BPH should undergo a renal function test (creatinine), a PSA test, urinalysis, and a digital rectal examination.
- The International Prostate Symptom Score can characterize voiding symptoms based on a patient’s report of incomplete emptying, frequency, intermittency, urgency, weak stream, straining, and nocturia.
- Although there is no physiologic relationship between BPH and prostate malignancy, the age of onset of these two clinical entities overlaps.
- Distinguishing characteristics of prostate cancer include a firm, hard, and/or misshapened prostate gland on examination and/or an elevated or elevating PSA value. Both BPH and prostate malignancy can coexist in the same patient.
- The diagnosis of prostate cancer is made with transrectal biopsy of the prostate.
REFERENCES

CASE 6

A 43-year-old man presents with a 16-hour history of intermittent, crampy abdominal pain and bilious vomiting. He states that the symptoms began approximately 3 hours after lunch on the previous day, improved after vomiting, but returned after 1 to 2 hours. He had a bowel movement shortly after the onset of the pain, but there has been no passage of flatus or stool since then. The patient denies any similar episodes previously and has no current medical problems. He underwent exploratory laparotomy for a gunshot wound to the abdomen 3 years previously. On examination, his temperature is 100.5°F, pulse rate 105/min, blood pressure 140/80, and respiratory rate 24/min. The abdomen is distended, with a well-healed midline surgical scar. The abdomen is tender throughout with no masses or peritonitis. The bowel sounds are hypoactive with occasional high-pitched rushes. No hernias are identified. A rectal examination reveals no masses and no stool in the rectal vault. Laboratory studies reveal normal electrolyte levels. His white blood cell (WBC) count is 16,000/mm³ with 85% neutrophils, 4% bands, 10% lymphocytes, and 1% monocytes; the hemoglobin and hematocrit values are 18 g/dL and 48%, respectively. The serum amylase value is 135 IU/L. An abdominal radiograph was obtained (Figure 6–1).

◆ What is your next step in management?

◆ What are the complications associated with this disease process?

◆ What is the probable therapy?
Figure 6–1. Abdominal radiographs in the supine (A) and upright (B) positions show a dilated small bowel with air-fluid levels. (Reproduced, with permission, from Kadell BM, Zimmerman P, Lu DSK. Radiology of the abdomen, Zinner MJ, Schwarz SI, Ellis H, et al, eds. Maingot’s abdominal operations, 10th ed. New York: McGraw-Hill, 1997:24.)
ANSWERS TO CASE 6: Small Bowel Obstruction

Summary: A patient has signs, symptoms, and radiographic evidence of a high-grade mechanical small bowel obstruction.

- **Next step in management:** Place a nasogastric (NG) tube to decompress the stomach, begin fluid resuscitation, and place a Foley catheter to monitor urine output and assess his response to the fluid resuscitation.

- **Complications associated with this disease process:** Mechanical small bowel obstruction may lead to strangulation, bowel necrosis, and sepsis. Vomiting may result in aspiration pneumonitis. When unrecognized or untreated, intravascular fluid loss (from third-space fluid loss and vomiting) can lead to prerenal azotemia and acute renal insufficiency.

- **Probable therapy:** Exploratory laparotomy after fluid resuscitation.

Analysis

Objectives

1. Learn the clinical and radiographic features associated with mechanical small bowel obstruction and strangulating or complicated disease processes.
2. Learn the management strategy for mechanical small bowel obstruction.

Considerations

An otherwise healthy 43-year-old man who has undergone previous abdominal surgery presents with typical signs and symptoms associated with mechanical small bowel obstruction. Because of the previous abdominal operation, the cause of the obstruction is most likely related to
postoperative adhesions. The change in pain pattern from intermittent to persistent is a concern. **Persistent pain** in this setting can be produced by severe bowel distension (which may produce venous congestion, decreased bowel perfusion, and necrosis) or **bowel ischemia secondary to strangulation**. Other features of this patient’s presentation suggesting the presence of a complicated bowel obstruction include fever, tachycardia, leukocytosis, an elevated serum amylase level, and radiographic signs of a high-grade small bowel obstruction. Mechanical obstruction of the bowel results in the accumulation of fluid in the bowel lumen and bowel wall, in addition to extravasation of fluid into the peritoneal cavity. The net result of these fluid shifts is a depletion of intravascular volume and decreased perfusion of all organs. Therefore, **one of the most vital aspects of treatment is early recognition of the problem and restoration of the intravascular volume to reestablish organ perfusion**. Restoration of intravascular volume is critical in this patient prior to operative therapy because the induction of general anesthesia in a volume–depleted individual may lead to profound hypotension. Nonoperative therapy is frequently successful for mechanical small bowel obstruction due to adhesions, however, this approach is inappropriate in a patient exhibiting signs and symptoms consistent with existing or impending bowel ischemia and/or necrosis. The most appropriate management in this case consists of NG tube placement to prevent further vomiting and potential aspiration, fluid resuscitation, administration of broad-spectrum antibiotics, and urgent laparotomy.

**APPROACH TO SMALL BOWEL OBSTRUCTION**

Definitions

**Strangulating small bowel obstruction:** Mechanical obstruction leading to bowel ischemia and/or necrosis. This process may result from strangulation due to an abdominal wall hernia or an internal hernia, with high-grade and/or complete obstruction.

**Ileus:** Distension of the small bowel and/or colon from nonobstructive causes. Common causes include local or systemic inflammatory or infectious processes, a variety of metabolic de-
rangements, recent abdominal surgery, and adverse effects of medications.

**Internal hernia:** A congenital or acquired defect within the peritoneal cavity that can lead to small bowel obstruction.

**Gallstone ileus:** Mechanical obstruction of the small bowel due to large gallstone(s) in the bowel lumen. This condition generally occurs when a stone or stones in the gallbladder enter the adjacent duodenum. The typical clinical presentation is characterized by intermittent bowel obstruction for several days until the stone lodges in the distal small bowel and causes complete obstruction.

**Clinical Approach**

Mechanical small bowel obstruction is a common clinical problem. The cause of the obstruction, treatment considerations, and the approach to the disease differ based on the patient’s age, the duration of symptoms, and whether or not the patient has a history of abdominal operation or trauma. An obstruction in a neonate, an infant, or a young child is most likely the result of a hernia, malrotation, meconium ileus, Meckel diverticulum, intussusception, or intestinal atresia. In contrast, a small bowel obstruction in an adult is most frequently due to an adhesion, a hernia, Crohn’s disease, gallstone ileus, or a tumor. As a mechanical small bowel obstruction prevents the passage of small bowel luminal contents, the patient develops cramplike abdominal pain, nausea, and bilious vomiting. It is not uncommon for patients to describe the occurrence of a bowel movement at the onset of an acute obstruction, and this is generally due to the stimulation of peristalsis with evacuation of the distal gastrointestinal tract contents. Hence, the presence of a bowel movement does not rule out bowel obstruction. Whenever the small bowel obstruction is nearly complete or complete (high grade) there may be a cessation of flatus and stool passage following the initial bowel movement. The recommended approach to patient evaluation and treatment is outlined in Figure 6–2.

**Physical Examination**

The physical examination of a patient with small bowel obstruction may initially reveal a low-grade fever and tachycardia as a result of dehydration and inflammatory changes. The persistence of tachycardia
Patient with suspected small bowel obstruction

History & Physical, Laboratories, urinalysis, abdominal radiograph

Suspicious for large bowel obstruction

CT scan

Small bowel obstruction and no colon obstruction

No large or small bowel obstruction

Large bowel obstruction

Treat ileus

Treatment

Operative treatment (yes)

Incarcerated hernia present

CT scan (no)

History of abdominal operation? (yes)

Selective treatment based on findings

Indications of complication? (indicator present)

Resuscitation and early operative treatment

Initial trial of nonoperative management

Incarcerated hernia present (no)

Figure 6–2. Algorithm for the management of small bowel obstruction. CT, computed tomography.
after the completion of fluid resuscitation may suggest unresolved inflammation from small bowel ischemia and/or necrosis. Similarly, the early presence of a high fever should raise a suspicion of bowel ischemia or pulmonary complications due to aspiration. In most patients abdominal examinations will reveal mild, diffuse tenderness. Nonspecific tenderness that improves following successful decompression by the placement of an NG tube is observed commonly in patients with an uncomplicated obstruction. Localized tenderness directly over distended bowel loops suggests the presence of severe distension or bowel ischemia; although a worrisome finding, this localized tenderness is not specific for ischemia. A digital rectal examination of patients with small bowel obstruction often reveals little or no stool in the rectal vault, which is due to continued peristalsis and evacuation of stool from the distal bowel. The finding of a large amount of stool in the rectum is unusual and may suggest ileus rather than mechanical obstruction as the cause of distension.

Pathophysiology

Mechanical obstruction of the small bowel leads to diminished absorptive function and the accumulation of luminal fluid. Additionally, there is a fluid shift into the extravascular space due to local inflammatory stimulation and venous congestion. As the obstruction continues, transudative fluid loss into the peritoneal cavity occurs. These losses, along with vomiting, generally produce tremendous intravascular volume depletion and place untreated patients at risk for the development of remote organ dysfunction due to hypoperfusion. Generally, patients with proximal small bowel obstruction have more frequent vomiting, and those with more distal obstruction have more distension and less vomiting. With long-standing distal small bowel obstruction, bacterial overgrowth can develop and lead to feculent vomiting. Prolonged distal small bowel obstruction can lead to further intra-abdominal and pulmonary (aspiration) infectious complications.

Laboratory and Radiographic Evaluations

The initial laboratory evaluation should include a complete blood count with a differential count, serum electrolyte and amylase determinations,
urinalysis, and arterial blood gas studies (for selected patients). With dehydration and a physiologic response to bowel obstruction, patients with uncomplicated small bowel obstruction may initially present with mild leukocytosis (WBC count 10,000 to 14,000/μL) and a left-shifted differential. Generally, the leukocytosis resolves with therapy. **Persistent leukocytosis after hydration should raise a suspicion of complications** and may mandate early surgical intervention or an additional diagnostic evaluation. An elevation in the serum amylase level is most commonly associated with pancreatitis but may also develop with complicated small bowel obstruction.

Plain radiographs of the abdomen are generally obtained to evaluate all patients with suspected bowel obstruction. These usually reveal dilated small bowel with or without colonic air. These findings are not pathopneumonic for obstruction and may also be observed in the setting of ileus. Not uncommonly, radiographs of an advanced obstruction demonstrate a fluid-filled bowel with a paucity of air rather than a dilated bowel. Similarly, patients with an obstruction involving the proximal small bowel may have radiographs showing little or no air-filled bowel.

Additional Radiographic Studies

A **computed tomography (CT) scan may be useful** for patients in whom the etiology is obscure, such as those with a functional obstruction (ileus), inflammatory bowel disease, a tumor, or gallstone ileus. CT scans can reliably identify the transition from dilated to decompressed bowel, which is diagnostic for mechanical obstruction. In addition, CT imaging may be useful in visualizing peritoneal tumor spread (carcinomatosis), primary small bowel tumors, Crohn’s disease, gallstone ileus, and clinically obscure hernias. Alternatively, contrast radiography such as upper gastrointestinal and small bowel follow-through (UGI/SBFT) can be used to differentiate between mechanical obstruction and ileus or to assist in determining the location and severity of a bowel obstruction. It is important to bear in mind that CT scanning and UGI/SBFT require the administration of contrast into the bowel lumen and can aggravate patient vomiting and may contribute to aspiration. **The goals in patient evaluation are to diagnose the bowel obstruction and to identify factors that may indicate the presence**
of a strangulated bowel. Table 6–1 lists some of the more commonly used indicators to identify bowel strangulation.

Treatment

Patients with uncomplicated partial small bowel obstruction from adhesions can be initially treated with a trial of nonoperative therapy consisting of nothing by mouth (NPO), placement of an NG tube, close monitoring of fluid status, serial clinical examinations, and laboratory and radiographic follow-up. Most patients who are successfully treated non-operatively demonstrate improvement within 6 to 24 hours after the initiation of treatment. These improvements include a decrease in abdominal discomfort and distension, a decrease in the volume of NG aspirate, and radiographic resolution of bowel distension. The absence of early improvement with nonoperative treatment should prompt further evaluation with a CT scan or UGI/SBFT to confirm the diagnosis and/or further define the obstruction for possible surgical therapy. When operative treatment is determined to be necessary, perioperative broad-spectrum antibiotics are administered to prevent wound and intra-abdominal infectious complications. Operative therapy for

Table 6–1

INDICATORS SUGGESTIVE OF STRANGULATED SMALL BOWEL OBSTRUCTION

<table>
<thead>
<tr>
<th>HISTORY</th>
<th>PHYSICAL EXAMINATION</th>
<th>LABORATORY FINDINGS</th>
<th>RADIOGRAPHY‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant pain*</td>
<td>Localized tenderness*</td>
<td>Leukocytosis*</td>
<td>Complete obstruction* †</td>
</tr>
<tr>
<td>Constipation*</td>
<td>Fever*†</td>
<td>Elevated amylase level†</td>
<td>Fluid-filled bowel*</td>
</tr>
<tr>
<td></td>
<td>Tachycardia*†</td>
<td>Elevated lactate level†</td>
<td>Thickened bowel wall*</td>
</tr>
<tr>
<td></td>
<td>Peritonitis*†</td>
<td></td>
<td>Mesentery edema (CT)*†</td>
</tr>
<tr>
<td></td>
<td>Tender mass*†</td>
<td></td>
<td>Free-fluid (CT)*†</td>
</tr>
</tbody>
</table>

*Nonspecific (ie, may occur without strangulation).
†Not sensitive (ie, may not occur with strangulation).
‡CT, computed tomography.
adhesive small bowel obstruction consists of careful exploration and identification of the obstruction source. Adhesive bands responsible for the obstruction are divided, and ischemic or necrotic bowel is resected.

Early Postoperative Small Bowel Obstruction

Early postoperative small bowel obstruction is characterized by symptoms developing within 30 days following an abdominal operation. This condition can result from narrowing of the lumen because of mechanical causes or ileus. An exact determination of the cause is generally not necessary because nonoperative observation is the usual treatment for both. A CT scan may be useful in some patients to identify or rule out an intra-abdominal infection as the cause.

Outcome

The mortality associated with small bowel obstruction has improved over the past 50 years with improved medical technology and supportive care. Despite this overall improvement in patient outcome, there continues to be a significant increase in morbidity and mortality associated with complicated small bowel obstruction. Therefore, one of the major goals in patient treatment is early diagnosis and treatment of uncomplicated small bowel obstruction to prevent a progression to strangulation and bowel necrosis. Patients with a high-grade bowel obstruction or suspected of having strangulated bowel should undergo prompt resuscitation and early operative therapy, which may prevent the development and/or progression of bowel necrosis.

Comprehension Questions

[6.1] A 79-year-old woman who has had no previous abdominal surgery presents with intermittent bowel obstruction symptoms of 1 week's duration and persistent vomiting for the past 1 day. Her physical examination does not reveal any hernias and is consistent with that of distal small bowel obstruction. She is afebrile. Her WBC count is 4000/µL. What is the most appropriate next step?
A. Attempt nonoperative treatment for 48 hours.
B. Perform upper gastrointestinal tract endoscopy.
C. Proceed with an immediate exploration laparotomy.
D. Perform a CT scan.

[6.2] Which of the following situations is most likely to respond to non-surgical management?

A. A 72-year-old woman with a bowel obstruction due to mid-gut volvulus
B. Small bowel obstruction due to gallstone ileus
C. A 45-year-old female who has small bowel obstruction after open gall bladder surgery 20 days previously
D. A 2-day-old male who has small bowel obstruction due to jejunal atresia

[6.3] A 67-year-old white male arrives in the emergency room with nausea and vomiting following an appendectomy performed 25 days previously. He is afebrile. The abdomen is slightly tender and distended. The WBC count is 18,000/μL. Electrolyte studies reveal a sodium level of 140 mEq/L, potassium 4.2 mEq/L, chloride 105 mEq/L, and bicarbonate 14 mEq/L. Which of the following is the best therapy for this patient?

A. Placement of an NG tube and observation
B. Colonoscopy for possible intussusception
C. A barium enema to relieve a volvulus
D. Surgical therapy

Answers

[6.1] D. Patients without previous abdominal surgery or hernias who present with symptoms and signs of bowel obstruction may benefit from CT imaging (to identify possible malignancy, gallstone ileus, or internal hernia).

[6.2] C. Early small bowel obstruction (within 30 days) following abdominal surgery is generally due to adhesions or persistent in-
flammation that frequently resolves with non-surgical therapy (hospitalization, nothing by mouth, intravenous hydration).

[6.3] **D.** The patient has anion gap acidosis as evidenced by the low bicarbonate level, which is probably caused by lactic acid, reflecting ischemic bowel or severe fluid depletion. Elderly patients often have a minimum of symptoms and are afebrile. Surgical therapy may be indicated if CT imaging confirms intraabdominal sepsis or high grade obstruction.

### CLINICAL PEARLS

- A significant proportion of patients with small bowel obstruction can be treated conservatively (NPO, placement of an NG tube, close monitoring of fluid status, serial clinical examinations, and laboratory and radiographic follow-up), while constantly being assessed for bowel ischemia or strangulation.
- Persistent pain, fever, tachycardia, leukocytosis, an elevated serum amylase level, and radiographic signs of high-grade small bowel obstruction are often signs of complicated bowel obstruction and the need for surgical therapy.
- Computed tomography imaging plays an important role in patient evaluation. The exceptions to this rule include patients with simple adhesive obstruction and an absence of indicators of complicated small bowel obstruction (Table 6–1), as well as patients in whom early operative intervention is clinically indicated.

### REFERENCE

CASE 7

A 34-year-old diabetic woman complains of a 6-month history of progressive numbness and pain in her right hand that wakes her up at night. She states that her thumb is especially affected. She says that she is beginning to drop objects she is carrying in her right hand. She denies a history of trauma, exposure to heavy metals, or a family history of multiple sclerosis. The only medication she takes is an oral hypoglycemic agent.

◆ What is the most likely diagnosis?

◆ What is the mechanism of the disorder?

◆ What is your next step?
ANSWERS TO CASE 7: Carpal Tunnel Syndrome

Summary: A 34-year-old diabetic woman complains of a 6-month history of progressive numbness and pain in her right hand occurring especially at nighttime and affecting her thumb. She states that she is beginning to drop objects that she carries in her right hand.

✦ Most likely diagnosis: Carpal tunnel syndrome.

✦ Mechanism of the disorder: Median nerve compression.

✦ Next step in therapy: Nighttime splint and nonsteroidal anti-inflammatory drugs (NSAIDs).

Analysis

Objectives

1. Know the clinical presentation, pathophysiology, and risk factors for carpal tunnel syndrome.
2. Know the medical and surgical options for treating carpal tunnel syndrome.

Considerations

The distribution of the progressive numbness and pain is suggestive of median nerve compression. In addition, exacerbation of the patient’s symptoms at night is typical of carpal tunnel syndrome. The mechanism of this disorder is compression of the median nerve as it passes within the carpal tunnel. This causes axonal damage and narrowing of the nerve. Median nerve compression causes numbness and pain in the thumb, index finger, and middle and lateral aspects of the ring finger. The median nerve may be compressed anywhere along its length from the brachial plexus down to the hand, but the most common site of compression is within the carpal tunnel, where it is dorsal to the transverse
The carpal canal is a rigid structure that causes physiologic dysfunction by producing median nerve ischemia. The best initial management is a nighttime splint for the wrist and avoidance of excess activity with the hand.

**APPROACH TO CARPAL TUNNEL SYNDROME**

**Definitions**

- **Carpal tunnel syndrome:** Median nerve compression at the wrist leading to paresthesias of the radial three fingers and sometimes hand weakness.
- **Tinel sign:** Reproduction of the patient’s symptoms by percussion of the median nerve at the wrist.
- **Electrophysiologic studies:** Investigation of nerve conduction and muscle innervation.

**Clinical Approach**

The carpal canal serves as a mechanical conduit for the digital flexor tendons. The walls and floor on the dorsal surface of the canal are formed by the carpal bones, and the ventral aspect is confined by the strong, inelastic, transverse carpal ligament. The smallest cross-sectional area of the canal is created by extremes of flexion and extension of the wrist (Figure 7–1). Exacerbation of symptoms at night is thought to be caused by edema; tenosynovitis may also be present. Carpal tunnel syndrome has been associated with endocrine conditions, diabetes, myxedema, hyperthyroidism, acromegaly, and pregnancy. Other causes are autoimmune disorders, lipomas of the canal, bone abnormalities, and hematomas. The etiology is often multifactorial. Females are more commonly affected in a ratio of approximately three to one.

The diagnosis of carpal tunnel syndrome is clinical, and the symptoms are typical. The exertion of direct digital pressure by the examiner over the median nerve at the carpal tunnel frequently reproduces the symptoms in about 30 seconds. In the Phalen maneuver, gravity-induced wrist flexion also produces the classic symptoms of this condition. A positive Tinel sign is present when direct percussion over the
nerve reproduces paresthesia. Sensory loss, particularly vibration sense, and motor loss may be present with thenar muscle wasting and decreased abductor muscle resistance. Electrophysiologic studies may be helpful. A comparison of median and ulnar or median and radial sensory stimulation values at the wrist is useful in confirming the diagnosis. Radiographs, including a "carpal tunnel view" are recommended in order to detect arthritis or fractures. Computed tomography and magnetic resonance imaging are rarely needed.

Conservative therapy consists of the use of splints and nonsteroidal anti-inflammatory agents. Splints should be light and should hold the wrist in a neutral or slightly extended position. Local steroid injections are effective in 80% to 90% of patients, but symptoms tend to return after months or sometimes years. Injections should not be given more frequently than on two or three occasions per year. Care must be taken not to inject directly into the median nerve. Surgery is indicated for intractable symptoms that are refractory to medical management. It consists of complete division of the transverse carpal ligament extending distally from the ulnar side of the median nerve. The results of surgery are generally good. Poor results are usually associated with either a

Figure 7–1. The carpal tunnel. The wrist in cross section reveals that the median nerve is susceptible to impingement.
misdiagnosis or failure to completely divide the ligament. The surgery can be performed with an open or an endoscopic approach. A tourniquet is used to exsanguinate the limb, and the operative field is infiltrated with a local anesthetic agent such as Xylocaine; in addition, intravenous sedation can be used. The Palmer fascia and the ligament are divided vertically from the proximal end of the carpal tunnel to its most distal point, and a wide separation of the ends of the ligament is observed (Figure 7–2). The underlying median nerve is carefully protected. A small tissue flap is left attached to the hook of the hamate, and

Figure 7–2. Carpal tunnel release. The transverse carpal ligament is incised.
the skin is closed. Postoperatively the wrist is splinted in slight extension for about 2 weeks.

The potential advantages of the endoscopic approach are less discomfort, minimal scarring, a shorter period of immobilization, and a more rapid recovery. Persistent or recurrent symptoms should be investigated by repeated electrophysiologic studies and by exclusion of other causes of nerve compression. Occasionally, the ulnar nerve is compressed at the wrist, but more commonly compression of this nerve occurs in the fibromuscular groove posterior to the medial epicondyle.

**Comprehension Questions**

[7.1] A 24-year-old medical student notes some numbness and tingling of her right hand. She states that primarily her little finger is affected. Which of the following is most likely to be the etiology?

A. Median nerve  
B. Radial nerve  
C. Ulnar nerve  
D. Long thoracic nerve

[7.2] Which of the following is most likely to be a risk factor for the development of carpal tunnel syndrome?

A. Diabetes insipidus  
B. Hypothyroidism  
C. Addison’s syndrome  
D. Fibromyalgia

**Answers**

[7.1] C. The sensory innervation of the little finger and the ulnar side of the ring finger is achieved with the ulnar nerve.

[7.2] B. Hypothyroidism (as well as diabetes mellitus, hyperthyroidism, pregnancy, acromegaly), is associated with carpal tunnel syndrome.
CLINICAL PEARLS

- Carpal tunnel syndrome usually involves pain to the radial three fingers, especially at night.
- The initial treatment of carpal tunnel syndrome includes administration of NSAIDs and the use of a wrist splint.
- Surgery is indicated when severe pain or progressive motor weakness occurs despite conservative measures.

REFERENCES

CASE 8

A 46-year-old woman presents with a 24-hour history of abdominal pain that began approximately 1 hour after a large dinner. The pain initially began as a dull ache in the epigastrium but then localized in the right upper quadrant (RUQ). She describes some nausea but no vomiting. Since her presentation to the emergency center, the pain has improved significantly to the point of her being nearly pain-free. She describes having had similar pain in the past with all previous episodes being self-limited. Her past medical history is significant for type II diabetes mellitus. On physical examination, her temperature is 38.1°C (99°F), and the rest of her vital signs are normal. The abdomen is nondistended with minimal tenderness in the RUQ. Findings from the liver examination appear normal. The rectal and pelvic examinations reveal no abnormalities. Her complete blood count reveals a white blood cell (WBC) count of 13,000/mm³. Serum chemistry studies demonstrate total bilirubin 1.8 mg/dL, direct bilirubin 0.6 mg/dL, alkaline phosphatase 140 U/L, AST 45 U/L, and ALT 30 U/L. Ultrasonography of the RUQ demonstrates stones in the gallbladder, a thickened gallbladder wall, and a common bile duct diameter of 4.0 mm.

◆ What is the most likely diagnosis?

◆ What is the best therapy?

◆ What are the complications associated with this disease process?
ANSWERS TO CASE 8: Gallstone Disease

Summary: A 46-year-old woman presents with a 1-day history of RUQ abdominal pain and a physical examination and laboratory findings suggestive of gallstone disease.

- **Diagnosis:** Cholecystitis, likely acute and chronic.

- **Best therapy:** Laparoscopic cholecystectomy is the preferred treatment for all patients with a reasonable life expectancy and no prohibitive risks for general anesthesia and abdominal surgery.

- **Complications:** Complications from gallstone disease include acute and chronic cholecystitis, pancreatitis, choledocholithiasis, cholangitis, and gallstone ileus.

**Analysis**

Objectives

1. Know the etiology of gallstone disease and learn the differences among biliary colic, acute cholecystitis, and chronic cholecystitis.
2. Know the basic diagnostic and therapeutic plans for patients with gallstone disease.
3. Become aware of the complications arising from gallstone disease.

Considerations

This patient provides a good history of recurrent RUQ abdominal pain episodes following meals, consistent with biliary colic. Although she demonstrates minimal tenderness to palpation in her right upper ab-
demen on physical examination, the elevated leukocyte count and ultrasound findings of gallbladder wall thickening are consistent with acute or chronic cholecystitis. If this patient had a normal WBC count and an ultrasound examination demonstrating stones in the gallbladder and no other abnormalities, the presentation would be consistent with biliary colic, which can be treated by elective cholecystectomy. For cholecystitis, the appropriate treatment consists of hospital admission, administration of intravenous antibiotics, and laparoscopic cholecystectomy prior to discharge from the hospital.

**APPROACH TO GALLSTONE DISEASE**

**Definitions**

**Biliary colic:** Also known as symptomatic cholelithiasis, it is characterized by waxing and waning postprandial epigastric or RUQ pain radiating to the back and normal transaminase levels. It is caused by gallbladder contraction stimulated by cholecystokinin secretion following food ingestion, gallstone obstruction at the gallbladder neck, or less commonly gallbladder dysfunction.

**Acute cholecystitis:** In 95% of patients, acute cholecystitis results from a stone or stones obstructing the cystic duct. Bacterial infection is thought to occur via the lymphatics, with the most commonly found organisms being *Escherichia coli*, *Klebsiella*, *Proteus*, and *Streptococcus faecalis*. Patients generally present with persistent RUQ pain, with or without fever, gallbladder tenderness, leukocytosis, and often mild, nonspecific elevated liver enzyme levels, which may or may not indicate common bile duct stones. Treatment includes hospital admission, administration of intravenous fluids, nothing by mouth, antibiotics directed at the above-listed organisms, and cholecystectomy during the hospitalization.

**Acalculous cholecystitis:** Gallbladder inflammation due to biliary stasis (in 5% of patients with acute cholecystitis) leading to gallbladder distension, venous congestion, and decreased perfusion; it nearly always occurs in patients hospitalized with a critical illness.
Chronic cholecystitis: Results from repeated bouts of biliary colic and/or acute cholecystitis leading to gallbladder wall inflammation and fibrosis. The patient may present with persistent or recurrent localized RUQ pain without fever or leukocytosis. Sonography may demonstrate a thickened gallbladder wall and a contracted gallbladder.

Cholangitis: Infection within the bile ducts, most commonly due to complete or partial obstruction of the bile ducts by gallstones or strictures. The classic Charcot triad (RUQ pain, jaundice, and fever) is seen in only 70% of patients. This condition may lead to life-threatening sepsis and multiple-organ failure. Treatment consists of antibiotic therapy, and supportive care; in cases of severe cholangitis, endoscopic decompression of the bile duct by endoscopic retrograde cholangiopancreatography (ERCP) or surgery is indicated.

Right upper quadrant ultrasonography: This technique has a 98% to 99% sensitivity in identifying gallstones in the gallbladder. The examination is also useful for measuring the diameter of the common bile duct, which can indicate the possible presence of stones in the common bile duct (choledocholithiasis). When present, common bile duct stones are visualized only 50% of the time with this imaging modality.

Biliary scintigraphy: The study of gallbladder function and biliary patency using an intravenous radiotracer. Normally the liver is visualized, followed by the gallbladder, followed by emptying of the radiotracer into the duodenum. Nonvisualization of the gallbladder in a patient with RUQ pain indicates gallbladder dysfunction due to acute or chronic cholecystitis.

Endoscopic retrograde cholangiopancreatography: Endoscopic common bile duct cannulation and direct injection of contrast material to visualize the duct. An endoscopic sphincterotomy in the duodenum during the procedure may facilitate bile drainage and the clearance of bile duct stones, which is especially useful in treating cholangitis and choledocholithiasis. The procedure requires sedation and may be associated with complication rates of 8% to 10%.
Pathophysiology

At least 16 million Americans have gallstones, and 800,000 new cases occur each year. Most stones are composed of pure cholesterol, mixed cholesterol, phospholipids, or pigmented stones. Patients with cholesterol stones can be shown to have supersaturated bile. The proportion of cholesterol, lecithin, and bile salts determines whether the cholesterol is maintained in solution or precipitates and results in stone formation. A small fraction, only about 20%, of all patients with gallstones have symptoms related to stones. While it is unknown why some patients with gallstones develop symptoms whereas others do not, it is clear that those who develop symptoms are at risk for the subsequent development of complications, including acute or chronic cholecystitis, choledocholithiasis, pancreatitis, and cholangitis.

Patient Evaluation and Treatment

The evaluation in every patient should consist of a history, a physical examination, a complete blood count, liver function studies, a serum amylase determination, and RUQ ultrasonography (Table 8–1). It is important to differentiate patients with biliary colic from patients with complicated gallstone disease, such as acute or chronic cholecystitis, choledocholithiasis, cholangitis, and biliary pancreatitis, because the management varies for these conditions. For example, a patient with choledocholithiasis may present with symptoms identical to those of biliary colic, but the condition may be differentiated on the basis of an elevation in serum liver enzyme levels and dilatation of the common bile duct by ultrasound. In contrast to patients with biliary colic, who are treated by elective cholecystectomy, patients with choledocholithiasis require in-hospital observation for the development of cholangitis and early endoscopic clearance of common bile duct stones, in addition to cholecystectomy. A major goal in patient evaluation is to make an accurate diagnosis without using unnecessary imaging and invasive diagnostic studies. Choledocholithiasis should be suspected if the RUQ ultrasound findings include a common bile duct diameter greater than 5 mm in the presence of elevated liver enzyme levels. Gallstone pancreatitis should be considered in the presence of significantly elevated amylase and lipase values.
Table 8–1
GALLSTONE DISEASE PRESENTATIONS*

<table>
<thead>
<tr>
<th>DISEASE</th>
<th>SYMPTOMS</th>
<th>PHYSICAL EXAMINATION</th>
<th>ULTRASONOGRAPHY</th>
<th>LABORATORY STUDIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biliary colic</td>
<td>Postprandial pain, usually &lt;6 h in duration</td>
<td>Afebrile, mild tenderness over gallbladder</td>
<td>Gallstones in gallbladder but no wall thickening, no CBD dilatation</td>
<td>Normal WBC count, normal LFT values, normal serum amylase level</td>
</tr>
<tr>
<td>Acute cholecystitis</td>
<td>Persistent epigastric or RUQ pain lasting &gt;8 h</td>
<td>May be febrile or afebrile; usually localized gallbladder tenderness</td>
<td>Gallstones in gallbladder; may have pericholecystic fluid; may or may not have CBD dilatation</td>
<td>Normal or elevated WBC count; may have normal or mildly elevated LFT values</td>
</tr>
<tr>
<td>Chronic cholecystitis</td>
<td>Persistent recurrent RUQ pain</td>
<td>Afebrile; may have localized tenderness over a palpable gallbladder</td>
<td>Stones in gallbladder, thickened gallbladder wall; in advanced cases contracted gallbladder</td>
<td>Normal WBC count; may have mild elevation in LFT values</td>
</tr>
<tr>
<td>Choledochoolithiasis</td>
<td>Postprandial abdominal pain that improves with fasting</td>
<td>May or may not be clinically jaundiced; nonspecific RUQ abdominal tenderness</td>
<td>Gallstones in gallbladder; CBD usually dilated</td>
<td>Elevation in LFT values; the pattern of elevation is dependent on the chronicity and partial versus complete obstruction</td>
</tr>
<tr>
<td>Biliary pancreatitis</td>
<td>Persistent epigastric and back pain</td>
<td>Epigastric tenderness to deep palpation is present</td>
<td>Gallstones in gallbladder; CBD dilatation may occur because of pancreatitis (does not always indicate CBD stones)</td>
<td>Leukocytosis, serum amylase level frequently &gt;1000 U/L, LFT values may be transiently elevated, but persistence may indicate CBD stones</td>
</tr>
</tbody>
</table>

*CBD, common bile duct; WBC, white blood cell; LFT, liver function test; RUQ, right upper quadrant.
Sometimes, acute and chronic cholecystitis may be difficult to differentiate clinically because in both cases patients may have localized tenderness over the gallbladder. When this situation arises, the patient should be treated as if they had acute cholecystitis. The treatment for both acute and chronic cholecystitis is cholecystectomy. The operation of choice is a laparoscopic cholecystectomy with or without cholangiography (radiopaque dye injected into the common bile duct and an radiograph taken). Some surgeons selectively perform cholangiograms if the common bile duct is dilated and liver enzyme levels are elevated. Other surgeons obtain cholangiograms with every laparoscopic cholecystectomy performed. Patients with gallstone pancreatitis are treated with bowel rest and intravenous hydration. When the pancreatitis clinically resolves, a laparoscopic cholecystectomy can be done. Generally, patients with uncomplicated biliary pancreatitis should undergo cholecystectomy during the same hospitalization. When cholecystectomy is delayed, 25% to 30% of patients may develop recurrent bouts of pancreatitis within a 6-week period.

**Comprehension Questions**

[8.1] A 65-year-old woman presents to the emergency room with post-prandial RUQ pain, nausea, and emesis over the last 12 hours. The pain is persistent and radiates to her back. She is afebrile, and her abdomen is tender to palpation in the RUQ. Sonography demonstrates cholelithiasis, gallbladder wall thickening, and a dilated common bile duct measuring 12 mm. Laboratory studies reveal the following values: WBC count 13,000/mm³, AST 220 U/L, ALT 240 U/L, alkaline phosphatase 385 U/L, and direct bilirubin 4.0 mg/dL. Which of the following is the most appropriate treatment at this time?

A. Admit the patient to the hospital, provide intravenous hydration, and check hepatitis serology values.
B. Admit the patient to the hospital and perform a laparoscopic cholecystectomy.
C. Admit the patient to the hospital, provide intravenous hydration, begin antibiotic therapy, and recommend ERCP.
D. Provide pain medication in the emergency room and ask the patient to follow up in the clinic.

[8.2] A 28-year-old woman undergoing an obstetric ultrasound during the second trimester of pregnancy and is found to have gallstones in her gallbladder. She claims to have had indigestion with frequent belching throughout her pregnancy. Which of the following is the most appropriate treatment?

A. A low-fat diet until the end of her pregnancy and then a postpartum laparoscopic cholecystectomy
B. Elective laparoscopic cholecystectomy during the second trimester
C. Follow-up after completion of her pregnancy
D. Open cholecystectomy during the second trimester

[8.3] Which of the following findings is most consistent with the diagnosis of acute cholecystitis?

A. Fever, intermittent RUQ pain, and jaundice
B. Persistent abdominal pain, RUQ tenderness, and leukocytosis
C. Intermittent abdominal pain and minimal tenderness over the gallbladder
D. Epigastric and back pain

[8.4] A 69-year-old man presents with confusion, abdominal pain, shaking chills, a rectal temperature of 94°F, and jaundice. An abdominal radiograph shows air in the biliary tree. Which of the following is the most likely diagnosis?

A. Acute cholangitis
B. Acute pancreatitis
C. Acute cholecystitis
D. Acute appendicitis
Answers

[8.1] **C.** Admission to the hospital, administration of intravenous fluids and antibiotics, and ERCP. This patient’s presentation is highly suggestive of cholangitis, with the presence of a significant elevation in her liver enzyme levels, common bile duct dilatation, and tenderness in the RUQ.

[8.2] **C.** Reevaluation after the completion of pregnancy is appropriate for this patient, who has stones in her gallbladder and symptoms that are most likely unrelated to gallstones and may be pregnancy-induced.

[8.3] **B.** Persistent abdominal pain, RUQ tenderness, and leukocytosis indicate acute cholecystitis. Choice A is most consistent with cholangitis; choice C is typical of biliary colic and choice D is consistent with acute pancreatitis.

[8.4] **A.** Elderly patients who present with fever (or hypothermia), jaundice, abdominal pain, and shaking chills often have acute cholangitis (purulent infection of the biliary tract). The presence of air in the biliary tree is consistent with this illness. This is a life-threatening condition and often requires urgent surgical or endoscopic decompression of the biliary system, in addition to aggressive supportive care, and broad-spectrum antibiotic therapy.
CLINICAL PEARLS

Cholecystectomy is generally not indicated unless there is a clear link between the patient’s symptoms and gallstones or if there is objective evidence of gallbladder dysfunction (e.g., a thickened gallbladder wall on ultrasonography, non-visualization of the gallbladder on biliary scintigraphy) or gallstone-related complications.

In general, the treatment of cholecystitis is hospitalization, administration of intravenous antibiotics, and a laparoscopic cholecystectomy prior to discharge from the hospital.

**Cholangitis, which can be diagnosed with the Charcot triad—RUQ pain, jaundice, and fever—is life-threatening.** Treatment consists of antibiotics therapy, supportive care, and in cases of severe cholangitis biliary duct decompression via ERCP.

Choledocholithiasis should be suspected if the RUQ ultrasound findings include a common bile duct diameter greater than 5 mm in the presence of elevated liver enzyme levels.

REFERENCES


CASE 9

A 38-year-old man presents at the emergency center with tarry stools and a feeling of light-headedness. The patient indicates that over the past 24 hours he has had several bowel movements containing tarry-colored stools and for the past 12 hours has felt light-headed. His past medical and surgical history are unremarkable. The patient complains of frequent headaches due to work-related stress, for which he has been self-medicating with six to eight tablets of ibuprofen a day for the past 2 weeks. He consumes two to three martinis per day and denies tobacco or illicit drug use. On examination, his temperature is 37.0°C (98.6°F), pulse rate 105/min (supine), blood pressure 104/80, and respiratory rate 22/min. His vital signs upright are pulse 120/min and blood pressure 90/76. He is awake, cooperative, and pale. The cardiopulmonary examinations are unremarkable. His abdomen is mildly distended and mildly tender in the epigastrium. The rectal examination reveals melenotic stools but no masses in the vault.

◆ What is your next step?

◆ What is the best initial treatment?
ANSWERS TO CASE 9: Upper Gastrointestinal Tract Hemorrhage

Summary: A 38-year-old man presents with signs and symptoms of acute upper gastrointestinal (GI) tract hemorrhage. The patient’s presentation suggests that he may have had significant blood loss leading to class III hemorrhagic shock.

◆ Next step: The first step in the treatment of patients with upper GI hemorrhage is intravenous fluid resuscitation. The etiology and severity of the bleeding dictate the intensity of therapy and predict the risk of further bleeding and/or death.

◆ Best initial treatment: Prompt attention to the patient’s airway, breathing, and circulation is mandatory for patients with acute upper GI hemorrhage. After attention to the airway, breathing, and circulation (ABC’s), the patient is prepared for endoscopy to identify the etiology or source of the bleeding and possible endoscopic therapy to control hemorrhage.

Analysis

Objectives

1. Be able to outline resuscitation and treatment strategies for patients presenting with acute upper GI tract hemorrhage and hemorrhagic shock.
2. Be familiar with most common causes of upper GI tract hemorrhage and their therapies.
3. Know the adverse prognostic factors associated with continued bleeding and increased mortality.

Considerations

The treatment of patients with suspected upper GI tract hemorrhage begins with an initial assessment to determine if the bleeding is acute or...
occult. Acute bleeding is recognized by a history of hematemesis, coffee-ground emesis, melena, or bleeding per rectum, whereas patients with occult bleeding may present with signs and symptoms associated with anemia and no clear history of blood loss. **A critical part of the initial evaluation is assessment of the patient’s physiologic status to gauge the severity of blood loss.** The sequence in the management of acute upper GI tract hemorrhage consists of (1) **resuscitation**, (2) **diagnosis**, and (3) **treatment**, in that order. In this patient’s case, his symptoms and physiologic parameters suggest severe, acute blood loss (class III hemorrhagic shock with up to 35% total blood volume loss) and should prompt immediate resuscitation with close monitoring of patient response (urine output, clinical appearance, blood pressure, heart rate, serial hemoglobin and hematocrit values, and consideration of central venous pressure monitoring). A nasogastric tube should be inserted following resuscitation to determine whether bleeding is active. The stomach should be irrigated with room-temperature water or saline until gastric aspirates are clear. For patients with **massive upper GI tract bleeding, agitation, or impaired respiratory status**, **endotracheal intubation is recommended prior to endoscopy.** Laboratory studies to be obtained include a complete blood count, liver function studies, prothrombin time, and partial thromboplastin time. A type and cross-match should be ordered. Platelets or fresh frozen plasma should be administered when thrombocytopenia or coagulopathy is identified, respectively. Early endoscopy has been shown to identify the bleeding source in patients with active ongoing bleeding and may achieve early control of bleeding. Given the history of nonsteroidal anti-inflammatory drug (NSAID) use, it would be appropriate to begin empirical therapy for a presumed gastric ulcer and gastric erosions with a proton pump inhibitor prior to endoscopic confirmation.

**APPROACH TO UPPER GI BLEEDING**

**Definitions**

**Mallory–Weiss tear:** A proximal gastric mucosa tear following vigorous coughing, retching, or vomiting. The bleeding is generally self-limiting, mild, and amenable to conservative management.
Dieulafoy’s erosion: Infrequently encountered, this problem describes bleeding from an aberrant submucosal artery located in the stomach. This bleeding is frequently significant and requires prompt diagnosis by endoscopy, followed by endoscopic or operative therapy.

Arteriovenous (AV) malformation: A small mucosal lesion located along the GI tract. Bleeding is usually abrupt, but the rate of bleeding is usually slow and self-limiting.

Esophagitis: Mucosal erosions frequently resulting from gastroesophageal reflux, infections, or medications. Patients most frequently present with occult bleeding, and treatment consists of correction or avoidance of the underlying causes.

Esophageal variceal bleeding: Engorged veins of the gastroesophageal region, which may ulcerate and lead to massive hemorrhage; related to portal hypertension and cirrhosis.

Clinical Approach

The sources of upper GI tract bleeding can be categorized as variceal versus non-variceal. Common sources of non-variceal bleeding include duodenal ulcers (25%), gastric erosions (25%), gastric ulcers (20%), and Mallory–Weiss tears (7%). Up to 30% of patients have multiple etiologies of bleeding identified during endoscopy. In addition, all studies indicate that a proportion of cases have no endoscopically discernible cause, and these cases are associated with an excellent outcome. Rare causes of upper GI tract bleeding include neoplasms (both benign and malignant), AV malformations, and Dieulafoy erosions. **Bleeding tends to be self-limited in approximately 80% of all patients with acute upper GI tract bleeding.** Continuing or recurrent bleeding occurs in 20% of patients and is the major contributor to mortality. The **overall mortality associated with upper GI tract bleeding is 8% to 10%** and has not changed over the last several decades. There are striking differences in the rates of rebleeding and mortality depending on the diagnosis at endoscopy (Table 9–1). **Patient mortality with acute upper GI tract bleeding increases with rebleeding, increased age, and in patients who develop bleeding in the hospital.** A number of clinical predictors and endoscopic stigmata have been identified with the development of recurrent bleeding, and these are listed in Table 9–2.
Table 9–1
RISK OF REBLEEDING BASED ON SOURCE

<table>
<thead>
<tr>
<th>SOURCE</th>
<th>REBLEEDING (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Esophageal varices</td>
<td>60</td>
</tr>
<tr>
<td>Gastric cancer</td>
<td>50</td>
</tr>
<tr>
<td>Gastric ulcer</td>
<td>28</td>
</tr>
<tr>
<td>Duodenal ulcer</td>
<td>24</td>
</tr>
<tr>
<td>Gastric erosion (gastritis)</td>
<td>15</td>
</tr>
<tr>
<td>Mallory–Weiss tear</td>
<td>7</td>
</tr>
<tr>
<td>No identified source</td>
<td>2.5</td>
</tr>
</tbody>
</table>


Table 9–2
FACTORS ASSOCIATED WITH INCREASED REBLEEDING AND MORTALITY

Clinical
- Shock on admission
- Prior history of bleeding requiring transfusion
- Admission hemoglobin <8 g/dL
- Transfusion requirement ≥5 units of packed red blood cells
- Continued bleeding noted in nasogastric aspirate
- Age greater than 60 y (increased mortality but no increase in rebleeding)

Endoscopic
- Visible vessel in ulcer base (50% rebleeding risk)
- Oozing of bright blood from ulcer base
- Adherent clot at ulcer base
- Location of ulcer (worse prognosis when located near large arteries, eg, posterior duodenal bulb or lesser curve of stomach)

The use of NSAIDs contributes to the development of NSAID-induced gastric ulcers. All NSAIDs produce mucosal damage. The risk of developing an ulcer is dose-related. Roughly, 2% to 4% of NSAID users have GI tract complications each year. **About 10% of patients who take NSAIDs daily develop an acute ulcer.** NSAID-induced ulcers have an increased incidence of bleeding, with gastric ulcers and duodenal ulcers having 10- to 20-fold and 5- to 15-fold risk ratios, respectively.

**Upper GI tract endoscopy establishes a diagnosis in more than 90% of cases and assesses the current activity of bleeding.** It aids in directing therapy and predicts the risk of rebleeding. Furthermore, it allows for endoscopic therapy. Endoscopic hemostasis can be achieved through a variety of ways, including thermotherapy with a heater probe, multipolar or bipolar electrocoagulation, and ethanol or epinephrine injections. As shown in Figure 9–1, endoscopy can demonstrate bleeding, esophageal varices, gastroduodenal bleeding, or no bleeding. For nonvariceal bleeding, endoscopic hemostasis is usually achieved with the use of epinephrine injections followed by thermal therapy. Permanent hemostasis occurs in roughly 80% to 90% of patients. Once bleeding is controlled, long-term medical therapy with antisecretory agents such as histamine-2 blockers or proton pump inhibitors is utilized to treat the underlying disease. Testing for *Helicobacter pylori* should be performed, and if this organism is present, treatment should be initiated. Any NSAID use should be discontinued. If this is not possible, a prostaglandin analog (such as misoprostol) should be used or, alternatively, one of the selective COX-2 inhibitors should be used to replace nonselective COX inhibitors.

If bleeding continues or recurs, surgery may be necessary. **Surgery is indicated for complicated peptic ulcer disease with massive, persistent, or recurrent upper GI tract hemorrhage or in association with nonhealing or giant ulcers (larger than 3 cm).** For a bleeding gastric ulcer where there is a concern for possible malignancy, either gastrectomy or excision of the ulcer is indicated. For other types of ulcers, the vessel may require ligation followed by a vagotomy procedure and pyloroplasty. If the bleeding source cannot be identified but active bleeding is clearly occurring, patients may undergo selective angiography. This treatment strategy can diagnose and treat bleeding in roughly 70% of patients, as arterial embolization with gel foam, metal
History and physical examination
Peptic ulcer disease
ASA, NSAID use
Alcohol intake
Cirrhosis

Laboratory studies
CBC
Liver function tests
PT, PTT
Platelets
Type and cross-match

NGT
Gastric irrigation

Upper GI (tract) hemorrhage
ABC’s
Resuscitation
Preparation for endoscopy
Endoscopy

Gastroduodenal source of bleeding
Endoscopic hemostasis
Bleeding not controlled
Surgery

Bleeding controlled
Medical therapy

Bleeding esophageal varices
See portal HTN

No bleeding source identified
Selective angiography
Arterial embolization
Bleeding controlled

Figure 9–1. An algorithm for the treatment of patients with hematochezia or melena without hematemesis. ASA, aminosalicylate; NSAID, nonsteroidal anti-inflammatory drug; NGT, nasogastric tube; HTN, hypertension; GI, gastrointestinal; ABC’s, airway, breathing, circulation; CBC, complete blood count; PT, prothrombin time; PTT, partial thromboplastin time.
coil springs, or a clot can be utilized to control bleeding. In addition, arterial vasopressin can cause bleeding to stop in some patients with peptic ulcer disease.

Comprehension Questions

[9.1] A 55-year-old male has undergone upper endoscopy. He is told by his gastroenterologist that although this disorder may cause anemia, it is unlikely to cause acute gastrointestinal hemorrhage. Which of the following is the most likely diagnosis?

A. Gastric ulcer  
B. Duodenal ulcer  
C. Gastric erosions  
D. Esophageal varices  
E. Gastric cancer

[9.2] A 32-year-old man comes to the emergency department with a history of vomiting “large amounts of bright red blood.” The first step in the treatment of this patient is:

A. Obtaining a history and performing a physical examination  
B. Determining hemoglobin and hematocrit levels  
C. Fluid resuscitation  
D. Inserting a nasogastric tube  
E. Performing urgent endoscopy

[9.3] A 65-year-old male is brought into the emergency room with acute upper gastrointestinal hemorrhage. A nasogastric tube is placed with bright red fluid aspirated. After 30 minutes of saline flushes, the aspirate is clear. Which of the following is the most accurate statement regarding this patient’s condition?

A. He has about a 20% chance of rebleed  
B. The mortality for his condition is much lower today than 20 years ago  
C. His age is a poor prognostic factor for rebleeding  
D. Mesenteric ischemia is a likely cause of his condition
[9.4] A 52-year-old alcoholic male with known cirrhosis comes into the ER with acute hematemesis. Bleeding esophageal varices are found during upper GI endoscopy. Which of the following is most likely to be effective treatment for this patient?

A. Balloon tamponade of the esophagus  
B. Proton pump inhibitor  
C. Triple antibiotic therapy  
D. Misoprostol oral therapy  
E. Endoscopic sclerotherapy

**Answers**

[9.1] **E.** Gastric cancer is relatively asymptomatic until late in its course. Weight loss and anorexia are the most common symptoms with this condition. Hematemesis is unusual, but anemia from chronic occult blood loss is common.

[9.2] **C.** Fluid resuscitation is the first priority in order to maintain sufficient intravascular volume to perfuse vital organs.

[9.3] **A.** About 20% of patients with acute upper GI hemorrhage will have continued or rebleeding episodes. The mortality has remained the same (about 8–10%) over the past 20 years.

[9.4] **E.** Endoscopic injection of sclerosing agents directly into the varix is effective in controlling acute hemorrhage due to variceal bleeding in about 90% of cases. Balloon tamponade is a therapy used infrequently for acute esophageal variceal bleeding due to its limited effectiveness in achieving sustained control of bleeding. Other therapies include vasopressin or octreotide to decrease portal pressure.
CLINICAL PEARLS

- Early endoscopy has been shown to be more efficacious in identifying the bleeding sources, and in patients with active ongoing bleeding it may help in achieving early control of bleeding.
- About 10% of patients who take daily NSAIDs develop an acute ulcer.
- Surgery is indicated for complicated peptic ulcer disease with massive, persistent, or recurrent upper GI tract hemorrhage or in association with nonhealing or giant ulcers (larger than 3 cm).
- Acute GI tract hemorrhage should be treated with aggressive fluid resuscitation, close monitoring of patient response, nasogastric tube insertion following resuscitation to determine whether bleeding is active, and gastric irrigation with room-temperature water or saline until gastric aspirates are clear.
- The most common cause of upper GI tract hemorrhage in a patient with cirrhosis and portal hypertension is variceal bleeding, which carries a high rate of mortality and risk of rebleeding.
- The most common cause of pediatric significant upper GI tract hemorrhage is variceal bleeding from extrahepatic portal venous obstruction.

REFERENCES

CASE 10

A 67-year-old man presented to the emergency center with a 6-hour history of bleeding per rectum. The patient’s symptoms began after he developed an urge to defecate that was followed by several voluminous bowel movements containing maroon-colored stool mixed with blood clots. The patient complains of feeling light-headed just prior to arriving at the hospital but denies any abdominal pain. His past medical history is significant for borderline hypertension managed with diet control. His surgical history is significant for a right inguinal hernia repair 2 years ago. His blood pressure is 100/80, pulse rate 110/min, and respiratory rate 20/min. The results of an examination of his abdomen are unremarkable. The rectal examination revealed no masses and a large amount of maroon-colored stool in the rectal vault.

What should be your next step?

What is the most likely diagnosis?

How would you confirm this diagnosis?
ANSWERS TO CASE 10: Lower Gastrointestinal Tract Hemorrhage

Summary: A 67-year-old man presents with acute lower gastrointestinal (GI) tract hemorrhage. The patient’s symptoms and vital signs indicate a significant acute hemorrhage.

◆ Next step: The patient’s presentation is highly suggestive of hypovolemic shock; therefore the initial treatment should consist of volume resuscitation with isotonic crystalloid solution and close monitoring of his response to resuscitation.

◆ Most likely diagnosis: Acute lower GI tract hemorrhage.

◆ How to confirm the diagnosis: Place a nasogastric (NG) tube to sample the upper GI tract contents; the possibility of gastric bleeding can be eliminated if nonbloody, bilious material is recovered. Esophagastroduodenoscopy (EGD) is the definitive method of evaluation to rule out a duodenal source of bleeding.

Analysis

Objectives

1. Be able to differentiate the clinical presentations of occult and acute anorectal, nonanorectal lower GI tract, and upper GI tract bleeding.
2. Learn a diagnostic and therapeutic approach to lower GI tract bleeding.

Considerations

The passage of maroon-colored stool and blood clots generally indicates acute bleeding from a lower GI tract source (distal to the ligament...
of Treitz). Maroon-colored stool represents a mixture of fecal material and blood, indicating that the bleeding source is located proximal to the lower rectal segment and anus. **The passage of blood clots can occur with brisk bleeding from an upper GI tract source.** Placement of an NG tube is useful during the initial evaluation for possible upper GI tract bleeding, although up to 16% of patients may have nonbloody NG aspirate with upper GI tract bleeding originating from the duodenum. In middle-aged and older adult patients, the **most likely causes of acute lower GI tract bleeding are diverticulosis, angiodysplasia, and neoplasm,** and these lesions are generally painless. When lower GI tract bleeding occurs in the presence of abdominal pain, the possibility of an ischemic bowel, inflammatory bowel disease, intussusception, and a ruptured abdominal aneurysm should be entertained. Following resuscitation, the primary goal in the treatment of a patient with acute and continued lower GI tract bleeding is localization of the bleeding site (colonoscopy, mesenteric angiography, and/or an isotope-labeled red blood cell [RBC] scan).

**APPROACH TO LOWER GI TRACT BLEEDING**

**Definitions**

**Occult gastrointestinal tract bleeding:** Slow bleeding originating anywhere along the upper aerodigestive or lower GI tract, most commonly associated with neoplasm, gastritis, and esophagitis. Patients generally do not report bleeding and commonly present with iron-deficiency anemia, fatigue, and hemoccult-positive stool.

**Overt lower gastrointestinal tract bleeding:** Hematochezia or melena. The most common causes in children and adolescents are Meckel diverticulum, inflammatory bowel disease, and polyps. In young and middle-aged adults the most common causes are diverticulosis, neoplasm, and inflammatory bowel disease. In older adults, the most common causes are diverticulosis, angiodysplasia, and neoplasm.

**Tagged red blood cell (RBC) scan:** Nuclear medicine imaging using RBCs labeled with technetium-99m. This technique is
highly sensitive in identifying active bleeding at a rate of 0.1 ml/min or greater; however, the images obtained may not accurately localize the GI tract bleeding site. Some recommend this imaging modality as an initial screening study prior to performing mesenteric angiography.

**Mesenteric angiography:** Selective angiography of the superior and inferior mesentery arteries can help identify bleeding from the midgut and hindgut. This procedure has greater specificity in localizing the bleeding site than a tagged RBC scan. Selective injection of vasopressin or gel foam can be applied to treat active bleeding in patients who are not suitable surgical candidates. The bleeding generally has to be greater than 0.5 to 1.0 mL/min in order to be visualized by angiography.

**Rigid proctosigmoidoscopy:** A simple bedside procedure in which a nonflexible endoscope is used to visualize the most distal 25-cm segment of the lower GI tract.

**Diagnostic colonoscopy:** Flexible fiberoptic endoscopy that evaluates the entire colon and rectum and is reserved for hemodynamically stable patients. The reported success rate in identifying the bleeding source and site is as high as 75%, but this figure is highly variable depending on the operator and the timing. The advantages of this procedure are that it can rule out the possibility of a colorectal bleeding source and that identified bleeding angiodysplasia can be treated with epinephrine injection or coagulation.

**Angiodysplasia:** Also known as vascular ectasia, a common degenerative vascular lesion characterized by small, dilated, thin-walled veins in the mucosa of the GI tract. It occurs most commonly in the cecum and ascending colon of individuals more than 50 years of age. Approximately 50% of patients have associated cardiac disease. Up to 25% of patients with angiodysplasia have aortic stenosis. Most patients with angiodysplasia present with low-grade, self-limiting bleeding, although approximately 15% present with massive bleeding.

**Clinical Approach**

A patient presenting with overt lower GI tract bleeding should be quickly assessed for intravascular volume status and hemodynamic sta-
Overt Lower GI Bleeding

Resuscitation

History

(anorectal source suspected)

(anorectal source not suspected)

Hemodynamic instability
or signs of active bleeding

Stable vital signs

Rigid proctosigmoidoscopy

(negative and continued bleeding)

NG tube or EGD

(-)

Tagged RBC Scan

(-)/continued bleeding

(+)

Observe

(-)

Local treatment
or resection

(+)

Mesenteric angiography

(+)

(source found)

Colonoscopy

Continued bleeding/no source identified

Tagged RBC scan or angiography

Figure 10–1. An algorithm for the management of hematochezia. Esophagogastroduodenoscopy (EGD), colonoscopy, and small bowel contrast radiography should be performed in patients whose gastrointestinal (GI) tract bleeding has resolved to eliminate the possibility of GI tract bleeding as the cause of bleeding. NG, nasogastric; RBC, red blood cell.
bility. A detailed history is important. The identification of coexisting medical problems may help identify patients whose bleeding is the result of coagulopathy or thrombocytopenia (medical causes of bleeding). If the patient has had a previous abdominal vascular reconstruction, the possibility of an aortoenteric fistula must be strongly considered and ruled out. The history elicited should include details regarding the quality and appearance of the bleeding. Melena (tarry stool) indicates the degradation of hemoglobin by bacteria and forms after blood has remained in the GI tract for more than 14 hours. Melena is usually associated with upper GI tract or small bowel bleeding but can occur with bleeding from the ascending colon. The passage of maroon-colored stools generally excludes a possible bleeding source in the rectum and anus. Bleeding from the rectum is usually characterized by the passage of formed stools streaked with blood or the passage of fresh blood at the end of a normal bowel movement. Most episodes of overt lower GI tract bleeding resolve spontaneously without specific therapy. It is important to rule out GI tract neoplasm as the source of bleeding in patients whose bleeding resolves. Patients whose bleeding creates adverse hemodynamic consequences or necessitates blood transfusion should undergo prompt evaluation to localize the source of bleeding so that operative excision can be accomplished. (See Figure 10–1 for management strategy.)

Comprehension Questions

[10.1] A 75-year-old man develops hematochezia and presents with hemodynamic instability. The patient’s vital signs improve slightly with crystalloid and packed red cells infusion. Which of the following is considered the most appropriate next step(s) in management?

A. EGD, proctosigmoidoscopy, and a barium enema
B. NG tube, proctosigmoidoscopy, and a tagged RBC scan with or without mesentery angiography.
C. NG tube, mesentery angiography, and colonoscopy
D. EGD and colonoscopy
Which of the following conditions is almost always associated with painless hematochezia?

A. Aortoenteric fistula developing 1 year after an abdominal aortic aneurysm repair  
B. Ischemic colitis involving the descending colon  
C. Bleeding duodenal ulcer  
D. Superior mesentery artery embolus

Which of the following diagnostic modalities has the greatest specificity in identifying the source of lower GI tract bleeding?

A. Tagged RBC scan  
B. Barium enema  
C. Colonoscopy  
D. Surgical exploration

**Answers**

[10.1] **B.** NG tube, proctosigmoidoscopy, and a tagged RBC scan are most appropriate for a patient who is unstable.

[10.2] **A.** Aortoenteric fistula following aortic reconstruction is nearly always associated with painless hematochezia.

[10.3] **C.** Colonoscopy has the highest specificity in identifying the source of lower GI tract bleeding (ie, the lowest false-positive rate for bleeding source identification).
CLINICAL PEARLS

- The primary goal in the treatment of a patient with acute and continued lower GI tract bleeding is localization of the bleeding site.
- The ability to localize the bleeding during an abdominal exploration is greatly compromised. Thus, exploratory laparotomy should be avoided prior to precise localization of the bleeding site.
- Tagged RBC scan results should be interpreted with great caution because localization of bleeding to a region of the abdomen does not necessarily localize bleeding from a specific segment of the GI tract.
- Colonoscopy should be reserved for stable patients with lower GI tract bleeding.

REFERENCES