Postoperative Complications

5

Ronald V. Maier and Abe Fingerhut

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Objectives

- Discuss common serious complications of operations for complex disease.
- Understand underlying pathophysiology.
- Explore decision-making process in approach to care.
- Elucidate prevention and treatment options.

Note: see individual chapters for specific complications.

5.1 Peritonitis/Abscess

- Both are manifestations of intra-abdominal infections:
  - Peritonitis: diffuse infection of the peritoneal space
    Site
    - Somewhat localized to one quadrant
    - Or generalized to two or more quadrants with a significantly increased risk of mortality
  - Abscess: localized infection in the abdomen
    Forms anywhere
    - Within the peritoneal space
    - In the extraperitoneal space, primarily the retroperitoneum
    - Or within the organs themselves, primarily the liver and spleen

R.V. Maier, MD, FACS (✉)
Jane and Donald D. Trunkey Professor and Vice Chair, Department of Surgery, University of Washington Surgeon-in-Chief, Harborview Medical Center, Seattle, WA 98104, USA
e-mail: ronmaier@u.washington.edu

A. Fingerhut, Doc hon c, FACS, FRCS(g), FRCS(Ed)
Department of Surgical Research, Clinical Division for General Surgery, Medical University of Graz, Graz, Austria
e-mail: abefingerhut@aol.com

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- Both occur more often today, postoperatively, due to the increasing severity of disease and complexity of procedures (including damage control) performed currently and the associated increased survival of the patient.
- Causes:
  - By far the most common cause is anastomotic leakage.
    Management depends on patient status.
    - Stable: nonoperative management is possible.
    - Unstable: surgery is indicated.
      - Laparotomy or for some laparoscopy

    If early intervention, the anastomosis can be redone, with or without protective stoma, if not, and most often.
    The two extremities should be brought out (double-barrel ileostomy or colostomy).
    Hartmann's procedure.
    Complete peritoneal toilet.
    Drainage.
  - Other causes are rare.
    Collections (abscess) in a stable patient can be drained percutaneously.
- There are no good guidelines on prevention of postoperative infections.
  - The current assumption is that factors that decrease SSI will also have a beneficial effect on the incidence of deep organ space infections, both peritonitis and abscesses.
  - These factors include:
    Avoidance of unintended injury to the bowel or other organs during any operative procedure (critical)
    Avoidance of hypoxia, hypothermia, and hyperglycemia
    Appropriate antibiotic prophylaxis and treatment
    Adequate delay in definitive completion of the surgery or closure of the wounds
- Diagnosis:
  - Primarily: pain and abdominal tenderness.
  - Fever and elevated WBC are frequent but may be absent early in the disease process.
  - Specific to diffuse peritonitis:
    Diffuse physical findings of tenderness, rebound, and guarding, such as following intestinal leak.
    Diagnosis can be made on physical examination leading to prompt surgical intervention.
  - Conversely, postoperative abscess or tertiary peritonitis can be significantly more difficult to diagnose.
    The clinical picture is less straightforward, and additional studies are frequently necessary to make the diagnosis.
    - Current multi-slice abdominal CT scans are the most useful.
- Treatment requires both source control and appropriate antibiotics.
  - Diffuse peritonitis (almost always indicating an uncontrolled GI source of contamination) mandates operative exploration for source control.
  - In contrast, intra-abdominal abscess may be sufficiently treated by drainage alone.
    Drainage is the appropriate initial step in the stable patient or patient responsive to initial therapy.
    Frequently can be placed percutaneously using radiologic guidance including fluoroscopy, CT, ultrasound, or laparoscopy.
    - There are no randomized prospective trials comparing open drainage to percutaneous drainage, but solid cohort studies suggest that the net success and mortality appear to be equal between the approaches, but percutaneous or laparoscopy avoid the potential iatrogenic morbidity of open drainage.
    - Open drainage is usually reserved for the patient in whom percutaneous drainage has failed or is not technically feasible.
  - Importantly, approximately one fourth of cases will require an additional intervention to resolve the infection.
    Need for reintervention is indicated when the patient fails to improve or worsens following intervention or when infection recurs.
    Mandatory or scheduled relaparotomies have not been shown to reduce the morbidity or mortality in these complex cases.

5.2 Paralytic Ileus

- Common postoperative disorder:
  - Occurring to some extent in most patients undergoing abdominal surgery
  - Most often transient, usually lasting 2–3 days, but may last for more than 7–10 days
- Caused by neural, humoral, and metabolic factors:
  - Direct intestinal exposure, manipulation, and desiccation
  - Retroperitoneal bleeding
  - Severe infection, both intraperitoneal and extraperitoneal, such as pneumonia
  - Electrolyte imbalances, particularly hypokalemia
  - Drugs, primarily narcotics
    Morphine binds to μ-opioid receptors in the CNS and colon causing nonpropulsive electrical activity.
- Of clinical importance, should increase suspicion and help identify preemptively the onset of intestinal ischemia or an intra-abdominal infectious process, such as a localized abscess or diffuse peritonitis, while still reversible
- Treatment:
  - Watchful support is in most cases appropriate and safe:
    NG suction and fluid resuscitation.
    Rapid correction of electrolyte imbalances, especially hypokalemia.
    The use of thoracic epidurals enhances return of bowel function.
  - In contrast, the development of secondary ileus after initial return of bowel function mandates evaluation for mechanical obstruction or intra-abdominal sepsis from abscess or peritonitis:
    Modern multi-slice CT scanners is exceptionally effective.
    Laparotomy may be necessary to definitively exclude these factors and to rule out intestinal ischemia or threatened viability of the intestinal wall due to intense and/or prolonged distension.

5.3 Bleeding/Coagulopathy

- Can occur after any invasive procedure with increasing risk paralleling the increase in complexity of the procedure.
- Diagnosis:
  - Should be suspected in any postoperative patient whom develops tachycardia, pallor, volume-dependent hypotension, oliguria, restlessness/anxiety, and/or abdominal distention.
    An anxious, agitated postoperative patient should never be sedated without evaluation for ongoing bleeding.
  - Note that the hematocrit fall may be delayed in the acute setting until intravascular volume is restored.
  - Evidence of bleeding site should be sought with physical exam and evaluation of all tubes and wounds/dressings, along with any evidence of diffuse bleeding from puncture sites indicative of a coagulopathy.
  - Coagulation tests, including platelet count, bleeding time, and PT and PTT along with fibrinogen levels and thromboelastograph (TEG) or rotational thromboelastometry (ROTEM) may differentiate primary versus secondary hemostasis failure.
- Causes:
  - Absence or loss of surgical hemostasis
  - Technical error
  - Resolution of vasoconstriction
  - Coagulopathy
- Management:
  - Absence or loss of surgical hemostasis and/or refractory hypotension, ACS, or ongoing need for blood transfusion usually requires returning to the OR and reoperation.
    A discreet bleeding point is frequently not found.
    However, evacuation of the dead space and blood, breaking the endogenous thrombolytic cycle, is frequently successful.
  - Hemostatic failure due to platelet or coagulation cascade failure.
  - Correction of hypothermia, suppression of drug-inducing agents.

  - Search for acquired secondary coagulopathy (consumption and/or dilution from tissue injury, volume resuscitation, sepsis, or transfusion with product poor blood component therapy).
  - Low fibrinogen level should be treated with FFP.
  - Early aggressive transfusion plus FFP and possibly platelets to achieve a near 1:1 ratio of packed RBC to FFP is associated with an improvement in overall survival following massive blood loss and transfusion and reduction in overall volume of blood products required (based on recent military observations after severe trauma).
- The variable impact on perfusion can further damage and cause progression in injured or diseased tissue or compromise the already completed repairs, leading to anastomotic break down, wound dehiscence, or intra-abdominal hypertension (IAH) and progress to abdominal compartment syndrome (ACS).

5.4 Abdominal Compartment Syndrome (ACS)

- Definition: end-organ dysfunction (new or ongoing) related to intra-abdominal hypertension (IAH)
  - Physiopathology:
    The abdominal compartment is contained with layers of initially elastic but ultimately poorly compliant tissue layers.
    Similar to cardiac tamponade, pressures may increase slowly until compliance of tissues is exceeded, with rapid increases occurring to small volume changes.
    When the intra-abdominal volume/pressure exceeds these limits, there is a direct effect on numerous organ functions, including cardiac, respiratory, renal, neurologic, and muscular systems.
    If not recognized and treated, the end result is worsening organ failure and potential death.
    In critically ill patients, ACS can be either primary from a direct increase in the intra-abdominal volumes or secondary due to illness outside the abdominal cavity:
    - Primary ACS is seen following events such as rupture of an AAA, spontaneous retroperitoneal bleed, pelvic bleeding, or direct injury to intra-abdominal organs.
    - Secondary ACS occurs following ischemia/reperfusion, burns, or infection, where total body, including intra-abdominal, edema occurs due to the host inflammatory response or systemic inflammatory response syndrome (SIRS).
      - In addition, the recent past trend of vigorously (and overly) resuscitating the patient with large volumes of crystalloid to reach an arbitrary goal, such as supranormal oxygen delivery, added an iatrogenic component to the edema, increased volume of tissues, and IAH.
    - Recurrent ACS is the redevelopment of ACS after treatment for primary or secondary ACS.
  - IAH can be easily measured using the fluid column height above the pubis in a Foley catheter, after instilling 50 cc of sterile saline inserted into the bladder.
    During ACS, IAH is defined as a pressure greater than 20 mmHg, but pressures can vary greatly between patients without signs of ACS.
    The primary effects of ACS are through impairment of perfusion and oxygenation:
    - Increased IAH
      - Decreases perfusion of all intra-abdominal organs and the abdominal wall compromising wound healing
      - Increases venous collapse and resistance with impaired renal, hepatic, and bowel function
      - Leads to IVC collapse responsible for decreased cardiac preload
      - Through elevation of the diaphragm compresses the heart similar to tam-

ponade, with decreased cardiac output and further decreases in organ perfusion
The restriction of the thoracic cavity compresses the lungs, elevates ventilatory pressures and causes loss of FRC, and decreases oxygenation with additional organ insult from worsening hypoxemia.
– Rapid decompression through opening of the abdomen creating an "open abdomen" is critical.

5.5 Damage Control (Open and Laparoscopic)

See also Chap. 1 (schwab, Leppaniemi)

- Definition: operations (whether via laparotomy or laparoscopy) that are limited, "incomplete" procedures performed in patients where persisting to complete the procedure would significantly increase the morbidity and mortality of the patient.
- Principal indications:
  – Operations performed for control of hemorrhage, contamination, or potential ischemia.
  – Injury to major vascular structures or highly vascular solid organs from extensive resections for malignancy, infection, or other diseases.
  Particularly true when significant blood loss and massive transfusion leads to the "Bloody Triad" of hypothermia, acidosis, and coagulopathy, associated with an unacceptably high mortality.
  – Laparoscopic procedures can produce or identify potentially morbid or lethal events that are unsafe to definitely pursue due to patient disease or comorbidity.
  Examples (can be best treated with placement of drainage to control the source, while life-threatening comorbidites are corrected):
    - Laparoscopic identification of a poorly identified bile leak after an ERCP
    - Abscess from unidentified perforated colonic processes
- Almost always require a less than optimal closure of the incisions and need for further operative intervention.

5.6 Reoperation: Timing

- Damage control reoperations
  – Necessary to:
  Complete repair or resection
  Perform anastomoses to restore intestinal continuity
  Evaluate for occult or missed injuries
  Rule out progression of ischemia
  Remove temporary packing used to control bleeding
  Remove temporary vascular shunts followed by vascular repair
  Manipulate or replace drains or drainage tubes
  Attempt delayed primary closure of the abdominal cavity
- Timing of reoperation
  – Dictated by:
  Disease and injuries present.
  Physiologic response of the patient to the initial or previous procedure.
  Somewhat variable (based on the above considerations): most reoperations occur between 12 and 72 h, preferring the soonest possible.
- Specific considerations for potential ongoing or progressive ischemia:
  – Whether from chronic or acute mesenteric ischemia or subsequent to repair of the mesenteric artery or ligation of the proximal mesenteric or portal vein.
  – Planned reoperation to rule out ischemia is indicated.
  Lack of improvement or progression of base deficit, lactate levels, or ongoing requirements for fluid resuscitation all indicate the likelihood of ongoing ischemia.

5.7 Wound Dehiscence/ Management

- Causes:
  - Inadequate perfusion due to the increased tension required for closure of swollen and noncompliant tissues or hypovolemia and hypoxia from any other cause (e.g., effect of smoking)
  - Infection causing direct breakdown of tissues and impairment of healing
  - Increased intra-abdominal pressure
  - Systemic effects of:
    Diabetes, malignancy, steroid or other immunosuppressive therapy, and chronic lung disease
- Prevention:
  - Consideration of time since insult helps determine whether a wound should be closed or reopened
    6–8 h is quoted for trauma, but no rules have been established in nontrauma surgical emergencies.
    - e.g., *a* contaminated ischemic lower limb may never be safe to close.

    Opening a wound and delayed primary closure is a viable option when in doubt.
  - Currently, there is no evidence that running versus interrupted initial fascia closure has an effect on the risk of dehiscence.
  - When in doubt – delay closure or reopen.
- In the critically ill patient, do not neglect both underlying malnutrition and inadequate levels of structural protein and cofactors for healing, as well as the additional stresses of the diseases involved.
- Diagnosis:
  - All wounds should be inspected if the patient displays any evidence of infection or if skin changes or significant drainage occurs at the wound site.
  - The classic salmon pink fluid drainage of peritoneal fluid from disrupted fascia mandates removal of any dermal closure and both visual and manual inspection of the wound fascia.
  - Similarly, any systemic sign of infection, or any local changes involving erythema, purulence, skin blistering, or darkening at the wound site, mandates close evaluation, and opening of the superficial wound if concern exists.
- Management:
  - In virtually all cases of wound dehiscence, unless physiologically prohibitive, the patient should be explored in the operating room.
  - The fascia should be taken down and carefully inspected for ischemia or infection.
    All diseased fascia should be resected back to healthy tissue.
  - Careful inspection of the abdomen is necessary to rule out anastomotic leaks, intra-abdominal abscess, or peritonitis that requires additional intervention. In cases where the fascia requires little or no debridement and tension on closure is acceptable, repeat fascial closure may be possible. To not repeat what has failed, additional techniques are required, most commonly the wide-based, interrupted "mass closure" encompassing both layers of fascia and rectus muscle with or without including the dermis and subcutaneous tissue in each bite. In cases where closure leads to unacceptable tension, the abdomen should be left open.

Pitfalls: Lack of Recognition

- Lethal pathophysiology – "Bloody Triad"
- Ongoing progressive disease: bleeding, ischemia, no source control
- Presence of IAH/ACS: possible recurrence
- Need for reoperation
- Wound compromise

5.8 Summary

Procedures should be limited to prevent a potential lethal outcome, and use a staged response to optimize survival. Aggressive restoration of physiology and minimization of comorbidity during constant monitoring is crucial. Make the commitment to a serial/ongoing process of care and plan on returning to "fight another day." In effectively dealing with complications, the surgeon must know and recognize the risks of complication, using a "worst case scenario" mentality.

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