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Stercoral Colitis: Advanced Nursing and Technical Competencies in Acute Injury Management

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Abstract

Background: Stercoral colitis is a serious inflammatory and ischemic condition of the colon caused by severe, chronic fecal impaction. Hardened fecal masses (fecalomas) exert persistent pressure on the colonic wall, leading to mucosal ulceration, ischemia, and risk of catastrophic perforation. It predominantly affects high-risk populations, including the elderly, immobile, and those with neurologic or psychiatric conditions.

Aim: This review aims to outline the pathophysiology, clinical presentation, diagnostic approach, and evidence-based management of stercoral colitis, with a focus on the acute nursing and technical competencies required for effective intervention.

Methods: A comprehensive synthesis of current literature is presented, detailing the etiology, epidemiology, and diagnostic pathway. Emphasis is placed on the critical role of contrast-enhanced computed tomography (CT) for diagnosis and risk stratification. Management strategies, from conservative disimpaction to emergent surgical resection, are discussed.

Results: Diagnosis hinges on clinical suspicion in at-risk patients and is confirmed by CT findings of fecal impaction, colonic dilation (>6 cm), wall thickening (>3 mm), pericolonic fat stranding, and mucosal discontinuity indicating ischemia. Initial management for stable patients involves bowel decompression via manual or endoscopic disimpaction, bowel rest, and antibiotics. Signs of peritonitis, perforation, or failed conservative therapy mandate emergent surgical resection, typically with colostomy formation. Mortality is high (32-60%), especially with perforation or extensive (>40 cm) colonic involvement.

Conclusion: Stercoral colitis is a preventable, life-threatening complication of chronic constipation. Outcomes depend on early recognition, prompt CT imaging, and timely intervention. Successful management requires an interprofessional team approach, integrating acute surgical care with long-term strategies to prevent recurrence.

Keywords: Stercoral colitis, Fecal impaction, Colonic ischemia, Computed tomography, Bowel perforation, Chronic constipation.

Introduction

Stercoral colitis is an uncommon but clinically significant inflammatory condition of the colon that develops when severe fecal impaction causes progressive colonic dilation and the formation of hardened fecal masses known as fecalomas. These fecalomas exert persistent pressure on the colonic wall, which can impair blood flow, produce localized pressure necrosis, and ultimately result in ulceration or full-thickness perforation. In addition, marked colonic distention increases intraluminal pressure and may further compromise vascular perfusion, predisposing the affected bowel segment to ischemic injury.[1] This dual mechanism—mechanical compression and vascular compromise—forms the pathological basis of stercoral colitis. Ulcerations

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associated with stercoral colitis tend to occur in regions of the colon that naturally have narrower luminal diameters and reduced vascular redundancy. The sigmoid colon and rectum are the most frequently involved sites, reflecting both their anatomic susceptibility and their role in stool storage and transit.[2] When disease progression leads to perforation, the outcomes are often catastrophic. Reported mortality rates range from 32% to 60%, underscoring the condition's potential severity and the need for prompt recognition and intervention.[3][1] Once perforation occurs, fecal contamination of the peritoneal cavity can lead to fulminant peritonitis, overwhelming systemic infection, septic shock, and multi-organ failure, particularly in vulnerable patients. Stercoral colitis is most commonly encountered in individuals with longstanding constipation, as chronic retention of stool leads to progressive desiccation and hardening of fecal material. Elderly patients are disproportionately affected, especially those with underlying cognitive impairment such as dementia, as they may have reduced awareness of bowel habits, decreased mobility, inadequate hydration, or impaired autonomic regulation of gut motility.[4] Patients residing in long-term care facilities, those who are bedbound, and individuals with severe debilitation similarly face increased risk due to immobility, reduced nutritional intake, and frequent use of constipating medications, including opioids and anticholinergic agents.

Although stercoral colitis is traditionally associated with the geriatric population, it can also occur in younger individuals. Psychiatric patients—particularly those with conditions such as schizophrenia, depression, or eating disorders—may exhibit poor bowel habits, reduced physical activity, or medication-induced gastrointestinal hypomotility. In this population, chronic constipation remains the primary precipitating factor. Indeed, across all demographic groups, chronic constipation stands as the most significant risk factor for the development of stercoral colitis, reflecting a sustained imbalance between stool formation and colonic motility that ultimately allows fecaloma formation.[1] The increasing incidence of chronic constipation, particularly in aging populations, highlights the importance of early detection and management of bowel dysfunction to prevent serious complications such as stercoral colitis. Because the clinical presentation may be subtle or mistaken for unrelated gastrointestinal complaints, clinicians must maintain a high index of suspicion in at-risk patients. Comprehensive assessment, timely imaging, and early therapeutic strategies aimed at relieving impaction and reducing colonic pressure can significantly influence outcomes. Ultimately, stercoral colitis serves as a reminder of the potentially life-threatening consequences of unmanaged constipation—an otherwise common and often underappreciated clinical concern.

Fig. 1: Stercoral colitis.

Etiology

The development of stercoral colitis is fundamentally rooted in chronic constipation, a condition that creates the physiological environment necessary for fecal impaction, progressive colonic distention, and the formation of densely compacted masses of desiccated stool known as fecalomas. When stool remains within the colon for prolonged periods, excessive water reabsorption by the colonic mucosa transforms the fecal material into hardened aggregates. These fecalomas lodge most frequently in the rectosigmoid region, an anatomical segment characterized by narrower luminal diameter, reduced compliance, and relatively diminished blood supply. Their presence generates persistent localized pressure on the colonic wall, ultimately initiating a cascade of ischemic injury.[3] Once a fecaloma becomes impacted, the mechanical pressure it exerts disrupts mucosal integrity and compromises microvascular perfusion. Initially, this manifests as ischemic pressure ulceration. As the pressure persists, ulcerations deepen, and focal areas of mucosal and submucosal necrosis develop. With continued ischemia, the necrotic process may extend through the muscularis propria, resulting in full-thickness perforation of the colon. Approximately 27% of individuals with stercoral colitis develop multiple discrete ulceration sites, reflecting the diffuse and severe physiologic stress placed upon the bowel wall in the setting of prolonged distention.[1]

Colonic distention itself plays a central role in the pathogenesis of stercoral injury. As intraluminal pressure rises due to progressive accumulation of fecal material, venous congestion develops within the bowel wall. This congestion exacerbates mucosal edema and further restricts arterial inflow, creating a vicious cycle of hypoperfusion and ischemic damage. The colonic wall becomes increasingly inflamed, friable, and susceptible to perforation. The feared complications of stercoral colitis—ischemic colitis and perforation—are direct consequences of this escalating vascular compromise. When perforation occurs, fecal contamination of the peritoneal cavity can lead to fulminant peritonitis and sepsis, conditions associated with extremely high mortality. The etiologic factors underlying chronic constipation, the principal precursor to stercoral colitis, are diverse and multifactorial. Dietary habits play a significant role; individuals consuming a low-fiber diet experience decreased stool bulk and delayed colonic transit, promoting fecal stasis. Genetic predispositions may influence gastrointestinal motility and fluid absorption patterns. Behavioral factors—such as inadequate hydration, sedentary lifestyle, or habitual suppression of the urge to defecate—further exacerbate constipation risk. Pharmacological contributors are highly significant: opioids, anticholinergics, antidepressants, iron supplements, and calcium channel blockers all reduce gut motility or alter stool consistency, placing patients at elevated risk for fecal impaction. Anatomic and physiologic abnormalities also contribute to stercoral pathology. Conditions that impair pelvic floor function, such as rectoceles, sigmoid redundancy, or obstructive defecation syndromes, predispose individuals to incomplete evacuation. Neurological disorders—including Parkinson disease, spinal cord injuries, multiple sclerosis, and diabetic autonomic neuropathy—can disrupt colonic innervation and weaken effective peristalsis. Similarly, endocrine and metabolic disturbances, including hypothyroidism and hypercalcemia, are known contributors to decreased bowel motility [5].

In elderly populations, the cumulative effect of impaired mobility, polypharmacy, cognitive dysfunction, and dehydration significantly increases constipation prevalence and thereby predisposes individuals to stercoral colitis. The condition is also seen in younger individuals with psychiatric illness, including depression or schizophrenia, where poor bowel habits, limited physical activity, and medication side effects converge. Ultimately, the etiology of stercoral colitis is best understood as the end stage of unrelieved constipation, in which mechanical obstruction, vascular compromise, and progressive colonic injury intersect to produce a potentially life-threatening inflammatory condition. Early identification and management of constipation are therefore critical in preventing the evolution toward stercoral pathology.[5]

Epidemiology

Stercoral colitis remains an uncommon and underrecognized clinical condition, with limited epidemiologic data available due to its rarity and the frequent difficulty in diagnosis. Although reported across diverse age groups, the condition most commonly affects older adults, particularly those who are frail, immobile, or residing in long-term care facilities. Elderly individuals with dementia or prior cerebrovascular accidents are especially vulnerable because impaired cognition, reduced mobility, and dependence on caregivers can obscure the early symptoms of constipation and delay timely intervention.[1] In addition, chronic opioid users—who frequently develop opioid-induced gastrointestinal hypomotility—comprise a significant proportion of affected patients. Younger individuals may also develop stercoral colitis, often in the context of psychiatric disorders where medications, behavioral patterns, or reduced self-care contribute to severe constipation. Neurologic and psychiatric comorbidities further complicate diagnosis by limiting the accuracy of patient-reported symptoms and diminishing the reliability of physical examinations. As a result, clinicians caring for these populations must maintain a heightened index of suspicion to avoid missing this potentially life-threatening condition [1][5].

Chronic constipation, the core risk factor for stercoral colitis, exhibits considerable variability in prevalence across populations. In the geriatric community, constipation is especially common; studies report that up to one-third of adults aged 60 to 110 years experience chronic or recurrent constipation.[5] Several age-related factors contribute to this increased risk. Tooth loss, poorly fitting dentures, and swallowing difficulties may limit the intake of dietary fiber. Older adults often consume inadequate fluids due to reduced thirst sensation, fear of incontinence, or mobility limitations. Physical

inactivity is another important contributor, as decreased ambulation leads to slower colonic transit times. Polypharmacy, particularly the use of medications such as opioids, calcium channel blockers, anticholinergics, iron supplements, and antidepressants, also plays a significant role in reducing gut motility. Additionally, hormonal and autonomic changes associated with aging further diminish bowel function. Epidemiologic studies consistently show that women experience constipation more frequently than men, attributed in part to hormonal differences, higher rates of pelvic floor dysfunction, and increased likelihood of using constipating medications.[5] Although stercoral colitis is most often associated with the elderly, it is not exclusively a disease of older adults. Children, particularly during key developmental milestones, experience high rates of constipation and therefore represent a vulnerable but often overlooked group. Constipation in pediatric populations is especially common during periods such as weaning, toilet training, and the transition to structured school environments, where changes in routine, diet, and toileting behaviors frequently contribute to stool withholding and hardening.[6][7] Pediatric patients with chronic constipation, particularly those with neurodevelopmental disorders or those taking medications that impair bowel motility, may progress to severe fecal impaction and are at risk for stercoral complications if the condition remains unaddressed. Despite its rarity, stercoral colitis carries significant morbidity and mortality, making epidemiologic awareness essential for early recognition and prevention. The condition's prevalence appears to be rising in tandem with global increases in the elderly population, widespread use of opioids, and higher survival rates following neurologic injuries. Yet due to underreporting, misdiagnosis, and the absence of large-scale epidemiologic studies, the true incidence of stercoral colitis is likely higher than current estimates suggest. Improved documentation, increased awareness among clinicians, and more robust research efforts are needed to better characterize the epidemiologic burden of this condition and enhance preventive strategies for at-risk populations [5][6][7].

Pathophysiology

The pathophysiology of stercoral colitis is rooted in the progressive consequences of chronic constipation, a condition that leads to prolonged stasis of fecal material within the colon. When stool remains in the colon for an extended period, the bowel continues to absorb water, resulting in increasingly desiccated and hardened fecal material. Over time, this material consolidates into dense masses known as fecalomas. As these fecalomas enlarge, they exert significant mechanical pressure on the surrounding colonic wall, contributing to luminal obstruction, localized ischemia, and eventual inflammatory injury. One of the earliest pathological events is colonic distention. Chronic fecal accumulation increases the intraluminal pressure, and the expanding bolus of stool stretches and deforms the colonic wall. This distention has two major consequences: structural compromise of the bowel and impairment of vascular perfusion. The colon, particularly in segments with limited compliance, cannot withstand prolonged excessive pressure without developing tissue injury. As the fecaloma becomes lodged, it compresses the mucosa and submucosa against the rigid colonic wall. This persistent mechanical pressure reduces capillary perfusion and restricts venous return in the affected area, eventually diminishing arterial inflow. When blood supply fails to meet the metabolic demands of the tissue, localized ischemia develops. The ischemic insult leads to mucosal ulceration, most commonly on the antimesenteric border of the bowel. The antimesenteric side is particularly susceptible because the mesenteric vessels deliver blood from the opposite side, leaving the antimesenteric surface relatively underperfused and more vulnerable when intraluminal pressure rises.[1] As ischemia persists, pressure necrosis progresses through the layers of the bowel wall. Without timely intervention, this necrosis can compromise the full thickness of the colon, resulting in perforation. A stercoral perforation exposes the peritoneal cavity to fecal contamination, precipitating fulminant peritonitis, sepsis, and, frequently, death. This is why stercoral colitis complicated by perforation carries an exceptionally high mortality rate [1].

Anatomical and physiological characteristics of the colon further shape the disease pattern. Approximately 77% of stercoral ulcerations develop in the sigmoid colon or rectum, which represent the narrowest portions of the colon and the segments where stool becomes most desiccated prior to defecation. The reduced luminal diameter and natural angulation of the sigmoid colon make it a frequent site for fecal stasis and fecaloma formation. Additionally, stool in these distal segments contains less water and tends to be firmer, increasing the likelihood of localized impaction and pressure-related

injury. In some cases, the pathology extends beyond localized ulceration adjacent to a fecaloma. Severe colonic distention may compress the vasculature of a long bowel segment, causing diffuse ischemic injury. When the bowel dilates significantly, venous congestion develops, compromising venous outflow and promoting intramural edema. As intramural pressure rises, arterial perfusion decreases further, exacerbating ischemia and paving the way for ischemic colitis. This diffuse vascular compression can impact substantial lengths of the colon, and involvement of more than 40 cm has been identified as one of the strongest predictors of mortality. Intestinal perforation, likewise, is a critical prognostic marker associated with poor outcomes due to the rapid progression to septic shock. Overall, the pathophysiology of stercoral colitis represents a continuum—from chronic constipation and fecal stasis to mechanical obstruction, vascular compromise, tissue ischemia, and potentially fatal perforation. Understanding this progressive cascade emphasizes the importance of early identification and intervention in patients at risk, particularly those with chronic constipation, limited mobility, neurological disorders, or impaired ability to perceive or report symptoms [1][5].

History and Physical

The clinical presentation of stercoral colitis is deeply influenced by its underlying pathogenesis—chronic constipation leading to progressive fecal stasis, luminal obstruction, and colonic ischemia. For most patients, the medical history reveals long-standing constipation, sometimes spanning years. This constipation may arise from a range of etiologies, including neurologic disorders (such as Parkinson disease, spinal cord injuries, or diabetic neuropathy), psychiatric illnesses (including depression, schizophrenia, or severe cognitive impairment), metabolic disturbances, or the chronic use of constipating medications such as opioids or anticholinergics.[3][8] Because stercoral colitis is most commonly seen in patient populations who may be unable to provide a complete, reliable history—such as elderly individuals with dementia, stroke survivors, bedbound nursing home residents, and patients with altered mental status—the clinician must maintain heightened vigilance. These limitations in communication can obscure early symptoms and delay diagnosis, contributing significantly to morbidity and mortality. When symptoms are reported, they are often nonspecific, which further complicates early identification. Patients may describe vague abdominal discomfort, cramping, or diffuse pain that gradually intensifies as intraluminal pressure increases. Fever is a frequent accompanying symptom, often reflecting the inflammatory response associated with ischemic changes or early infection. Some patients may report a recent change in stool caliber or consistency, or the passage of small, pellet-like stools—a phenomenon known as "overflow diarrhea," which represents liquid stool leaking around a fecaloma. Nausea, reduced appetite, or generalized malaise may also be present but are not specific to this condition. On physical examination, the most common findings include abdominal distention and diffuse tenderness to palpation. The abdomen may feel firm or tympanitic due to significant gaseous distention of the colon. Unlike patients with mechanical intestinal obstruction—who often present with an empty rectal vault—those with stercoral colitis frequently have stool detectable during a digital rectal examination. This finding is clinically important, as it helps differentiate stercoral colitis from conditions characterized by complete obstruction. Patients may still pass small volumes of stool or flatus early in the disease process, contributing to diagnostic uncertainty [3][8].

A thorough digital rectal examination (DRE) serves as an essential component of the evaluation. The presence of hard stool masses in the rectal vault supports the diagnosis of fecal impaction and may offer immediate insight into the severity of the underlying colonic burden. However, the absence of hard stool on DRE does not rule out stercoral colitis, as fecalomas may be located more proximally, particularly in the sigmoid colon. In advanced or complicated cases, the physical examination may reveal signs of peritonitis such as rebound tenderness, guarding, or rigidity, indicating possible colonic perforation. Patients with perforation or severe ischemic colitis frequently present with systemic toxicity, including fever, tachycardia, hypotension, and altered mental status. Rapid deterioration into septic shock or multiorgan failure can occur, especially in elderly or frail individuals. These severe presentations require immediate recognition, as delays in treatment significantly worsen outcomes. Given the subtlety of early symptoms and the nonspecific nature of initial clinical findings, clinicians must rely on a combination of historical clues, risk factor assessment, and careful physical

examination. A high index of suspicion is essential, particularly in populations where communication barriers exist or where constipation is chronic and severe. Early identification of stercoral colitis through vigilant clinical evaluation can substantially alter the disease trajectory, reducing the likelihood of ischemic injury, perforation, and life-threatening complications [3][4][5][8].

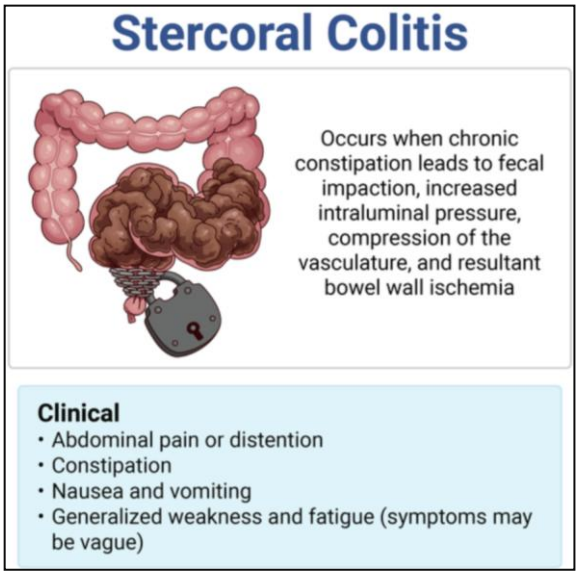

Fig. 2: Stercoral colitis symptoms.

Evaluation

The evaluation of stercoral colitis requires a high index of suspicion and a systematic approach that integrates clinical assessment, laboratory testing, and, most importantly, imaging studies. Because both the presenting symptoms and initial investigations tend to be nonspecific, definitive diagnosis relies heavily on characteristic radiologic findings. Early and accurate identification is crucial, as delays in diagnosis significantly increase the risk of bowel ischemia, perforation, sepsis, and death.[3][4][1] Laboratory abnormalities in stercoral colitis are generally nonspecific and primarily reflect systemic inflammation or evolving sepsis rather than the underlying mechanical process itself. Leukocytosis is a common finding and may be accompanied by elevated acute-phase reactants such as C-reactive protein and erythrocyte sedimentation rate, indicating an ongoing inflammatory response. These changes, while supportive of an inflammatory or infectious condition, cannot distinguish stercoral colitis from other intraabdominal pathologies. More concerning are elevations in serum lactic acid and the development of an anion gap metabolic acidosis. In the context of suspected stercoral colitis, these metabolic derangements should raise an immediate alarm for possible bowel ischemia or impending perforation, as they reflect tissue hypoperfusion and anaerobic metabolism.[3] Because patients with stercoral colitis—especially those with perforation or extensive ischemia—are at high risk for sepsis and septic shock, early sepsis workup is warranted. Blood cultures should be obtained prior to initiating broad-spectrum antibiotics in patients with systemic signs of infection. In addition to a complete blood count and basic metabolic panel, preoperative laboratory evaluation should include coagulation studies and a type and screen in anticipation of potential operative intervention and the need for transfusion. These baseline values help guide resuscitation and perioperative management. However, despite their importance for risk stratification and supportive care, laboratory tests alone do not establish the diagnosis nor reliably distinguish stercoral colitis from other causes of abdominal pain [3][4][1].

Given the limited specificity of history, physical examination, and laboratory testing, imaging plays a central and indispensable role in evaluating suspected stercoral colitis. The selection of imaging modality is guided by the patient's hemodynamic status and the degree of clinical suspicion for perforation. In patients presenting with signs of peritonitis or acute abdomen—such as rigid abdomen, rebound tenderness, or severe guarding—an upright chest radiograph is often obtained initially to assess for free intraperitoneal air under the diaphragm, a classic sign of bowel perforation. While plain films can identify pneumoperitoneum and occasionally gross colonic distension, they are neither sensitive nor specific enough to fully characterize stercoral pathology.[4] The cornerstone of imaging in stercoral colitis is contrast-enhanced computed tomography (CT) of the abdomen and pelvis. CT with intravenous contrast is both the most sensitive and the most specific modality for detecting stercoral colitis, provided that renal function permits the use of contrast agents.[4] It allows simultaneous evaluation of the colonic lumen, wall, and surrounding tissues and helps distinguish uncomplicated fecal impaction from complicated stercoral inflammation and ischemia. Key CT findings that support the diagnosis of stercoral colitis include marked fecal impaction with colonic dilation, typically most pronounced in the rectosigmoid colon. However, in patients presenting later in the disease course, more proximal segments or even the entire colon may be affected. Fecalomas—large, dense, and desiccated fecal masses—may be visible as radiopaque intraluminal concretions. These fecalomas are often surrounded by

dilated colon and can be seen lodged at points of narrowing or angulation. Additional CT features reflect the progression from mechanical compression to ischemic injury. Focal thickening of the colonic wall adjacent to fecalomas is a hallmark of pressure-induced ulceration and early necrosis. Diffuse colonic wall thickening and edema may be present when intraluminal pressure compromises venous and arterial flow over a broader segment. Importantly, CT may demonstrate mucosal discontinuity—areas where the normal enhancement pattern of the colonic mucosa is lost due to impaired perfusion. Mucosal discontinuity represents decreased uptake of intravenous contrast in regions where blood supply is critically reduced or absent, providing radiologic evidence of ischemia and differentiating stercoral colitis from simple fecal impaction, in which the colonic wall remains thin and normally enhancing.[4]

Pericolonic fat stranding is another frequent CT finding, indicating inflammation in the fat surrounding the affected bowel segment. This stranding, often most prominent near impacted stool, supports the diagnosis of an active inflammatory colitis rather than a purely mechanical process. In more advanced or complicated cases, CT may demonstrate extraluminal gas bubbles or fluid collections. The presence of free air adjacent to the colon or within the peritoneal cavity strongly suggests perforation, whereas well-defined fluid collections with gas may indicate pericolonic abscess formation.[1][4] These findings are critical for surgical planning and prognostication. The utility of CT in characterizing stercoral colitis and predicting outcomes has been supported by clinical imaging studies. In one small series evaluating CT features in 41 patients with stercoral colitis, the rectosigmoid colon was involved in all 41 cases, underscoring its predilection as the primary site of pathology.[4] Colonic dilation greater than 6 cm and bowel wall thickening greater than 3 mm in the affected segment were present in all patients, highlighting the importance of these measurements as diagnostic markers. Pericolonic fat stranding was likewise observed in all 41 patients, reinforcing its near-universal association with active inflammation in stercoral disease. Mucosal discontinuity, consistent with focal ischemia, was identified in 6 of 41 cases, while free fluid in the peritoneal cavity was noted in 4 patients and pericolonic abscess in 1 patient. The most powerful radiologic predictors of mortality in this study were the presence of frank perforation and the extent of colonic involvement, particularly when more than 40 cm of bowel was affected.[4] In cases requiring operative intervention, intraoperative findings and histologic examination can confirm the diagnosis. Intraoperative observations may include focal or diffuse ulceration, transmural necrosis, perforation, and the presence of large fecalomas within the lumen. Histopathology typically reveals ulceration, necrosis, and chronic inflammatory changes associated with pressure injury. In summary, the evaluation of stercoral colitis hinges on recognizing nonspecific systemic and abdominal signs in high-risk patients, followed by prompt, contrast-enhanced CT imaging to delineate the extent of disease and identify complications such as ischemia and perforation. The combination of fecal impaction, colonic dilation, wall thickening, pericolonic fat stranding, and, in severe cases, mucosal discontinuity or extraluminal air provides a robust radiologic basis for diagnosis. Early, accurate evaluation is essential for guiding timely medical and surgical management and improving clinical outcomes in this potentially lethal condition.[1][3][4]

Treatment / Management

The management of stercoral colitis relies on early recognition, accurate assessment of disease severity, and timely initiation of therapy aimed at relieving colonic pressure, reversing inflammation, and preventing life-threatening complications such as ischemia and perforation. The therapeutic approach is determined by the patient's clinical stability, the presence or absence of peritonitis, the extent of colonic involvement, and the response to initial conservative interventions. Because stercoral colitis often affects frail, elderly, or neurologically impaired individuals, clinicians must maintain vigilance throughout evaluation and treatment to prevent rapid deterioration. For patients who do not exhibit signs of peritonitis—such as rebound tenderness, guarding, rigidity, hypotension, or signs of septic shock—management can begin conservatively. Non-operative therapy centers on disimpaction, which is essential to relieve colonic distention and halt progressive ischemic injury. Disimpaction may be performed manually through the rectum, a method that can be immediately effective in patients with distal fecalomas. In other cases, endoscopically guided disimpaction is preferred, as it allows direct visualization of the impacted stool and provides a controlled approach to breaking down and extracting fecalomas.[1][3] Endoscopic disimpaction may also

identify mucosal ulcerations, areas of ischemia, or early perforation that are not yet clinically evident.

Patients undergoing conservative treatment are typically admitted to the hospital for close monitoring, including serial abdominal examinations, laboratory studies, and strict attention to fluid and electrolyte balance. During the acute phase, patients should be kept NPO (nothing by mouth) to reduce bowel stress and to ensure they are prepared for surgery should their condition worsen. Supportive management includes avoidance of opioids, as these agents impair colonic motility, worsen constipation, and may aggravate the underlying disorder. Non-opioid analgesics are preferred to minimize gastrointestinal dysmotility. Patients demonstrating clinical signs of sepsis or septic shock require immediate resuscitation with intravenous fluids following standard sepsis protocols. Early administration of broad-spectrum intravenous antibiotics targeting gram-negative organisms and anaerobes is essential, as bacterial translocation, mucosal breakdown, and early ischemia can precipitate bacteremia and septic complications. Resuscitation may also include hemodynamic support with vasopressors if hypotension persists despite adequate fluid therapy. Blood cultures should be obtained before the initiation of antibiotics, and ongoing monitoring is crucial to evaluate for progression to multiorgan dysfunction. Operative intervention becomes necessary in several clinical scenarios: when there is radiologic or clinical evidence of perforation, when patients exhibit peritonitis, when conservative measures fail to relieve colonic obstruction, or when extensive colonic involvement places the patient at high risk of ischemia. Surgery typically involves resecting the affected bowel segment, with formation of a colostomy and creation of a Hartmann pouch. This approach allows removal of necrotic or perforated tissue, diversion of fecal flow, and prevention of further contamination. Limited or segmental resections are generally associated with poorer outcomes because remaining diseased bowel segments retain the potential for recurrent perforation, ischemia, or fecal impact. For this reason, a more extensive resection is often preferred to reduce the risk of future complications and improve long-term prognosis.[1]

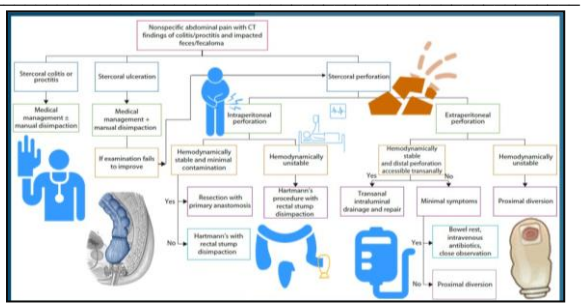

Fig. 3: Management of stercoral colitis.

Following stabilization or recovery from acute illness, attention must turn to the long-term management of constipation, which is the fundamental underlying risk factor for stercoral colitis. Preventive strategies include promoting an appropriate diet rich in fiber, fresh fruits, vegetables, and adequate fluid intake. Education plays a vital role in reducing recurrence: patients should understand bowel-friendly dietary practices, the importance of hydration, and awareness of early signs of constipation that require intervention. Pharmacologic therapy is essential for many patients, particularly those with reduced mobility, neurologic conditions, or chronic medication use that predisposes them to constipation. Osmotic laxatives (such as polyethylene glycol or lactulose) and stimulant laxatives (such as senna or bisacodyl) are considered first-line therapies for maintaining regular bowel movements and preventing fecal impaction.[9] In some cases, stool softeners, enemas, or combination regimens may be beneficial. For patients with refractory constipation, a specialist evaluation may be required to assess colonic transit time, pelvic floor dysfunction, or other contributing physiologic abnormalities. Ultimately, effective treatment of stercoral colitis depends on early recognition, appropriate escalation of care, and long-term strategies to prevent recurrence. Conservative management may be successful when initiated early, but surgical intervention remains crucial in advanced disease. Through a combination of vigilant monitoring, timely decompression, supportive therapy, and comprehensive management of chronic constipation, clinicians can significantly reduce the morbidity and mortality associated with this potentially fatal condition [1][3][4][8].

Differential Diagnosis

The clinical presentation of stercoral colitis is often nonspecific, with symptoms such as abdominal pain, distension, fever, and altered bowel habits, making it indistinguishable at first glance from many other intra-abdominal pathologies. As a result, a careful and structured differential diagnosis

is crucial. Diverticulitis is a common consideration, particularly in older adults presenting with left lower quadrant pain and fever. Both conditions can show bowel wall thickening and pericolonic fat stranding on imaging, but diverticulitis is usually associated with inflamed diverticula rather than fecalomas. Large bowel obstruction must also be considered, especially when colonic dilation and abdominal distension are prominent; however, mechanical obstruction often demonstrates a clear transition point caused by malignancy, volvulus, or strictures, whereas stercoral colitis is driven by fecal impaction and pressure effects. Ulcerative colitis and infectious colitis can present with abdominal pain, diarrhea, fever, and systemic symptoms. Unlike stercoral colitis, these inflammatory conditions typically involve diffuse or continuous mucosal disease, often accompanied by bloody diarrhea and a history of chronic inflammatory bowel disease or recent infection. Similarly, acute mesenteric ischemia may produce severe abdominal pain and metabolic acidosis; however, it usually arises from vascular occlusion rather than luminal fecal compression and is characterized by more diffuse small or large bowel ischemia. Malignancy should always be considered, particularly when imaging reveals mass lesions or unexplained strictures; colorectal cancer can coexist with or predispose to fecal impaction and may be part of the etiologic chain [4][8]. Bowel perforation—whether due to peptic ulcer disease, malignancy, diverticulitis, or ischemia—must be ruled out urgently in patients with signs of peritonitis. Intra-abdominal abscess formation can occur as a complication of several of these conditions and may mimic or coexist with stercoral pathology. Careful integration of clinical features, risk factors (such as chronic constipation and immobility), laboratory results, and, most importantly, cross-sectional imaging helps distinguish stercoral colitis from these differential diagnoses and guides appropriate management [8].

Prognosis

The prognosis of stercoral colitis is closely linked to the timeliness of diagnosis and the rapidity with which effective management is instituted. Early recognition and prompt decompression of the colon can significantly reduce the risk of catastrophic complications, whereas delayed diagnosis often leads to a steep rise in morbidity and mortality. Among the prognostic factors, colonic perforation is the most critical; reported mortality rates in patients with stercoral perforation range from 32% to 59%, reflecting the fulminant nature of fecal peritonitis and the high likelihood of septic shock in this setting.[3] Another major predictor of poor outcome is the extent of colonic involvement—segments greater than 40 cm are associated with more profound ischemia, greater systemic inflammatory response, and higher risk of treatment failure. Ischemic bowel, as suggested clinically by severe pain, metabolic acidosis, and elevated lactic acid levels, also portends a worse prognosis. Patients who present in septic shock, with hypotension and multiorgan dysfunction, frequently require prolonged intensive care and have a substantially increased risk of death despite aggressive management. Interestingly, current literature has not demonstrated a significant correlation between age or sex and mortality in stercoral colitis.[3] However, this may reflect the underdiagnosis and underreporting of the condition rather than a true absence of demographic influence. Many cases are only discovered intraoperatively or at autopsy, and mild or early presentations may never be formally identified as stercoral disease. For patients without ischemia or perforation who receive early, appropriate non-operative management, the prognosis is more favorable, particularly when long-term constipation is aggressively addressed to prevent recurrence. Nonetheless, even survivors of severe stercoral colitis remain at risk for future episodes if underlying risk factors—such as chronic constipation, neurologic or psychiatric disease, or opioid dependence—are not adequately managed. Overall, the prognosis is highly variable and depends on early recognition, the presence or absence of complications, comorbid conditions, and the adequacy of both acute and preventive care [3].

Complications

Stercoral colitis carries a substantial risk of serious and potentially fatal complications, particularly when diagnosis and treatment are delayed. The most feared complication is colonic perforation, which occurs when progressive pressure necrosis and ischemia extend through the full thickness of the bowel wall. Once perforation develops, fecal contamination of the peritoneal cavity leads to diffuse peritonitis, overwhelming sepsis, and a marked increase in mortality. For this reason, perforation is considered a major predictor of adverse outcomes.[3] Ischemic colitis can also occur in the absence of frank perforation, as sustained intraluminal pressure compromises both venous and

arterial blood flow, resulting in transmural ischemia, necrosis, and systemic inflammatory response. Sepsis and septic shock are common downstream complications when the bowel wall barrier is disrupted. Bacterial translocation and toxin release into the bloodstream can precipitate multiorgan dysfunction, including respiratory failure, cardiovascular collapse, hepatic injury, and disseminated intravascular coagulation. Renal failure may arise both from septic hypoperfusion and from mechanical factors; massive bowel dilation can exert extrinsic pressure on the ureters, contributing to obstructive uropathy and further worsening kidney function.[3] Urinary retention may also develop due to compression of the bladder outlet or reflex dysfunction related to severe pelvic distension. Patients managed non-operatively remain at risk of recurrent ulceration and subsequent perforation within previously affected colonic segments, particularly if the underlying constipation is not effectively resolved. Without a robust long-term bowel regimen, recurrent fecal impaction can reestablish the same pathological sequence. Operative management, while often life-saving, introduces its own set of complications. Anastomotic leakage following resection and reconnection of bowel segments is a serious postoperative risk, leading to recurrent sepsis, intra-abdominal abscess formation, and the potential need for reoperation or prolonged drainage.[3] Patients undergoing Hartmann procedures or colostomy creation face risks of stoma-related complications, wound infection, and prolonged functional impairment. Taken together, these complications underscore the importance of early detection, meticulous perioperative care, and comprehensive postoperative follow-up [3].

Postoperative and Rehabilitation Care

Postoperative and rehabilitation care in patients with stercoral colitis is critical to reducing morbidity, preventing recurrence, and optimizing long-term outcomes. All patients diagnosed with stercoral colitis should be admitted to the hospital, with the level of care determined by severity of illness, comorbidities, and the presence of complications. For those managed non-operatively, close inpatient monitoring is essential. This includes serial abdominal examinations to detect signs of worsening tenderness, peritonitis, or distension, as well as regular monitoring of laboratory values such as white blood cell count, lactic acid, and inflammatory markers to assess progression toward sepsis or ischemia. Repeat imaging—most commonly CT scanning—may be necessary if clinical status deteriorates or fails to improve, to reassess colonic dilation, wall integrity, and the presence of complications such as abscess or perforation. Patients who require emergent surgery for perforation or extensive ischemic bowel are typically critically ill and should be managed postoperatively in an intensive care or high-dependency setting. These individuals often need advanced hemodynamic monitoring, aggressive fluid resuscitation, vasopressor support, mechanical ventilation, and broad-spectrum antibiotics. Postoperative management focuses on controlling sepsis, maintaining adequate organ perfusion, and monitoring for complications such as anastomotic leak, wound infection, stoma dysfunction, or abscess formation.[3] Early involvement of nutrition services is important, as many patients are malnourished or catabolic; parenteral or enteral nutrition may be required, especially when large bowel segments have been resected. Rehabilitation extends beyond immediate postoperative care. Once stabilized, patients benefit from gradual mobilization, respiratory physiotherapy, and careful advancement of diet, tailored to their gastrointestinal function and surgical reconstruction. A key aspect of long-term care is the development of an individualized bowel regimen to prevent recurrent constipation. This may include fiber supplementation, osmotic and stimulant laxatives, scheduled toileting, and close outpatient follow-up. Education about recognizing early warning signs—such as reduced bowel frequency, straining, or increasing abdominal discomfort—is vital. Coordination between surgeons, internists, gastroenterologists, dietitians, and rehabilitation specialists ensures that postoperative care addresses both acute recovery and long-term prevention of recurrence [3].

Patient Education

Effective deterrence of stercoral colitis hinges on proactive management of chronic constipation and patient education aimed at breaking the cycle of fecal stasis and colonic overload. Patients at risk—particularly older adults, individuals with neurologic or psychiatric disorders, those taking constipating medications, and persons with limited mobility—should be counseled about the importance of maintaining regular bowel habits. Education should emphasize dietary strategies, including increasing intake of fiber-rich foods such as fruits, vegetables, and whole grains, as well as ensuring adequate daily fluid consumption to soften stool and

promote transit.[5] When lifestyle modification alone does not adequately control constipation, pharmacologic interventions should be introduced early. First-line agents include osmotic laxatives, which draw water into the bowel lumen, and stimulant laxatives, which enhance colonic motility. Patients should be instructed on proper use of these medications, the importance of regular dosing rather than episodic use, and the need to avoid overreliance on agents that may worsen constipation, such as opioids, when alternatives exist. A periodic review of home medications is essential to identify and, when possible, modify or discontinue drugs that contribute to bowel hypomotility.[5] For individuals who have already experienced complications of chronic constipation—such as fecal impaction or stercoral colitis—ongoing follow-up is critical. Referral to a dietitian can help patients develop sustainable dietary patterns tailored to their preferences and comorbid conditions. Consultation with a gastroenterologist may be necessary to evaluate for secondary causes of constipation, particularly in refractory cases. Diagnostic tools such as colonoscopy, anorectal manometry, barium enema studies, and colonic transit tests can identify structural, neuromuscular, or functional abnormalities that may require targeted therapy.[5] Equally important is educating patients and caregivers about early warning signs of severe constipation, such as progressive reduction in stool frequency, persistent straining, abdominal bloating, or pain. Caregivers of cognitively impaired or bedbound patients should be trained to monitor bowel patterns and seek medical evaluation promptly when concerns arise. By promoting awareness, encouraging preventive habits, and integrating multidisciplinary follow-up, healthcare providers can significantly reduce the likelihood of stercoral colitis and its serious sequelae [5].

Other Issues

Several key points should guide clinicians when considering stercoral colitis in the differential diagnosis of abdominal pain. First, the condition frequently mimics more common intra-abdominal disorders such as diverticulitis, large bowel obstruction, or ischemic colitis, which can lead to diagnostic delay. A high index of suspicion is therefore essential, particularly in high-risk populations—elderly, immobile, neurologically impaired, or chronically constipated patients. Any patient with severe constipation, abdominal pain, and systemic signs of inflammation should prompt consideration of stercoral pathology, especially when imaging shows marked fecal loading and colonic distention.[1] Second, timely recognition and appropriate management are crucial for preventing catastrophic complications. Once ischemic colitis, perforation, or sepsis develops, morbidity and mortality rise dramatically. Early CT imaging with contrast is invaluable for distinguishing uncomplicated fecal impaction from stercoral colitis, as findings such as wall thickening, mucosal discontinuity, and pericolonic fat stranding indicate more advanced disease requiring closer monitoring or intervention. Management decisions should be primarily guided by the presence or absence of sepsis, bowel ischemia, or perforation rather than solely by the degree of fecal loading.[3] Another important clinical pearl is that stercoral colitis is, at its core, a preventable condition. Aggressive treatment of chronic constipation, avoidance or careful monitoring of constipating medications, and proactive bowel regimens in at-risk patients can significantly reduce incidence. For those who have suffered from stercoral colitis, long-term preventive strategies must be integrated into their care plan to prevent recurrence. Finally, clinicians should recognize that stercoral colitis remains under-recognized and underreported; raising awareness through education, case reporting, and incorporation into clinical teaching can improve early detection and outcomes. In summary, vigilance, early imaging, and prompt, etiology-focused management are the cornerstones of minimizing the substantial morbidity and mortality associated with this condition.[1][3]

Enhancing Healthcare Team Outcomes

The complex and often rapidly progressive nature of stercoral colitis makes it a condition ideally managed by an interprofessional healthcare team. Because it is relatively rare and under-documented, many clinicians may be unfamiliar with its presentation and management, underscoring the importance of education across disciplines about its risk factors, clinical features, and diagnostic hallmarks.[10] Early involvement of multiple specialties can significantly improve patient outcomes. Internists or hospitalists typically coordinate initial evaluation, stabilization, and the workup for abdominal pain and sepsis. Radiologists play a central diagnostic role by interpreting CT scans and identifying key features such as fecalomas, colonic wall thickening, mucosal discontinuity, and perforation. A surgical consultation should be

obtained early in the course of suspected stercoral colitis, even when patients are initially hemodynamically stable, given the high risk of sudden deterioration and need for emergent operative management. Gastroenterologists may assist with endoscopic disimpaction, evaluate for underlying colorectal pathology, and guide long-term constipation management. Pharmacists contribute by optimizing antibiotic regimens based on culture results, reviewing medications for constipating side effects, and recommending bowel regimens tailored to the patient's overall condition. Infectious disease specialists can provide input on complex or resistant infections and sepsis management. In critically ill patients, intensivists oversee hemodynamic support, organ function monitoring, and ventilatory management in the intensive care unit. Nurses and allied health professionals are vital to continuous patient monitoring, early recognition of clinical deterioration, and implementation of bowel regimens. Dietitians help design individualized nutrition plans that promote bowel regularity, while social workers and case managers address psychosocial issues, arrange follow-up care, and help mitigate contributory factors such as inadequate home support or limited access to appropriate nutrition. After acute management, outpatient follow-up with gastroenterology or primary care is essential to investigate and address the cause of fecal impaction, whether it stems from chronic constipation, neuropsychiatric disease, opioid use, or mobility limitations. Coordination with psychiatry, pain management, or neurology may be needed to modify contributing conditions or therapies [10]. Ultimately, stercoral colitis exemplifies a condition in which interdisciplinary communication and shared decision-making directly affect survival. By working collaboratively—sharing information promptly, adjusting treatment plans in response to evolving clinical data, and jointly focusing on both acute care and long-term prevention—the healthcare team can improve outcomes in this rare but highly morbid disease.[10]

Conclusion:

In conclusion, stercoral colitis represents a severe and often preventable consequence of unmanaged chronic constipation. Its pathogenesis involves a cascade from fecaloma formation and colonic distension to pressure necrosis, ischemia, and potential perforation. Timely diagnosis is paramount and relies on a high index of suspicion in vulnerable patients, confirmed by characteristic findings on contrast-enhanced CT imaging. Management is dictated by disease severity: stable patients may be treated conservatively with decompression and medical support, while evidence of peritonitis or perforation necessitates urgent surgical resection and fecal diversion. The condition carries a soberingly high mortality, particularly when complicated by perforation. Ultimately, prevention through aggressive, long-term management of constipation in at-risk individuals is the most effective strategy. Optimal care demands a coordinated, interprofessional approach to ensure rapid diagnosis, appropriate intervention, and comprehensive follow-up to mitigate recurrence.

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