e-CliniC 2026; Vol. 14, No. 2: 339-348
DOI: https://doi.org/10.35790/ecl.v14i2.67458
URL Homepage: https://ejournal.unsrat.ac.id/index.php/eclinic

Ileal Perforation in Geriatric Patients: A Systematic Review of Etiology and Histopathological Findings

Jeannchristy A. Pondaag,[1] Yulius A. M. Chietra,[2] Andly R. Mallo[3]

[1]General Practitioner, Manado, Indonesia
[2]Department of Surgery, RSUD Trikora Salakan, Banggai Islands Regency, Indonesia
[3]Department of Surgery, RSAL dr. R. Oetojo Sorong, Southwest Papua, Indonesia
Received: April 27, 2026; Accepted: July 6, 2026; Published online: July 16, 2026

Abstract: Ileal perforation in elderly patients is a rare but life-threatening surgical emergency associated with high morbidity and mortality. Its heterogeneous etiologies and atypical clinical presentations in geriatric populations often result in delayed diagnosis and poor outcomes. Understanding the underlying etiologies and histopathological characteristics is essential to improve diagnostic accuracy and clinical management. This study aimed to systematically evaluate the etiologies and histopathological findings of ileal perforation in patients aged ≥60 years. This was a systematic literature search was conducted in CLIB (The Cochrane Library), Embase, MEDLINE, PubMed, Scopus, Web of Science and Google Scholar for studies published between 2006 and 2026. Eligibility criteria included case reports, case series, observational studies, or randomized controlled trials studies involving geriatric patients with ileal perforation and available histopathological data or macroscopic findings. Study selection, data extraction, and quality assessment were performed independently by two reviewers using the Joanna Briggs Institute (JBI) Critical Appraisal Checklist. A total of 10 studies were included, all of which were case reports. The results showed that the included studies demonstrated heterogeneous etiologies, including infections, NSAID-induced injury, diverticular disease, trauma, ischemia, foreign bodies, and neoplasms such as gastrointestinal stromal tumors. Despite etiological diversity, all studies consistently reported transmural inflammation and necrosis as the final common histopathological pathway. Specific findings reflected underlying mechanisms, such as granulomatous inflammation in infectious causes and vascular thrombosis in ischemic conditions. In conclusion, ileal perforation in geriatric patients is characterized by diverse etiologies but a convergent histopathological pathway of transmural tissue destruction. Delayed diagnosis due to atypical clinical presentation significantly contributes to poor outcomes, emphasizing the importance of early recognition, comprehensive evaluation, and timely surgical intervention.
Keywords: ileal perforation; geriatric patients; histopathology; peritonitis; small bowel perforation; etiology

INTRODUCTION

Ileal perforation is defined as a loss of transmural integrity of the ileal wall, allowing the leakage of intestinal contents into the peritoneal cavity.[1-3] This condition directly leads to secondary peritonitis due to contamination by enteric bacteria and fecal material, triggering a localized peritoneal inflammatory response that may progress to systemic inflammation.[3,4] If not promptly managed, this process can rapidly evolve into sepsis and septic shock, with a high risk of mortality.[3-5] Necrosis of Peyer's patches has been recognized as an important contributing factor in intestinal perforation, particularly in certain infectious conditions.[1,2,5]

Ileal perforation may arise from a wide spectrum of etiologies, including infectious causes such as tuberculosis and typhoid fever, as well as non-infectious conditions including nonsteroidal anti-inflammatory drug (NSAID) use, intestinal ischemia, inflammatory bowel disease, malignancy, trauma, foreign body ingestion, and iatrogenic complications.[6–13] The underlying pathogenesis generally involves a progressive inflammatory process leading to mucosal injury, bowel wall necrosis, and eventual perforation, which precipitates peritonitis.[1,2,13]

Although less common than gastroduodenal perforation, ileal perforation remains a highly fatal surgical emergency, particularly in geriatric populations.[1,2,14–16] Aging is associated with an increased burden of comorbidities, reduced physiological reserve, and exposure to risk factors such as smoking, alcohol consumption, and NSAID use, all of which contribute to atypical clinical presentations and increase the likelihood of delayed or missed diagnosis.[14–16] Consequently, elderly patients are more susceptible to rapid progression toward diffuse peritonitis, sepsis, and multiple organ dysfunction syndrome (MODS), leading to significantly higher mortality rates.[3,4,15,16] Advanced age (≥60 years), presence of comorbidities, and development of MODS have been identified as independent predictors of mortality in perforation-related peritonitis.[15]

Despite its clinical significance, the majority of available literature on ileal perforation predominantly focuses on younger populations, while studies specifically addressing geriatric patients remain limited. Furthermore, observational studies and clinical trials are scarce, and histopathological findings are often inconsistently reported across studies. This lack of comprehensive and systematically synthesized evidence limits a deeper understanding of the disease process, particularly in elderly patients with complex comorbidities.

In addition, variations in reported etiologies and histopathological features across different studies indicate a lack of consensus regarding the underlying mechanisms of ileal perforation in geriatric populations. This highlights the need for a systematic evaluation integrating both clinical and histopathological perspectives.

Given the increasing burden of elderly patients and the high mortality associated with delayed diagnosis in acute abdominal emergencies, this issue is particularly relevant in clinical practice, including in developing countries such as Indonesia, where early diagnosis and timely surgical intervention remain challenging. Therefore, this systematic review aims to synthesize current evidence regarding the clinical characteristics, etiologies, and histopathological findings of ileal perforation in patients aged ≥60 years. The article is structured to present the methodology of study selection and analysis, followed by the synthesis of findings and discussion of their clinical implications in geriatric surgical care.

METHODS

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines.[17] The review was registered in PROSPERO (ID: CRD420261352895).

Studies were included based on PICOS criteria: Population—patients aged ≥60 years or studies providing extractable data for this age group; Intervention/Exposure—ileal or small bowel perforation causing peritonitis; Comparison—not restricted; Outcome—clinical characteristics, etiologies, and histopathological findings; Study design—case reports, case series, observational studies, or randomized controlled trials. Studies published between 2006 and 2026 in English or

Indonesian with accessible full text were included. Exclusion criteria included animal studies, editorials, commentaries, letters without original data, studies not specifically addressing ileal perforation or peritonitis, incomplete data, and duplicate publications.

A systematic literature search was conducted in CLIB (The Cochrane Library), Embase, MEDLINE, PubMed, Scopus, and other important databases such as Web of Science and Google Scholar. The search covered articles published from 2006 to 2026. Keywords included "ileal perforation", "ileum perforation", "terminal ileum perforation", "small bowel perforation", "peritonitis", "elderly", and "geriatric", "aged", "histopathology", "etiology", "causes", combined using Boolean operators (AND/OR).

Study selection was performed independently by two authors following PRISMA 2020 guidelines. Screening was conducted in two stages: title and abstract screening followed by full-text review. Discrepancies were resolved through discussion and consensus, and when necessary, a third author acted as an adjudicator. Data extracted from each study included author and year of publication, patient demographics (age and sex), etiology of perforation, clinical presentation, and histopathological findings.

The methodological quality of included studies was assessed using the Joanna Briggs Institute (JBI) Critical Appraisal Checklist.[18] Assessment was conducted independently by both authors, with disagreements resolved by consensus. Given the descriptive nature of the included studies, no quantitative effect measures were applied. Outcomes were summarized qualitatively.

A narrative synthesis approach was used to summarize the findings. Data were categorized based on etiologies and corresponding histopathological patterns. The synthesis focused on identifying common pathways and variations across different etiological groups.

RESULTS

In study selection, a systematic literature search was conducted across CLIB, Embase, MEDLINE, PubMed, Scopus, Web of Science and Google Scholar. Initially, the search was limited to the past 10 years; however, due to the limited number of studies specifically reporting ileal perforation in patients aged ≥60 years, the search period was extended to 20 years (2006–2026). Earlier literature (2006–2013) demonstrated significant limitations, with most studies focusing on younger populations or reporting gastrointestinal perforation without specific stratification by anatomical location and age.

A total of 332 records were identified. After screening 260 records based on title and abstract, 219 were excluded. Forty-one full-text articles were assessed for eligibility, of which 31 were excluded due to the absence of specific data on patients aged ≥60 years or limited full-text access. Ultimately, 10 studies met the inclusion criteria, all of which were case reports describing ileal perforation in elderly patients with peritonitis. The study selection process is illustrated in the PRISMA 2020 flow diagram (Figure 1).

All 10 included studies were case reports published between 2006 and 2026. These studies originated from various regions, including Asia and Africa, indicating a geographically diverse but limited evidence base. No observational studies or randomized controlled trials specifically addressing ileal perforation in geriatric populations were identified within the search period.
Overall, the available evidence on ileal perforation in elderly patients remains scarce and is predominantly based on individual case reports. Detailed characteristics of the included studies are presented in Table 1.

Across the 10 included case reports, all patients presented with acute peritonitis, characterized by diffuse abdominal pain, abdominal distension, and signs of peritoneal irritation requiring urgent surgical intervention. In geriatric patients, clinical presentations were frequently atypical due to reduced physiological reserve and diminished inflammatory response, leading to delayed diagnosis. Such delays were associated with rapid progression to diffuse peritonitis, sepsis, multiple organ dysfunction syndrome (MODS), and death, particularly in cases with prolonged symptom duration prior to definitive intervention.[19,20,25,27]

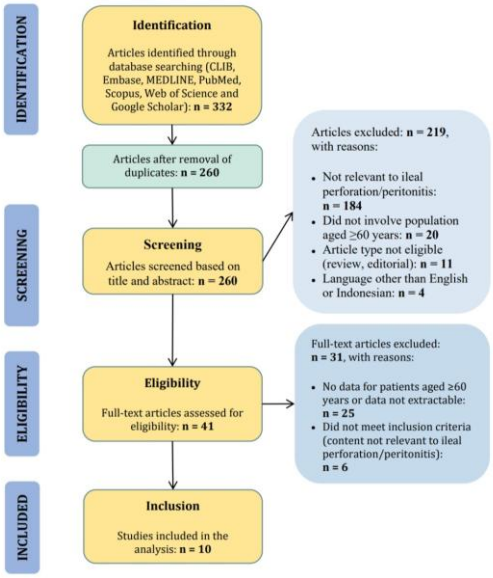

Figure 1. PRISMA 2020 flow diagram illustrating the study selection process.

Related to clinical findings, etiologies, and histopathology, across the 10 included case reports, all patients presented with acute peritonitis, characterized by diffuse abdominal pain, abdominal distension, and signs of peritoneal irritation requiring urgent surgical intervention. In geriatric patients, clinical presentations were frequently atypical due to reduced physiological reserve and diminished inflammatory response, leading to delayed diagnosis. Such delays were associated with rapid progression to diffuse peritonitis, sepsis, multiple organ dysfunction syndrome (MODS), and death, particularly in cases with prolonged symptom duration prior to definitive intervention.[19,20,25,27]

The etiologies of ileal perforation were heterogeneous, reflecting diverse pathophysiological mechanisms leading to transmural bowel wall damage. Infectious causes, such as tuberculosis and brucellosis, demonstrated chronic granulomatous inflammation or acute severe inflammatory infiltration resulting in progressive tissue destruction.[19,23] Ischemic etiologies, including strangulated hernia and vascular compromise, led to hypoperfusion, necrosis, and loss of bowel wall integrity.[20,27]

Iatrogenic and mechanical factors exhibited distinct mechanisms but similar outcomes. NSAID-related injury resulted in mucosal damage progressing to ulceration and perforation.[21] Mechanical causes, including foreign body ingestion and blunt abdominal trauma, led to direct structural injury to the bowel wall.[24,26] Local inflammatory processes were also observed in non-Meckel ileal diverticulitis, which could progress to abscess formation and perforation.[22] Neoplastic processes, particularly gastrointestinal stromal tumors (GIST), caused perforation through tumor necrosis and local invasion.[28] Some cases were classified as non-specific, demonstrating extensive destructive inflammation without a clearly identifiable cause.[25]

Histopathologically, all studies demonstrated a consistent pattern of transmural inflammation and necrosis, representing the final common pathway in ileal perforation. Mixed inflammatory cell infiltration (neutrophils, lymphocytes, plasma cells, and histiocytes) involving all layers of the bowel wall reflected a progressive injury response.[19–28] Specific findings, such as granulomas with acid-fast bacilli in tuberculosis,[19] submucosal venular thrombosis and crypt loss in ischemia,[27] necrotic debris in NSAID-induced injury,[21] and spindle cell proliferation with necrosis in GIST,[28] further supported the relationship between etiology and tissue damage mechanisms.

The methodological quality of the included studies was evaluated using the Joanna Briggs Institute (JBI) Critical Appraisal Checklist for Case Reports. As shown in Table 2, all studies consistently met the eight evaluated domains, including clarity of patient demographics, timeline,

clinical condition, diagnostic methods, intervention details, post-operative condition, reporting of adverse events, and key clinical lessons. This indicates that the studies adhered well to established reporting standards for case reports.

Several biases were noted across the included case reports. Confounding factors, such as nutritional status and cardiac comorbidities, were not consistently accounted for in systemic conditions like tuberculosis[19] and ischemic enteritis[27]. Selection and publication bias were evident, as rare etiologies (e.g., gastrointestinal stromal tumors[28], brucellosis[23]) were disproportionately represented. Information and recall bias occurred in cases of foreign body ingestion, where patient recollection was unreliable.[26] Limitations in access to diagnostic tests or specialized evaluations affected the completeness of clinical assessment in some reports, potentially influencing interpretation and generalizability of findings.

However, the overall risk of bias is still considered moderate due to inherent limitations of the case report design. These include the absence of control groups and the selective reporting of rare or unusual cases, which may not reflect the full clinical spectrum. No subgroup, sensitivity, or quantitative heterogeneity analyses could be performed due to the nature and small number of included reports.

All included studies were case reports describing ileal perforation in patients aged ≥60 years. Due to the descriptive nature of the data, no quantitative effect estimates were calculated. Key study characteristics, including patient demographics, etiologies, and histopathological findings, are presented in Table 1.

Across studies, all patients presented with acute peritonitis requiring surgical intervention. Etiologies were heterogeneous, including infectious, ischemic, drug-induced, mechanical, diverticular, and neoplastic causes. Histopathological findings consistently demonstrated transmural inflammation and necrosis, with variations reflecting the underlying etiology.

A narrative synthesis was performed due to the heterogeneity of study designs and outcomes, and the absence of data suitable for meta-analysis.

Across all included studies, a consistent pathological pattern of transmural inflammation and necrosis was identified as the final common pathway of ileal perforation. Despite etiological variability, this finding was observed uniformly across cases.

The overall risk of bias was moderate, primarily due to the inherent limitations of case reports, including lack of control groups and unaddressed confounding factors. No subgroup, sensitivity, or quantitative heterogeneity analyses were conducted.

DISCUSSION

The findings of this review underscore the multifaceted nature of ileal perforation in the geriatric population, where the etiology often shifts from predominantly infectious agents seen in younger patients to vascular and iatrogenic causes. The "masquerading normalcy" of early symptoms in the elderly, combined with physiological attenuation of inflammatory responses, contributes significantly to the observed high mortality rates. The increasing global life expectancy has heightened attention to the geriatric population, particularly in the context of abdominal surgical emergencies.[29] Perforation of the ileum in elderly patients is relatively rare but carries significant clinical consequences, with a wide spectrum of etiologies, including infectious causes such as tuberculosis[19] and brucellosis,[23] incarcerated obturator hernia,[20] nonsteroidal anti-inflammatory drug (NSAID) use,[21] non-Meckel ileal diverticulum,[22] trauma,[24] foreign bodies,[26] ischemia,[27] and neoplasms such as gastrointestinal stromal tumor (GIST)[28]. Despite this etiologic heterogeneity, the findings of this review reveal a consistent histopathological pattern of transmural inflammation and necrosis as the final common pathway of ileal wall injury. Only one included case report lacked microscopic descriptions of the resected specimen[20].

Pathophysiologically, ileal perforation in geriatric patients can be understood as the outcome of a progressive destructive process, in which initial injury—whether inflammatory, ischemic, infectious, or traumatic—extends from the mucosa to involve the full thickness of the intestinal

wall. Histopathological findings, including mixed inflammatory cell infiltration, extensive necrosis, and vascular alterations such as submucosal thrombosis, support the concept that perforation is not an isolated acute event but rather the end stage of failed protective and reparative mechanisms. This pattern appears consistent across different etiologies, demonstrating convergence of pathogenic pathways despite differing initiating factors.

In infectious etiologies, chronic granulomatous inflammation in tuberculosis[19] and acute massive inflammation in brucellosis[23] indicate that both chronic and systemic infections can progressively destroy intestinal tissue until perforation occurs. In ischemic conditions, perfusion disturbances due to strangulation or vascular thrombosis[20,27] directly lead to transmural necrosis. Similar mechanisms are observed in blunt abdominal trauma,[24] where secondary vascular injury triggers ischemia and tissue damage.

Iatrogenic and mechanical factors demonstrate different mechanisms but result in the same final outcome. NSAID use[21] causes mucosal injury through disruption of mucosal defenses and increased permeability, progressing to ulceration and perforation. Foreign bodies[26] and ileal diverticulitis[22] induce local injury via mechanical trauma and progressive focal inflammation. In neoplastic etiologies such as GIST[28], perforation results from tumor necrosis and local invasion, representing an advanced destructive process with poorer prognostic implications.

Interestingly, despite differing etiologies, all cases exhibit similar histopathological patterns, confirming that transmural inflammation and necrosis constitute a final common pathway in the pathogenesis of ileal perforation. This emphasizes that etiological identification relies not only on clinical presentation but also on comprehensive histopathological evaluation to understand the underlying disease mechanisms.

Clinically, ileal perforation in elderly patients is often challenging to recognize early. Age-related immunosenescence results in suboptimal inflammatory responses, causing classic signs such as fever or leukocytosis to be less pronounced. Coupled with reduced physiological reserve and high comorbidity prevalence, these factors contribute to delayed diagnosis and rapid progression to sepsis, septic shock, and multiple organ dysfunction syndrome (MODS).[25,27] Delayed surgical intervention, as observed in several cases within this review, is directly associated with increased mortality.

From a broader perspective, the findings underscore the importance of heightened clinical vigilance, rapid and comprehensive diagnostic approaches, and timely surgical intervention in reducing mortality from ileal perforation in elderly patients. Additionally, there is a critical need for improved data collection through national registries and multicenter studies to better understand epidemiology, risk factors, and outcomes in this vulnerable population. Despite its relative rarity, ileal perforation must be considered in the differential diagnosis of acute abdomen in geriatric patients, particularly when clinical manifestations are subtle or atypical. Failure to recognize this condition early may narrow the therapeutic window and contribute to progression toward advanced peritonitis and poor outcomes.

This review is limited by the small number of included studies and the predominance of case report designs, requiring cautious generalization of findings. Variability in clinical and histopathological reporting also introduces data heterogeneity. Nevertheless, the consistent presence of transmural inflammation and necrosis reinforces the concept that ileal perforation represents the terminal stage of a progressive pathological process. Early recognition in geriatric patients remains challenging due to atypical presentation and immunosenescence, with delayed diagnosis closely correlated with increased risk of sepsis, multiple organ dysfunction syndrome (MODS), and mortality. These findings emphasize that timely surgical intervention, coupled with high clinical awareness, is a primary determinant in reducing mortality, especially in patients with comorbidities.

CONCLUSION

Ileal perforation in geriatric patients represents a surgical emergency with heterogeneous etiology but exhibits a consistent final common pathway characterized by transmural

inflammation and bowel wall necrosis. Clinical manifestations in older adults are often atypical, contributing to delayed diagnosis and rapid progression to sepsis and multi-organ dysfunction. Therefore, high clinical vigilance, comprehensive diagnostic approaches, and timely surgical intervention are key determinants in improving outcomes. Integration of histopathological findings is crucial for establishing a definitive diagnosis and guiding therapy, including for rare etiologies such as gastrointestinal stromal tumors (GIST).

Further studies with stronger designs, such as large-scale cohort or observational studies, are warranted to identify risk factors, distribution of etiologies, and clinical outcomes more comprehensively in geriatric populations. Standardization of histopathological reporting and integration with clinical data are also essential to improve diagnostic accuracy and support precise therapeutic decision-making.

Conflict of Interest

The authors declare no conflicts of interest in this study.

Acknowledgments

The authors thank all researchers whose work contributed to this review. This manuscript is also dedicated to the memory of the father of one of the authors, whose clinical course of ileal perforation and peritonitis served as a profound reflection on the complexity of diagnosis and urgency of management in abdominal emergencies. This experience not only inspired scientific inquiry but also highlighted the fatal consequences of delayed diagnosis and intervention, which can rapidly progress to sepsis, multi-organ failure, and death.

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions

All authors approved the final version of the manuscript. Moreover, the authors declare that no AI-assisted technologies were used in the writing of this manuscript.

REFERENCES

1. Gan T, Evers BM. Small intestine. In: Townsend CM, Beauchamp RD, Evers BM, Mattox KL, editors. Sabiston Textbook of Surgery (21st ed). Philadelphia: Elsevier; 2022. p. 1240–300.
2. Landmann A, Bonds M, Postier R. Acute Abdomen. In: Townsend CM, Beauchamp RD, Evers BM, Mattox KL, editors. Sabiston Textbook of Surgery (21st ed. Philadelphia: Elsevier; 2022. p. 1134–1149.
3. Sartelli M, Chichom-Mefire A, Labricciosa FM, Hardcastle T, Abu-Zidan FM, Adesunkanmi AK, et al. The management of intra-abdominal infections from a global perspective: 2017 WSES guidelines for management of intra-abdominal infections. World J Emerg Surg. 2017;12:29. Doi: https://doi.org/10.1186/s13017-017-0141-6. Erratum in: World J Emerg Surg. 2017 Aug 2;12:36. Doi: 10.1186/s13017-017-0148-z
4. Singer M, Deutschman CS, Seymour CW, Shankar-Hari M, Annane D, Bauer M, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 20163;315(8):801-10. Doi: https://doi.org/10.1001/jama.2016.0287
5. Ashok E, Ekka NM, Sinha DK, Gaurav K, Kumar B, Ahmad N, et al. Clinical presentations, treatments, and complications of ileal perforation at a tertiary center: a cross-sectional study. Cureus. 2024;16(10):e72027. Doi: https://doi.org/10.7759/cureus.72027
6. Onken F, Senne M, Königsrainer A, Wichmann D. Classification und treatment algorithm of small bowel perforations based on a ten-year retrospective analysis. J Clin Med. 2022;11(19):5748. Doi: https://doi.org/10.3390/jcm11195748
7. Shrimanker N, Heller NP, Souza F, Kim DE. Late anastomotic perforation of the ileum 3 years after intestinal resection. BMJ Case Rep. 2024;17(8):e260668. Doi: https://doi.org/10.1136/bcr-2024-260668
8. Thiéfin G, Beaugerie L. Toxic effects of nonsteroidal antiinflammatory drugs on the small bowel, colon, and rectum. Joint Bone Spine. 2005;72(4):286-94. Doi: https://doi.org/10.1016/j.jbspin.2004.10.004
9. Maccioni L, Fu Y, Horsmans Y, Leclercq I, Stärkel P, Kunos G, Gao B. Alcohol-associated bowel disease: new insights into pathogenesis. eGastroenterology. 2023;1(1):e100013. Doi: https://doi.org/10.1136/egastro-2023-100013

10. Shringi B, Rathi V, Meena P, Singh K. Multifocal small bowel gastrointestinal stromal tumor causing perforation peritonitis: a rare cause of acute abdomen. Radiol Case Rep. 2026;21(5):2087-94. Doi: https://doi.org/10.1016/j.radcr.2026.01.080
11. Sartelli M, Coccolini F, Kluger Y, Agastra E, Abu-Zidan FM, Abbas AES, et al. WSES/GAIS/SIS-E/WSIS/AAST global clinical pathways for patients with intra-abdominal infections. World J Emerg Surg. 2021;16:49. Doi: https://doi.org/10.1186/s13017-021-00387-8
12. Ho MJ, Kyang LS, Schlichtemeier S. Bowel perforation with generalised peritonitis secondary to recurrent primary fallopian tube carcinoma after 17 years. BMJ Case Rep. 2024;17(4):e258764. Doi: https://doi.org/10.1136/bcr-2023-258764
13. Jones MW, Kashyap S, Boget B, Zabb CP. Bowel perforation. Treasure Island (FL): StatPearls Publishing; 2026. Available from: https://www.ncbi.nlm.nih.gov/books/NBK537224/
14. Norman DC. Clinical features of infection in older adults. Clin Geriatr Med. 2016;32(3):433-41. Doi: https://doi.org/10.1016/j.cger.2016.02.005
15. Dananjaya AAGP, Gede S, Nyoman G. Validitas faktor-faktor risiko terjadinya kematian pada pasien peritonitis perforasi di RSUP Sanglah Denpasar. Medicina. 2019;50(1):221–7. Doi: https://doi.org/10.15562/medicina.v50i1.385
16. Kim HS, Kim HI, Yoon YJ, Yeom JH, Kim MG. Analysis of prognostic factors for postoperative complications and mortality in elderly patients undergoing emergency surgery for intestinal perforation or irreversible intestinal ischemia. Ann Surg Treat Res. 2023;105(4):198–206. Doi: https://doi.org/10.4174/astr.2023.105.4.198
17. Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ. 2021;372:n71. Doi: https://doi.org/https://doi.org/10.1136/bmj.n71
18. Moola S, Munn Z, Tufanaru C, Aromataris E, Sears K, Sfetcu R, et al. Chapter 7: Systematic reviews of etiology and risk. In: Aromataris E, Munn Z (Editors). JBI Manual for Evidence Synthesis. JBI. 2020. Available from https://synthesismanual.jbi.global
19. Leung VK, Chu W, Lee VH, Chau TN, Law ST, Lam SH. Tuberculosis intestinal perforation during anti-tuberculosis treatment. Hong Kong Med J. 2006;12(4):313-5. Available from: https:// pubmed.ncbi.nlm.nih.gov/16912360/
20. Zhang H, Cong JC, Chen CS. Ileum perforation due to delayed operation in obturator hernia: a case report and review of literatures. World J Gastroenterol. 2010;16(1):126-30. Doi: https://doi.org/10.3748/wjg.v16.i1.126
21. Park WS, Kim SW, Lee S, Lee ST, Park HS. Multiple ileal perforations due to regular diclofenac sodium injections: a case report. BMC Res Notes. 2013;6(1):129. Doi: https://doi.org/10.1186/1756-0500-6-129
22. Rajaguru K, Sheong SC. Case report on a rare cause of small bowel perforation perforated ileal diverticulum. Int J Surg Case Rep. 2021;87:106465. Doi: https://doi.org/10.1016/j.ijscr.2021.106465
23. Wang C, Yang X, Zhou J, Liu X, Yang Y, Li X. Perforation of the terminal ileum caused by brucellosis: a case report. Clin Case Rep. 2024;12(2):e8496. Doi: https://doi.org/10.1002/ccr3.8496
24. Haqim RM, Nirbira A, Setyawan N. Delayed peritonitis due to ileal perforation from blunt abdominal trauma in Sardjito Hospital. Jurnal Bedah Nasional. 2024;8(2):S67. Doi: https://doi.org/10.24843/JBN.2024.v08.is02.p067
25. Rifqyan A, Islami AAN, Bellynda W, Tinelo IF, Hertanto ACH. Generalized peritonitis due to multiple perforation and necrosis of ileum: a case report. J Agromed Med Sci. 2024;10(3):127–31. Doi: https://doi.org/10.19184/ams.v10i3.4736
26. Panday B, Bhatta OP, Gyawali P, Dangol RS, Joshi MD, Neupane BR. Ileal perforation peritonitis secondary to accidental ingestion of a chicken bone: A case report. Int J Surg Case Rep. 2024;123(C):110284. Doi: https://doi.org/10.1016/j.ijscr.2024.110284
27. Gebrehiwet CL, Abereha AY, Mulualem DA, Gurmu ML, Abera SA, Tadesse AK, et al. Spontaneous multiple small bowel perforations due to ischemic enteritis: a case report. BMC Geriatr. 2025;25(1):263. Doi: https://doi.org/10.1186/s12877-025-05911-9
28. Zivanovic M, Mitrovic-Jovanovic M, Stosic K, Bidzic N, Vasin D, Sekulic D, et al. Spontaneous terminal ileum GIST perforation causing an acute abdomen in an elderly patient – a rare case. Diagnostics (Basel). 2025;15(14):1816. Doi: https://doi.org/10.3390/diagnostics15141816
29. Crimmins EM, Beltrán-Sánchez H. Mortality and morbidity trends: is there compression of morbidity? J Gerontol B Psychol Sci Soc Sci. 2011;66(1):75-86. Doi: https://doi.org/10.1093/geronb/gbq088

Table 1. Study characteristics and histopathological findings of ileal perforation in patients aged ≥60 years

No.	Authors, Year	Study types	Age (Years)	Sex	Etiology	Histopathological Findings	Outcome
1.	Leung et al, 2006[19]	Case report	63	M	Tuberculosis	Non-caseating granulomas, and AFB were identified on Ziehl-Neelsen staining	Alive
2.	Zhang et al, 2010[20]	Case report	83	F	Incarcerated obturator hernia	(Gross) stenosis at the terminal ileum, perforation of small bowel was noted proximal to the port of incarceration	Alive (despite of the complication of multiple organ dysfunction syndrome)
3.	Park et al, 2013[21]	Case report	69	F	NSAID (diclofenac)	Nonspecific inflammation of the ileal wall, with ulceration and perforation; necrotic debris intermixed with predominantly neutrophilic inflammatory cells and granulation tissue formation. Inflammatory features exclude IBD, vasculitis, and vascular thrombi.	Alive
4.	Rajaguru et al, 2021[22]	Case report	74	M	Non-Meckel's diverticulosis	Acute diverticulitis of the terminal ileum with perforation and abscess formation.	Alive
5.	Wang et al, 2024[23]	Case report	69	M	Brucellosis	Massive full-thickness infiltration of neutrophils and lymphocytes in ileal wall adjacent to the perforation consistent with transmural necrosis.	Alive
6.	Haqim et al, 2024[24]	Case report	≥60		Blunt abdominal trauma	Non-specific (macroscopic: ischemic changes with perforation site observed intraoperatively)	Alive
7.	Rifqyan et al, 2024[25]	Case report	63	M	Non-spesific	Microscopic results of ileal tissues with almost the entire surface of mucosa was eroded, necrotic with extensive infiltration of neutrophil inflammatory cells, lymphocytes, plasma cells and histiocytes, with 2 atypical cells, prominent impression of nucleus and several mitoses and several focal mucosal gland tissues with columnar lining epithelium. There was no malignancy or specific infection process. At the end of resection, necrotic tissues are still visible,	Died
8.	Panday et al, 2024[26]	Case report	60	M	Foreign body ingestion (chicken bone)	Active inflammation and serositis around the zone of perforation	Alive
9.	Gebrehiwet et al, 2025[27]	Case report	75	M	Ischemic enteritis	Intense mixed inflammatory infiltrate with crypt loss and a submucosal venules luminal fibrin thrombus, flattened mucosal surface with ulceration, marked crypt destruction and transmural inflammation and necrosis	Alive
10.	Zivanovic et al, 2025[28]	Case report	86	M	Tumor (GIST)	Tumor composed of spindle-shaped cells with mild atypia; immunohistochemistry positive for smooth muscle actin, weak CD117, negative CD34 – consistent with GIST (atypical immunophenotype)	Alive

Abbreviations: M=Male; F=Female; AFB=Acid-fast bacilli; NSAID=Nonsteroidal anti-inflammatory drug; IBD=Inflammatory bowel disease; GIST=Gastrointestinal stromal tumor; CD117=known as c-kit or stem cell factor receptor; CD34=Cluster Differentiation 34, a transmembrane phosphoglycoprotein used in pathology as immunohistochemical marker

Table 2. Methodological quality assessment of included case reports using JBI checklist

Case Study	Demographics	Timeline	Clinical Condition	Diagnostics	Intervention	Post-op	Adverse Events	Lessons
Leung et al.[19]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Zhang et al.[20]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Park et al. [21]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Rajaguru et al.[22]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Wang et al.[23]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Haqim et al.[24]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Rifqyan et al.[25]	Yes	Yes	Yes	Yes	Yes	Yes*	Yes	Yes
Panday et al.[26]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Gebrehiwet et al.[27]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Zivanovic et al.[28]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Abbreviations: *) Rifqyan A, et al. (2024) reported mortality on day 7 post-surgery