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PAIN MANAGEMENT IN PERITONEAL CARCINOMATOSIS: A MECHANISM-BASED APPROACH

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ABSTRACT

Background

Peritoneal carcinomatosis usually represents an advanced stage of malignancy, most commonly of gastrointestinal or gynaecological origin. Pain in this condition is a significant clinical problem because it is often multifactorial and may arise from overlapping visceral, somatic, neuropathic, tumour related, and complication related mechanisms.

Aim

This narrative review aims to summarize current evidence on pain management in peritoneal carcinomatosis, with emphasis on tumour related mechanisms of pain, clinical complications of peritoneal metastases, and mechanism based therapeutic approaches.

Materials and methods

A narrative review was conducted using PubMed, Scopus, and Web of Science. The literature search was performed on 04.04.2026 and included publications from 2000 to 2026. The search combined terms related to peritoneal carcinomatosis, peritoneal metastases, cancer pain, pain mechanisms, pain management, malignant ascites, malignant bowel obstruction, visceral pain, somatic pain, and neuropathic pain. A total of 44 publications were analysed qualitatively. As this is a narrative review, no formal protocol, risk of bias assessment, or meta analysis was conducted.

Results

Pain in peritoneal carcinomatosis may arise from peritoneal infiltration, tumour associated inflammation, malignant ascites, bowel obstruction, visceral distension, somatic involvement of the parietal peritoneum or abdominal wall, and neuropathic mechanisms related to tumour invasion, nerve compression, or perineural invasion. Visceral pain is often the predominant pain phenotype, but somatic and neuropathic components may also contribute substantially to symptom burden. Pain management should be individualized and based on assessment of the dominant pain mechanism and relevant clinical complications. Pharmacological treatment remains central and may include opioids, non opioid analgesics, adjuvant analgesics, and corticosteroids, depending on the clinical presentation. Interventional procedures and treatment direct-

ed at malignant ascites or malignant bowel obstruction may be required in selected patients.

Conclusions

Pain in peritoneal carcinomatosis is heterogeneous and may change during disease progression. Management should be based on the dominant pain mechanism, clinical complications, overall clinical status, prognosis, and patient defined goals of care. Evidence specific to pain management in peritoneal carcinomatosis remains limited, and further studies are needed to clarify pain mechanisms and develop mechanism based treatment strategies for patients with peritoneal metastases.

Keywords: peritoneal carcinomatosis (PC), peritoneal metastases, mechanisms of pain, palliative care, malignant ascites, malignant bowel obstruction

INTRODUCTION

Peritoneal carcinomatosis (PC) refers to the dissemination of cancer cells across the peritoneal surfaces lining the abdominal and pelvic cavities. It typically represents an advanced stage of malignancies, most commonly of gastrointestinal and gynaecological origin, or less frequently, may occur as a primary malignancy of the peritoneum, such as peritoneal mesothelioma or primary peritoneal carcinoma [1].

The presence of peritoneal metastases is generally associated with an unfavourable prognosis. In many cases, PC reflects advanced stage disease with limited curative treatment options. Although outcomes for patients with peritoneal metastases have improved with the introduction of cytoreductive surgery and modern systemic therapies, this benefit is restricted to a selected group of patients with limited disease and favourable biology; thus, the majority of patients are not eligible for such interventions [2,3]. Consequently, management is frequently palliative, with a primary focus on symptom control and best supportive care [4,5]. Effective pain management is therefore a significant component of care in this patient population.

PC often remains clinically silent in its early stages or manifests with nonspecific symptoms, which is a major reason for delayed diagnosis. The most common initial presentations include abdominal distension, new onset ascites, and vague abdominal or pelvic pain, although symptoms may vary depending on the primary tumour type

[4,6]. With disease progression, symptom burden increases, with pain being one of the major factors impairing quality of life.

Although pain is one of the most frequent and clinically difficult symptoms in patients with peritoneal metastases, its pathophysiology is complex and often multifactorial [7,8]. In this setting, pain may arise from direct peritoneal tumour infiltration, tumour associated inflammation, malignant ascites, visceral distension, bowel obstruction, somatic involvement of the abdominal wall or diaphragm, and neuropathic mechanisms related to tumour invasion or anticancer treatment [4,8,9,10]. These mechanisms frequently coexist in the same patient and may change during disease progression, which makes pain assessment and treatment selection particularly challenging [7,11].

Despite the high clinical burden of pain in PC, available publications usually address this problem within the broader context of advanced cancer pain or palliative care [12,13]. Specific evidence focusing on pain mechanisms in peritoneal metastases and their direct implications for pharmacological, interventional, and supportive treatment remains limited [4,5]. This gap is clinically relevant because inadequate recognition of the dominant pain mechanism may result in suboptimal analgesic selection, excessive reliance on opioids, delayed use of adjuvant analgesics, and insufficient consideration of procedures aimed at ascites, bowel obstruction, or localized pain sources [14,15,16,17]. Therefore, a mechanism based synthesis of pain management in PC is needed to support more rational and individualized symptom control in patients with advanced intra-abdominal malignancies.

AIM

The aim of this narrative review is to summarize current evidence on pain management in peritoneal carcinomatosis, with emphasis on tumour related mechanisms of pain, clinical complications of peritoneal metastases, and mechanism based therapeutic approaches.

Objectives:

1. To describe the main tumour related and complication related mechanisms of pain in peritoneal carcinomatosis, including peritoneal infiltration, inflammation, ascites, bowel obstruction, and neuropathic involvement.

2. To summarize pharmacological, interventional, and supportive strategies used for pain control in patients with peritoneal carcinomatosis.
3. To discuss the clinical relevance and limitations of mechanism based pain management in the palliative care of patients with advanced intra-abdominal malignancies and peritoneal metastases.

MATERIALS AND METHODS

A narrative review was conducted to synthesise current evidence on mechanism-based management of pain in patients with peritoneal carcinomatosis. The literature search was conducted in PubMed, Scopus, and Web of Science, with the last search performed on 04.04.2026. The search combined terms related to peritoneal carcinomatosis and peritoneal metastases with terms related to pain management, cancer pain, analgesia, palliative care, malignant ascites, malignant bowel obstruction, visceral pain, somatic pain, and neuropathic pain.

The search strategy included the following keywords and combinations: “peritoneal carcinomatosis” OR “peritoneal metastases” OR “peritoneal metastasis” AND “pain management” OR “cancer pain” OR “analgesia” OR “opioids” OR “adjuvant analgesics” OR “interventional pain management” OR “palliative care” AND “malignant ascites” OR “bowel obstruction” OR “malignant bowel obstruction” OR “neuropathic pain” OR “visceral pain” OR “somatic pain”.

Due to the limited amount of recent data specifically addressing the management of pain associated with peritoneal carcinomatosis, studies focusing on the broader management of cancer-related pain were also included. Publications published prior to 2000 addressing the pathophysiology of pain were likewise considered. The exclusion criteria included publications unrelated to pain pathophysiology or management, studies focusing on non-cancer pain conditions, conference abstracts, editorials, letters, duplicate records, and articles lacking sufficient clinical or mechanistic relevance to the review objectives. A total of 44 publications were then analysed qualitatively. The sources in the final reference list included original research, narrative and systematic reviews, and guidelines addressing the pathophysiology and treatment of pain in peritoneal carcinomatosis.

Given that the identified studies exhibited substantial heterogeneity in study design, patient populations, cancer types, and outcome measures, a narrative synthesis was

undertaken to summarise the available evidence. No formal protocol, risk-of-bias assessment, or meta-analysis was conducted.

RESULTS

Mechanisms of pain in peritoneal carcinomatosis

Visceral Pain. The most common type of pain in peritoneal carcinomatosis is visceral pain, typically characterised as dull, diffuse, and poorly localised pain, often referred to remote cutaneous sites. In the setting of peritoneal carcinomatosis, visceral pain has a multifactorial origin, including direct tumour infiltration of visceral organs, malignant ascites, bowel obstruction, and mesenteric or bowel ischaemia, among other contributing mechanisms.

Malignant ascites is a common symptom and, at times, the initial manifestation of peritoneal carcinomatosis. It contributes to the onset of visceral pain through increased intra-abdominal pressure, stretching of the visceral peritoneum, distension and compression of solid organs (particularly their capsules), and reduced mesenteric blood flow. Fluid accumulation leads to increased intraperitoneal pressure, rising from physiological levels of around 5 mmHg to values exceeding 20 mmHg. This results in stretching of the visceral peritoneum and activation of mechanosensitive nociceptors, leading to the transmission of poorly localised, dull pain via C fibres. Concurrent stretching of the parietal peritoneum generates sharper, more localised sensations through A-delta fibres, as discussed in the subsequent section of this paper. Elevated intra-abdominal pressure additionally causes distension of solid organ capsules, such as the liver and spleen, resulting in pain mediated by unmyelinated C fibres. As ascitic fluid preferentially accumulates in subdiaphragmatic regions, it irritates the diaphragmatic peritoneum and may produce referred shoulder pain via the phrenic nerve (C3–C5) [18].

Pain may also be induced by visceral ischaemia caused by mesenteric hypoperfusion due to increased intra-abdominal pressure, direct compression of mesenteric vessels by tumour masses, and tumour-related thrombosis. All of these mechanisms may result in secondary visceral ischaemia and the onset of pain [19].

Peritoneal carcinomatosis may cause either mechanical or functional bowel obstruction, which is another source of visceral pain. Mechanical obstruction is induced by ex-

trinsic compression of the bowel by omental tumour masses and malignant adhesions, or by intramural infiltration narrowing the gastrointestinal tract. This initially results in increased peristaltic activity (clustered contractions), producing acute, colicky pain. Hyperperistalsis triggers the release of prostaglandins, secretagogues, and nociceptive mediators, as well as vasoactive intestinal polypeptide (VIP), which causes hyperaemia, oedema, and luminal fluid accumulation. [20]. Functional bowel obstruction refers to general gastrointestinal dysmotility resulting from mechanisms distinct from those in mechanical obstruction. Direct tumour infiltration of the coeliac plexus, mesentery, or enteric nervous system impairs coordinated peristalsis, while damage to the interstitial cells of Cajal further weakens pacemaker activity and promotes dysmotility [21]. Additional contributors include paraneoplastic autonomic neuropathy and opioid-induced bowel dysfunction. The clinical manifestations of this condition are persistent abdominal pain with associated symptoms, including nausea, vomiting, and constipation.

Direct tumour infiltration of intra-abdominal organs produces visceral pain through distortion of organ capsules and mesenteric attachments, infiltration of the bowel wall, and encasement of pelvic organs and presacral structures [18,22]. Tumour deposits in the mesentery and omentum cause mechanical traction and distortion of these structures, stimulating visceral pain via activation of high-threshold sympathetic C-fibre nociceptors [23]. Pain may also result from infiltration of smooth muscle within structures such as the gastrointestinal tract, bile ducts, and ureters, leading to spasm and clinically presenting as cramping, colicky visceral pain [22].

Somatic Pain. Somatic pain arises due to direct tumour infiltration of the parietal peritoneum, abdominal wall, or musculoskeletal structures. It is usually characterised as sharp, well-localised pain that exacerbates with movement or palpation.

The parietal peritoneum is innervated by densely distributed, fast-conducting myelinated A-delta fibres that travel with segmental somatic nerves. Thus, direct infiltration of the parietal peritoneum produces localised tenderness and peritoneal signs. As mentioned above, stretching of the peritoneum related to fluid accumulation also stimulates mechanosensitive A-delta nociceptors in the parietal layer, producing somatic pain. A similar mechanism is observed in direct malignant invasion of abdominal wall structures and intra-abdominal soft tissues [7,24,25].

Somatic pain may also be indirectly caused by malignant adhesions between tumour-invaded peritoneal surfaces, generating traction on the abdominal wall and parietal

peritoneum. Adhesions themselves may be a source of pain due to the presence of sensory nerve fibres within them [26].

An important mechanism contributing to somatic pain is tumour-induced inflammation and chemical irritation of the peritoneum. Tumour cells and associated inflammatory infiltrates release algogenic mediators—such as prostaglandins, bradykinin, histamine, and cytokines—which sensitise nociceptors, lower their activation threshold, and induce localised hyperalgesia. This process is further amplified by tumour-related inflammatory peritonitis, in which pro-inflammatory cytokines (e.g., tumour necrosis factor alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6)) modulate nociceptor activity and contribute to heightened pain sensitivity [25,27].

It is important to note that sources of somatic pain also include interventions aimed at symptom relief, such as peritoneal drainage, surgical procedures (including cytoreductive surgery), hyperthermic intraperitoneal chemotherapy (HIPEC), and radiation-induced tissue injury [8,28]. However, the impact of HIPEC itself on the occurrence of postoperative pain has not been clearly established [29,30].

Neuropathic Pain

Neuropathic pain in peritoneal carcinomatosis arises from damage, compression, or invasion of neural structures by tumour. It is sustained by aberrant signalling within dysfunctional peripheral and central nervous system pathways and is typically characterised by burning, shooting, tingling, or electric shock-like sensations [12,13].

Perineural invasion (PNI) is the most direct mechanism of tumour-induced neuropathic pain in peritoneal carcinomatosis. It is defined as the process by which tumour cells invade and interact with nerves within the tumour microenvironment. It is frequently observed in malignancies commonly associated with peritoneal spread, including pancreatic, colorectal, gastric, and ovarian cancers [31]. Tumour dissemination occurs along anatomical neural pathways, including the coeliac plexus in upper abdominal malignancies and the inferior hypogastric and lumbosacral plexuses (via the splanchnic nerves) in pelvic malignancies [32].

At the molecular level, PNI is mediated by complex tumour–nerve interactions involving the secretion of neurotrophins—such as nerve growth factor (NGF), brain-derived neurotrophic factor (BDNF), and glial cell line-derived neurotrophic factor (GDNF)—which activate sensory neurons, alongside neuroinflammatory processes characterised by macrophage infiltration and increased expression of pro-inflammatory cytokines, including IL-1β and TNF-α. These changes are accompanied by activation of in-

tracellular signalling pathways, including cyclic adenosine monophosphate (cAMP), cAMP response element-binding protein (CREB), and extracellular signal-regulated kinases 1 and 2 (ERK1/2), leading to progressive neuronal hyperexcitability and amplification of pain signalling [9,33,34].

Direct invasion of the lumbar plexus may contribute to lumbosacral plexopathy, with the initial manifestation being severe, unrelenting, and sometimes burning pain located in the lumbar region, hip, and buttocks, with pseudoradicular radiation to the lower extremity [35]. Peripheral nerves within the abdominal wall or pelvis may be compressed by bulky peritoneal disease and malignant ascites, producing focal neuropathic pain syndromes. In advanced disease, nerve roots may become invaded by direct extension of tumour from the peritoneum to the retroperitoneal space, resulting in radicular neuropathic pain with a dermatomal distribution.

Table 1 summarises the main mechanisms and clinical characteristics of pain associated with PC.

Table 1. Mechanisms and clinical features of pain in peritoneal carcinomatosis

PAIN MECHANISM OR CLINICAL CONTRIBUTOR	MECHANISM	CLINICAL FEATURES
Visceral	C-fiber activation in visceral organs; referred pain	Diffuse, dull, poorly localized; May be referred to remote cutaneous sites
Somatic	A-delta fiber activation in parietal peritoneum/abdominal wall	Well-localized, sharp, movement-related
Neuropathic	Nerve infiltration/compression, deafferentation	Burning, shooting, tingling
Ascites-related distension	Increased intra-abdominal pressure, stretching of the visceral peritoneum, distention and compression of solid organs	Diffuse pressure, abdominal tightness
Malignant bowel obstruction	Increased peristaltic activity (clustered contractions), hyperemia, edema, luminal fluid accumulation; release of prostaglandins, secretagogues, vasoactive intestinal polypeptide (VIP);	Colicky, intermittent; nausea/vomiting
Inflammatory pain	Tumour associated inflammation and inflammatory mediator release	Persistent abdominal pain, tenderness, worsening with disease progression

Management of pain in peritoneal carcinomatosis

Management of pain in PC requires identification of the underlying causes. Typically mechanisms of pain overlap, and all of them need to be addresses with appropriate treatment.

The treatment of pain associated with peritoneal metastases is based on established principles of cancer pain control, adapted to the individual patient's clinical presenta-

tion. Methods include pharmacological agents as well as interventional procedures aimed at directly reducing pain or addressing its underlying cause.

Pharmacological treatment. Pharmacological management constitutes the foundation of pain treatment. Opioids remain the primary approach, as pain in PC is typically moderate to severe. The World Health Organization (WHO) step 2 of the analgesic ladder (weak opioids) may be bypassed in favour of low-dose strong opioids [14]. Such an approach is effective in visceral pain, which, as previously noted, is the predominant type of pain in peritoneal carcinomatosis. The choice of a specific opioid should be individualised and based on patient factors, prior exposure, comorbidities, and the adverse effect profile. A single experimental study in mice has suggested that pain associated with peritoneal carcinomatosis may be relatively opioid-resistant, potentially requiring higher doses to achieve adequate analgesia. This effect was attributed to downregulation of μ-opioid receptors in substance P–positive dorsal root ganglion neurons [36]. However, there is a lack of corresponding studies in humans to confirm these findings.

Paracetamol, as non-opioid analgesic, may serve as useful adjunct, though evidence for additive benefit with opioids is equivocal [37].

Another commonly used non-opioid analgesics are non-steroidal anti-inflammatory drugs (NSAIDs). They are particularly effective in somatic component of pain, as its aetiology is associated with inflammatory process [37]. The use of NSAIDs as adjunctive agents has been reported to reduce opioid requirements and improve pain scores [38]. It is essential to monitor for adverse effects, as many patients with peritoneal carcinomatosis are at increased risk of renal, gastrointestinal, and cardiovascular toxicities, as well as thrombocytopenia and bleeding disorders [15].

In the presence of a significant somatic pain component related to tumour infiltration of the abdominal wall, parietal peritoneum, musculoskeletal structures, and connective tissues, and where pharmacological management proves inadequate, interventional strategies should be considered (as discussed later).

The neuropathic component of pain requires the use of adjuvant analgesics, including tricyclic antidepressants (e.g., nortriptyline, desipramine), serotonin–noradrenaline reuptake inhibitors (e.g., duloxetine, venlafaxine), and gabapentinoids (e.g., gabapentin, pregabalin) [11]. Initiation of these medications should be preceded by evaluation of potential drug interactions and the risk of adverse effects, such as anticholinergic ef-

fects associated with tricyclic antidepressants. Another recommended adjuvant analgesic is topical lidocaine in the form of patches [15].

Corticosteroids, most commonly dexamethasone, may be effective adjuvant agents by reducing peritumoral oedema, alleviating inflammatory and neuropathic pain, and contributing to symptom control in malignant bowel obstruction (MBO) [38].

In cases of refractory pain, intravenous lidocaine infusions may be considered, although data remain limited [15].

The pharmacological treatment described above should always be individualised and regularly reassessed to optimise analgesia while minimising toxicity.

Interventional treatment. As pain in PC is predominantly visceral, interventional treatment is guided by the anatomical location of disease and the specific pain generators involved. Interventional techniques include sympathetic plexus neurolysis (coeliac plexus, superior hypogastric plexus, ganglion impar), intrathecal drug delivery systems (IDDS), and neuraxial techniques, as well as, in selected cases, neurostimulation or neuroablative procedures. The choice of intervention depends on whether pain is predominantly upper abdominal (coeliac plexus neurolysis), lower abdominal and pelvic (superior hypogastric plexus block), or diffuse (IDDS). Intrathecal drug delivery systems (IDDS) allow the delivery of opioids and local anaesthetics directly to the spinal cord and should be considered for pain not responding to conventional management [16,39].

Somatic pain components can be effectively targeted with segmental thoracic epidural blocks or truncal fascial plane blocks, such as transversus abdominis plane (TAP) blocks and rectus sheath blocks, which inhibit somatic afferents travelling with intercostal nerves [40].

Management of pain in malignant ascites. As described above, malignant ascites contributes to the onset of pain through various mechanisms. Thus, reduction of fluid accumulation may alleviate symptoms. Therapeutic paracentesis remains an effective intervention, offering rapid, albeit temporary, relief of pain, and abdominal distension in the majority of patients. For those requiring repeated procedures, the use of indwelling tunnelled drainage catheters enables regular fluid evacuation in the outpatient or home setting [17,41]. Diuretics efficacy in reduction of malignant ascites is limited and depends on pathophysiological mechanism of fluid accumulation. The efficacy of diuretics in reducing malignant ascites is limited and depends on the underlying pathophysiological mechanism of fluid accumulation. They are most effective in pa-

tients with massive hepatic metastases, portal hypertension as the primary cause of ascites, and a serum–ascites albumin gradient (SAAG) ≥1.1 g/dl [10].

Management of pain in MBO. MBO is a frequent complication of PC and requires a specific pharmacological approach or, in selected cases, surgical or endoscopic interventions. As oral intake is usually not possible due to nausea and vomiting, parenteral and transdermal routes of drug administration are preferred.

Although opioids may contribute to functional bowel obstruction, they remain the foundation of analgesic therapy, alongside adjuvant agents such as octreotide, dexamethasone, anticholinergic drugs, and, in selected cases, metoclopramide. Octreotide reduces gastrointestinal secretions and distension, while dexamethasone may decrease peritumoral oedema and support the resolution of partial obstruction [15,42,43]. Anticholinergic agents (e.g., scopolamine, hyoscyamine) help alleviate colicky pain and reduce secretions [44]. Metoclopramide may be beneficial in partial obstruction but is contraindicated in complete obstruction. Combination therapy with dexamethasone, octreotide, and metoclopramide has shown promising results in improving nausea and pain control [43].

In selected cases, surgical management may also be considered; however, a detailed discussion of invasive techniques in the treatment of malignant bowel obstruction is beyond the scope of this review.

Acute abdominal pain. Patients with peritoneal carcinomatosis are prone to developing acute abdominal emergencies characterised by an abrupt onset of pain and peritoneal signs. Causes include, among others, mechanical bowel obstruction, gastrointestinal perforation, and acute bowel ischaemia associated with venous thrombosis. These conditions present with severe, rapidly worsening pain that is difficult to manage without addressing the underlying cause, often requiring surgical intervention. However, patients with peritoneal metastases are typically at an advanced stage of disease and frequently malnourished, which significantly increases surgical risk. Consequently, management decisions should be individualised, with careful consideration of the patient's overall condition, prognosis, potential benefits of surgical intervention versus risks, and goals of care [5]. While surgery may be indicated in selected patients, in others, a palliative approach focusing on symptom control may be more appropriate [4]. Table 2 summarises therapeutic approaches to pain management in PC.

Table 2. Mechanism based treatment strategies to pain management in peritoneal carcinomatosis

PAIN MECHANISM OR CLINICAL CONTRIBUTOR	TREATMENT STRATEGIES
Visceral	Opioids, NSAIDs, corticosteroids; Celiac or hypogastric plexus neurolysis in selected patients; ITDD
Somatic	NSAIDs, paracetamol, opioids; Regional nerve blocks (TAP blocks, rectus sheath blocks)
Neuropathic	Gabapentinoids (gabapentin, pregabalin), tricyclic antidepressants (TCAs), serotonin-norepinephrine reuptake inhibitors (SNRIs), corticosteroids for nerve compression; Interventional blocks, ITDD
Ascites-related distension	Paracentesis, indwelling catheters, diuretics in selected patients, limited role
Malignant bowel obstruction	Opioids, octreotide, dexamethasone, anticholinergics; nasogastric, endoscopic or surgical decompression in selected patients

DISCUSSION

Pain in PC is a complex and heterogeneous phenomenon arising from multiple overlapping visceral, somatic, and neuropathic mechanisms that frequently coexist and change during the course of disease progression [7,8,11]. Visceral pain appears to be the predominant pain phenotype; however, somatic and neuropathic components often contribute substantially to symptom burden and may require specific therapeutic interventions [8,9].

Given the diversity of mechanisms involved, effective pain management requires careful assessment of the factors contributing to pain in an individual patient [7,12]. Pharmacological treatment remains the cornerstone of management, with opioids constituting the main therapeutic option for moderate-to-severe pain [14,15]. Depending on the clinical presentation, additional benefit may be achieved through the use of non-opioid analgesics, adjuvant agents targeting neuropathic pain, and corticosteroids [11,14]. Interventional procedures may provide further analgesic benefit in selected patients, particularly when symptoms remain inadequately controlled despite optimisation of systemic therapy [16].

Pain associated with specific complications of peritoneal carcinomatosis, including malignant ascites and malignant bowel obstruction, often requires treatment directed at the underlying pathophysiological process in addition to conventional analgesic therapy [4,17]. Symptom relief may therefore depend not only on adequate analgesia but also on interventions aimed at reducing fluid accumulation, relieving bowel obstruction, or managing other disease-related complications [17]. Individualisation of

treatment remains essential and should take into account the predominant clinical manifestations, overall clinical status, prognosis, and patient-defined goals of care [4,14].

Limitations

Despite advances in cancer pain management, evidence specific to patients with peritoneal carcinomatosis remains limited. Many therapeutic recommendations are derived from studies conducted in broader populations of patients with cancer related pain. Further research is needed to improve understanding of pain mechanisms in peritoneal carcinomatosis and to establish evidence based, mechanism oriented treatment strategies for this patient population.

CONCLUSIONS

Pain in peritoneal carcinomatosis is multifactorial and is associated with tumour related and complication related mechanisms, including peritoneal infiltration, inflammation, ascites, bowel obstruction, and a neuropathic component. These mechanisms often coexist in the same patient and may change as the disease progresses.

Pain management in peritoneal carcinomatosis should be based on assessment of the dominant pain mechanism and relevant clinical complications. Pharmacological, interventional, and supportive approaches may be used in combination, particularly when pain is associated with malignant ascites, malignant bowel obstruction, visceral distension, and neuropathic involvement.

The evidence base specifically addressing pain management in peritoneal carcinomatosis remains limited. Further studies are needed to clarify pain mechanisms and to develop mechanism based treatment strategies for patients with peritoneal metastases.

DISCLOSURE

Author Contributions

Conceptualization: Julia Bąk, Agata Świątek, Natalia Turzyńska, Wojciech Frączyk, Mateusz Krysiak. Methodology: Kamil Andruszkiewicz, Bartosz Machnio, Agnieszka Stankowska, Natalia Turzyńska. Data collection: Julia Bąk, Bogna Błachowska, Mateusz Krysiak. Writing, original draft preparation: Julia Bąk, Kamil Andruszkiewicz, Katarzyna Mania. Writing, review and editing: Natalia Jasińska, Agnieszka Stankowska, Agata Świątek, Wojciech Frączyk, Bartosz Machnio, Katarzyna Mania. Supervision: Julia Bąk.

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Conflict of interest

The authors declare that they have no conflicts of interest.

Use of artificial intelligence tools

Artificial intelligence tools were used only to assist with grammar and readability. All authors have read and approved the final version of the manuscript.

REFERENCES

1. Cortés-Guiral D, Hübner M, Alyami M, Bhatt A, Ceelen W, Glehen O, et al. Primary and metastatic peritoneal surface malignancies. Nat Rev Dis Primers. 2021;7(1):91. https://doi.org/10.1038/s41572-021-00326-6
2. Roth L, Russo L, Ulugoel S, dos Santos RF, Breuer E, Gupta A, et al. Peritoneal metastasis: current status and treatment options. Cancers (Basel). 2022;14(1):60. https://doi.org/10.3390/cancers14010060
3. Quénet F, Elias D, Roca L, Goéré D, Ghouti L, Pocard M, et al. Cytoreductive surgery plus hyperthermic intraperitoneal chemotherapy versus cytoreductive surgery alone for colorectal peritoneal metastases (PRODIGE 7): a multicentre, randomised, open-label,

phase 3 trial. Lancet Oncol. 2021;22(2):256-266. https://doi.org/10.1016/S1470-2045(20)30599-4

4. Bleicher J, Lambert LA. A palliative approach to management of peritoneal carcinomatosis and malignant ascites. Surg Oncol Clin N Am. 2021;30(3):475-490. https://doi.org/10.1016/j.soc.2021.02.004
5. Lambert LA, Wiseman J. Palliative management of peritoneal metastases. Ann Surg Oncol. 2018;25(8):2165-2171. https://doi.org/10.1245/s10434-018-6335-7
6. Franko J, Shi Q, Meyers JP, Maughan TS, Adams RA, Seymour MT, et al. Prognosis of patients with peritoneal metastatic colorectal cancer given systemic therapy: an analysis of individual patient data from prospective randomised trials from the ARCAD database. Lancet Oncol. 2016;17(12):1709-1719. https://doi.org/10.1016/S1470-2045(16)30500-9
7. Varrassi G, Paladini A, Van Tran Y, Pham VP, Al Alwany AA, Farì G, et al. Advances in the pathophysiology and management of cancer pain: a scoping review. Cancers (Basel). 2026;18(2):259. https://doi.org/10.3390/cancers18020259
8. Portenoy RK, Ahmed E. Cancer pain syndromes. Hematol Oncol Clin North Am. 2018;32(3):371-386. https://doi.org/10.1016/j.hoc.2018.01.002
9. Shi RJ, Ke BW, Tang YL, Liang XH. Perineural invasion: a potential driver of cancer-induced pain. Biochem Pharmacol. 2023;215:115692. https://doi.org/10.1016/j.bcp.2023.115692
10. Sangisetty SL, Miner TJ. Malignant ascites: a review of prognostic factors, pathophysiology and therapeutic measures. World J Gastrointest Surg. 2012;4(4):87-95. https://doi.org/10.4240/wjgs.v4.i4.87
11. Finnerup NB, Kuner R, Jensen TS. Neuropathic pain: from mechanisms to treatment. Physiol Rev. 2021;101(1):259-301. https://doi.org/10.1152/physrev.00045.2019
12. Portenoy RK. Treatment of cancer pain. Lancet. 2011;377(9784):2236-2247. https://doi.org/10.1016/S0140-6736(11)60236-5
13. Paice JA, Ferrell B. The management of cancer pain. CA Cancer J Clin. 2011;61(3):157-182. https://doi.org/10.3322/caac.20112
14. Paice JA, Bohlke K, Barton D, Craig DS, El-Jawahri A, Hershman DL, et al. Use of opioids for adults with pain from cancer or cancer treatment: ASCO guideline. J Clin Oncol. 2023;41(4):914-930. https://doi.org/10.1200/JCO.22.02198
15. National Comprehensive Cancer Network. NCCN Clinical Practice Guidelines in Oncology: Adult Cancer Pain. Version 1.2026 [Internet]. Plymouth Meeting: National Comprehensive Cancer Network; 2026 [cited 2026 May 9]. Available from: https://www.nccn.org/guidelines/guidelines-detail?category=3&id=1413

16. Nagar SD, Nagar SJ, Jordan V, Dawson J. Sympathetic nerve blocks for persistent pain in adults with inoperable abdominopelvic cancer. Cochrane Database Syst Rev. 2024;2024(6):CD015229. https://doi.org/10.1002/14651858.CD015229.pub2

17. Raptopoulos V, Gourtsoyiannis N. Peritoneal carcinomatosis. Eur Radiol. 2001;11(11):2195-2206. https://doi.org/10.1007/s003300100998

18. Nigri G, Tsoulfas G, editors. Gastrointestinal Surgical Emergencies. Chicago: American College of Surgeons; 2021.

19. Cousins SE, Tempest E, Feuer DJ. Surgery for resolving symptoms associated with malignant bowel obstruction in advanced gynaecological and gastrointestinal cancers. Cochrane Database Syst Rev. 2016;2016(1):CD002764. https://doi.org/10.1002/14651858.CD002764.pub2

20. Guo T, Li J, Li J, Kong D, Bi C, He Z, et al. Association between hyperpolarization-activated channel in interstitial cells of Cajal and gastrointestinal dysmotility induced by malignant ascites. Oncol Lett. 2017;13(3):1601-1608. https://doi.org/10.3892/ol.2017.5652

21. Rigor BM. Pelvic cancer pain. J Surg Oncol. 2000;75(4):280-300. https://doi.org/10.1002/1096-9098(200012)75:4%3C280::AID-JSO8%3E3.0.CO;2-E

22. Holzer-Petsche U, Brodacz B. Traction on the mesentery as a model of visceral nociception. Pain. 1999;80(1-2):319-328. https://doi.org/10.1016/S0304-3959(98)00233-4

23. Bohren Y, Cachemaille M, Timbolschi ID, Perruchoud C. Understanding the physiopathology of pain pathways for a practical approach of cancer pain management. Cardiovasc Intervent Radiol. 2025;48(7):899-906. https://doi.org/10.1007/s00270-024-03920-9

24. Reyes-Gibby CC, Wu X, Spitz M, Kurzrock R, Fisch M, Bruera E, et al. Molecular epidemiology, cancer-related symptoms, and cytokines pathway. Lancet Oncol. 2008;9(8):777-785. https://doi.org/10.1016/S1470-2045(08)70197-9

25. Sulaiman H, Gabella G, Davis C, Mutsaers SE, Boulos P, Laurent GJ, et al. Presence and distribution of sensory nerve fibers in human peritoneal adhesions. Ann Surg. 2001;234(2):256-261. https://doi.org/10.1097/00000658-200108000-00016

26. Sodek KL, Murphy KJ, Brown TJ, Ringuette MJ. Cell-cell and cell-matrix dynamics in intraperitoneal cancer metastasis. Cancer Metastasis Rev. 2012;31(1-2):397-414. https://doi.org/10.1007/s10555-012-9351-2

27. Wang X, Li T. Postoperative pain pathophysiology and treatment strategies after CRS + HIPEC for peritoneal cancer. World J Surg Oncol. 2020;18(1):79. https://doi.org/10.1186/s12957-020-01842-7

28. Kim JH, Park KN, Park EY, Jang MJ, Park YJ, Kim Y, et al. Impact of warm saline irrigation, hyperthermic intraperitoneal chemotherapy on postoperative pain in primary ovarian cancer from the KOV-HIPEC-01 randomized trial. Gynecol Oncol. 2023;177:32-37. https://doi.org/10.1016/j.ygyno.2023.08.006

29. van Driel WJ, Koole SN, Sikorska K, Schagen van Leeuwen JH, Schreuder HWR, Hermans RHM, et al. Hyperthermic intraperitoneal chemotherapy in ovarian cancer. N Engl J Med. 2018;378(3):230-240. https://doi.org/10.1056/NEJMoa1708618

30. Wang M, Pu N, Bo X, Chen F, Zhou Y, Cheng Q. Significance and mechanisms of perineural invasion in malignant tumors. Front Oncol. 2025;15:1572396. https://doi.org/10.3389/fonc.2025.1572396

31. Capek S, Howe BM, Amrami KK, Spinner RJ. Perineural spread of pelvic malignancies to the lumbosacral plexus and beyond: clinical and imaging patterns. Neurosurg Focus. 2015;39(3):E14. https://doi.org/10.3171/2015.7.FOCUS15209

32. Mousa SA, Shaqura M, Mohamed D, Hong X, Krug SM, Metwally EY, et al. Tumor-macrophage-nerve interactions drive neuroinflammation and neuropathic pain in prostate cancer perineural invasion. Brain Behav Immun. 2026;133:106280. https://doi.org/10.1016/j.bbi.2026.106280

33. Shi DD, Guo JA, Hoffman HI, Su J, Mino-Kenudson M, Barth JL, et al. Therapeutic avenues for cancer neuroscience: translational frontiers and clinical opportunities. Lancet Oncol. 2022;23(2):e62-e74. https://doi.org/10.1016/S1470-2045(21)00596-9

34. Antoine JC, Camdessanché JP. Peripheral nervous system involvement in patients with cancer. Lancet Neurol. 2007;6(1):75-86. https://doi.org/10.1016/S1474-4422(06)70679-2

35. Suzuki M, Narita M, Hasegawa M, Furuta S, Kawamata T, Ashikawa M, et al. Sensation of abdominal pain induced by peritoneal carcinomatosis is accompanied by changes in the expression of substance P and μ-opioid receptors in the spinal cord of mice. Anesthesiology. 2012;117(4):847-856. https://doi.org/10.1097/ALN.0b013e31826a4ac8

36. Vardy J, Agar M. Nonopioid drugs in the treatment of cancer pain. J Clin Oncol. 2014;32(16):1677-1690. https://doi.org/10.1200/JCO.2013.52.8356

37. Zhang X, Dang DS, Sun X, Kang Y. The role of combined adjuvant therapeutic drugs in modulating opioid dosage and pain status in cancer patients: a systematic review and meta-analysis. Support Care Cancer. 2025;33(11). https://doi.org/10.1007/s00520-025-09998-2

38. Mercadante S, Klepstad P, Kurita GP, Sjogren P, Giarratano A. Sympathetic blocks for visceral cancer pain management: a systematic review and EAPC recommendations. Crit Rev Oncol Hematol. 2015;96(3):577-583. https://doi.org/10.1016/j.critrevonc.2015.07.014

39. Boezaart AP, Smith CR, Chembrovich S, Zasimovich Y, Server A, Morgan G, et al. Visceral versus somatic pain: an educational review of anatomy and clinical implications. Reg Anesth Pain Med. 2021;46(7):629-637. https://doi.org/10.1136/rapm-2020-102084

40. Poisson C, Sampetrean A, Renard P, Khoury-Abboud RM, Scotté F, Vigouret-Viant L, et al. Palliative semi-permanent abdominal drain for the management of refractory malignant ascites: a retrospective study in a comprehensive cancer center. Support Care Cancer. 2025;33:496. https://doi.org/10.1007/s00520-025-09551-1

41. Vierra MA, Morgan RB, Bhutiani N, White MG, Eng OS. Contemporary management of malignant ascites. J Surg Res. 2025;307:157-175. https://doi.org/10.1016/j.jss.2025.01.025

42. Jones APM, McGauran MFG, Jagasia N, Hiscock RJ, Hyde S, Grant P. Efficacy of dexamethasone in the management of malignant small bowel obstruction in advanced epithelial ovarian cancer. Support Care Cancer. 2022;30(3):2821-2827. https://doi.org/10.1007/s00520-021-06694-9

43. Walter M, Hansen E, Hamid S, Carozza D, Mann G, Roche C, et al. Palliative management of inoperable malignant bowel obstruction: prospective, open label, phase 2 study at an NCI comprehensive cancer center. J Pain Symptom Manage. 2024;67(1):20-26. https://doi.org/10.1016/j.jpainsymman.2023.09.014

44. O'Connor B, Creedon B. Pharmacological treatment of bowel obstruction in cancer patients. Expert Opin Pharmacother. 2011;12(14):2205-2214. https://doi.org/10.1517/14656566.2011.597382

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