NARRATIVE REVIEW

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Aetiology of pelvic neuropathies in women: a narrative review

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ABSTRACT

Pelvic neuropathy is an under-recognised contributor to chronic pelvic pain in women, yet a structured overview of its identifiable causes has been lacking. In this narrative review we searched PubMed, Embase and Web of Science from inception to 1 March 2024 for clinical studies describing pelvic neuropathy in women with a defined or inferable aetiology, and synthesised them thematically. We included 306 studies (215 case reports, 53 case series, 24 retrospective and 14 prospective cohort studies) describing 2,413 women. Five recurring aetiological categories emerged: iatrogenic injury (the most frequent), nerve invasion or traction by disease processes (notably endometriosis and tumours), pregnancy- and childbirth-related injury, external trauma, and compression by muscular, vascular, benign or malignant structures. Aetiologies of particular relevance to gynaecological practice included nerve-invasive endometriosis, which characteristically produces cyclical (catamenial) symptoms, and obstetric neuropathies, which are usually self-limiting. Specific neuropathic syndromes—piriformis, May–Thurner, pelvic congestion and Alcock's canal (pudendal) syndrome—were inconsistently defined across studies. Diagnostic strategies and reporting quality varied widely, neuropathy was frequently recognised late, and the evidence base was dominated by case reports and small series; the overall certainty of evidence is therefore low and no randomised controlled trials were identified. Greater awareness of nerve-specific injury mechanisms—particularly endometriosis-related and obstetric neuropathies—together with standardised diagnostic pathways, is needed to improve recognition and outcomes for women with neuropathic pelvic pain.

Keywords: Endometriosis, iatrogenic disease, neuralgia, peripheral nervous system diseases, pelvis

Introduction

Chronic pelvic pain (CPP) affects an estimated 7-24% of women globally and represents a significant clinical and economic burden on healthcare systems and patients alike.[1] It is defined as persistent or recurrent pain in pelvic structures that lasts more than 6 months and often has no clearly identifiable cause.[1,2] In many cases, CPP is multifactorial, involving gynaecological, urological, gastrointestinal, musculoskeletal, neurological, and psychological components.[3] Effective treatment typically requires a multidisciplinary approach, including physical therapy, pharmacological management, and psychological support.[4,5]

Pain perceived in the pelvis can arise through distinct mechanisms, and distinguishing between them is clinically important. Nociceptive pain results from actual or threatened damage to non-neural tissue

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with an intact somatosensory system; neuropathic pain is caused by a lesion or disease of the somatosensory nervous system itself; and nociplastic pain arises from altered nociceptive processing without clear evidence of tissue or nerve damage, as seen in central sensitisation states. These mechanisms frequently coexist in CPP, but only neuropathic pain reflects a definable structural or functional nerve lesion that may be amenable to nerve-targeted treatment. The present review focuses specifically on pelvic neuropathies—that is, the neuropathic component of pelvic pain for which an identifiable aetiology can be determined.

Within this complex framework, pelvic neuropathies are a potentially underdiagnosed and undertreated cause of CPP. Neuropathic pelvic pain is characterised by moderate to severe symptoms such as burning, tingling, paraesthesia, muscle weakness, or organ dysfunction.[6] It is caused by damage or disease affecting the somatosensory nervous system.[7] These neuropathies may arise from various mechanisms, including iatrogenic injury, compression, inflammation, invasion by endometriosis or tumours, or obstetric trauma. Although neuropathic mechanisms are increasingly recognised in pelvic pain syndromes, their specific aetiologies remain poorly synthesised, leading to suboptimal diagnostic accuracy and symptom-focused rather than cause-targeted treatment.

This narrative review aims to address this gap by mapping the existing literature on the causes of pelvic neuropathies in women. We do not aim to evaluate treatment effectiveness or diagnostic accuracy, but rather to systematically explore and categorise the reported aetiologies of pelvic neuropathies, thereby offering clinicians a comprehensive overview of possible causes to consider in their diagnostic reasoning.

Understanding these aetiologies is crucial, as accurate diagnosis may enable nerve-targeted treatment strategies, such as surgical decompression, pharmacological neuromodulation, or physical therapy. Even when the aetiology is known, however, multimodal pain management remains essential, given the neuroplastic and psychosocial dimensions of chronic pain. Furthermore, these findings may inform preventive strategies, especially for iatrogenic neuropathies—by far the most frequently reported group in the literature.

To support clinical decision-making, this narrative review provides an overview of all currently described causes of pelvic neuropathies in women, categorising them into meaningful subtypes, and identifies knowledge gaps. Because CPP is a core part of gynaecological practice, we give particular attention throughout to the aetiologies most relevant to gynaecologists, including nerve-invasive endometriosis, neuropathies related to pregnancy and delivery, and pudendal (Alcock's canal) entrapment. In doing so, we aim to contribute to better diagnostic practices, targeted treatments, and improved outcomes for women with neuropathic pelvic pain.

Methods

This study was conducted as a structured narrative review to map the breadth and diversity of reported aetiologies of pelvic neuropathies in women. A narrative review approach was chosen because the topic encompasses heterogeneous study designs, rare conditions, and predominantly descriptive evidence, for which quantitative synthesis or formal effectiveness assessment is neither feasible nor appropriate. To enhance transparency and reproducibility, an explicit and systematic literature search and predefined eligibility criteria were nevertheless applied.

Definition of Pelvic Neuropathy and Its Application in Study Selection

For the purposes of this review, pelvic neuropathy was defined as a condition involving structural or functional impairment of a specific pelvic nerve or nerve plexus, accompanied by symptoms consistent with neuropathic pain or dysfunction. This definition was aligned with the International Association for the Study of Pain definition of neuropathic pain and with descriptions commonly used in the neuropelveology literature.[6-11]

During study selection, we required that pelvic neuropathy be explicitly diagnosed or confirmed in the original publication. Confirmation could be based on clinical evaluation with a nerve-specific symptom distribution, imaging findings suggestive of nerve involvement, neurophysiological testing such as electromyography (EMG) or nerve conduction studies (NCS), intraoperative findings, or the use of established diagnostic criteria where applicable (e.g., the Nantes criteria for pudendal neuralgia). Studies describing pelvic pain without neuropathic features, or in which neuropathy was only hypothesised without diagnostic confirmation, were excluded. This approach was chosen to ensure conceptual clarity and specificity. However, it also introduces essential limitations. Pelvic neuropathies are frequently underdiagnosed in clinical practice, and diagnostic confirmation is often incomplete or absent

in the literature. By requiring explicit confirmation, this review likely underrepresents cases in which neuropathy was present but not recognised or formally documented.

Eligibility Criteria

Eligibility criteria were structured using the Population–Concept–Context framework. We included peer-reviewed case reports, case series, and observational cohort studies that described patients with a diagnosed pelvic neuropathy and an identifiable underlying cause. Studies were eligible regardless of publication year or language.

We excluded reviews, editorials, and opinion articles, as well as studies reporting idiopathic pelvic neuropathies without an identifiable aetiology. Studies describing pelvic conditions (such as endometriosis or fibroids) without explicit confirmation of nerve involvement were also excluded. This strategy was chosen to enable a focused exploration of reported causes rather than symptom-based associations.

Information Sources and Search Strategy

A comprehensive literature search was performed on 1 March 2024 in PubMed, Embase, and Web of Science (Core Collection). The search was designed and carried out in collaboration with a medical information specialist (R.d.V.), who developed the database-specific syntax and ran the searches. The search strategy combined MeSH and free-text terms related to pelvic neuropathy, pelvic nerves, neuralgia, nerve compression, and neuropelveology. The complete search strategy is provided in Appendix A. Reference lists of included studies were screened to identify additional relevant publications.

Study Selection

Two authors (RLE, ED) independently screened titles and abstracts, followed by full-text assessment of potentially eligible articles. Disagreements were resolved through discussion until consensus was reached. This dual-reviewer approach was used to reduce selection bias and enhance reproducibility.

Data Extraction

Data were extracted using a standardised form, capturing study characteristics, patient demographics, diagnostic methods, type of neuropathy, reported aetiology, and treatment where described. Extraction focused on how the authors of the original studies defined and substantiated the cause of neuropathy.

Quality of the Evidence

Given that the included literature consisted predominantly of case reports and small case series, a formal risk-of-bias or certainty-of-evidence assessment was not performed, in keeping with the descriptive aim of a narrative review. Instead, study design was used as a pragmatic indicator of evidential strength. Case reports and small series provide low-certainty evidence: they lack control groups, are prone to selection and reporting bias, and cannot support reliable estimates of incidence or causal attribution. The cohort studies, although larger, were generally retrospective and heterogeneous in their diagnostic criteria. The overall certainty of the evidence base is therefore low, and the frequencies reported below should be interpreted as descriptive rather than epidemiological.

Data Synthesis

We employed an inductive, thematic synthesis approach to analyse the extracted data. Reported aetiologies and mechanisms of pelvic neuropathy were descriptively coded and compared across studies. Codes were then grouped into broader thematic categories through iterative discussion and refinement, based on similarities in anatomical involvement, clinical context, and pathophysiology. This data-driven process allowed us to organise diverse cases into coherent categories that reflect shared underlying mechanisms and facilitate meaningful clinical interpretation.

Results

The search was performed in PubMed, Embase, and Web of Science on 1 March 2024 and yielded 7,234 references. After the title-abstract screening, 344 articles were evaluated for eligibility; 306 met our inclusion criteria (selection is shown in Figure 1).

Characteristics of Included Studies

The included studies were published between 1964 and 2024 and originated from a wide range of clinical specialities, including gynaecology, urology, neurology, general surgery, orthopaedic surgery, radiology, and pain medicine. The great majority of studies appeared from 2000 onward (Figure 2), coinciding with the wider availability of cross-sectional imaging and magnetic resonance neurography, whereas only a small minority (approximately 7%) predated 1990; this temporal skew should be borne in mind when interpreting the apparent rarity of conditions described mainly in older

literature. Study designs included case reports (n=215), case series (n=53), retrospective cohort studies (n=24), and prospective cohort studies (n=14). No randomised controlled trials were identified. Sample sizes ranged from single-patient case reports to prospective cohorts with up to 200 participants. The studies covered a wide age range, from infancy to postmenopausal age. Diagnostic confirmation of pelvic neuropathy was established through various modalities, including clinical examination, imaging [magnetic resonance (MRI), computed tomography (CT), ultrasound], neurophysiological testing (e.g., EMG), intraoperative visualisation, and, in selected cases, formal diagnostic criteria such as the Nantes criteria for pudendal neuralgia. A complete overview of the study characteristics is presented in Table 1. Most studies were conducted in the obstetrics and gynaecology department (24%). However, studies were also conducted in orthopaedic surgery (15%), neurology (10%), and rehabilitation medicine (10%) (according to the department of the first author). All departments are displayed in Figure 3. Most of the included studies were conducted in the United States of America (n=97, 32%). Twenty-three studies (8%) were performed in Turkey, 5% in South Korea, France, Italy, and Japan, and 4% in Canada, Taiwan, Spain, and the United Kingdom.

Aetiologies

Following the inductive, thematic synthesis approach, we arranged all the included studies by aetiology. We have described five main categories: iatrogenic, nerve invasion (e.g., by endometriosis or infection), pregnancy and birth, trauma, and compression. In addition, we added subcategories as displayed in Table 2 and visualised in Figure 4. The included 306 studies described 2,413 patients in total. Some studies described patients from more than one (sub)category, so the total number of studies per subcategory exceeds 306. The number of studies and patients per category and subcategory is shown in Table 3.

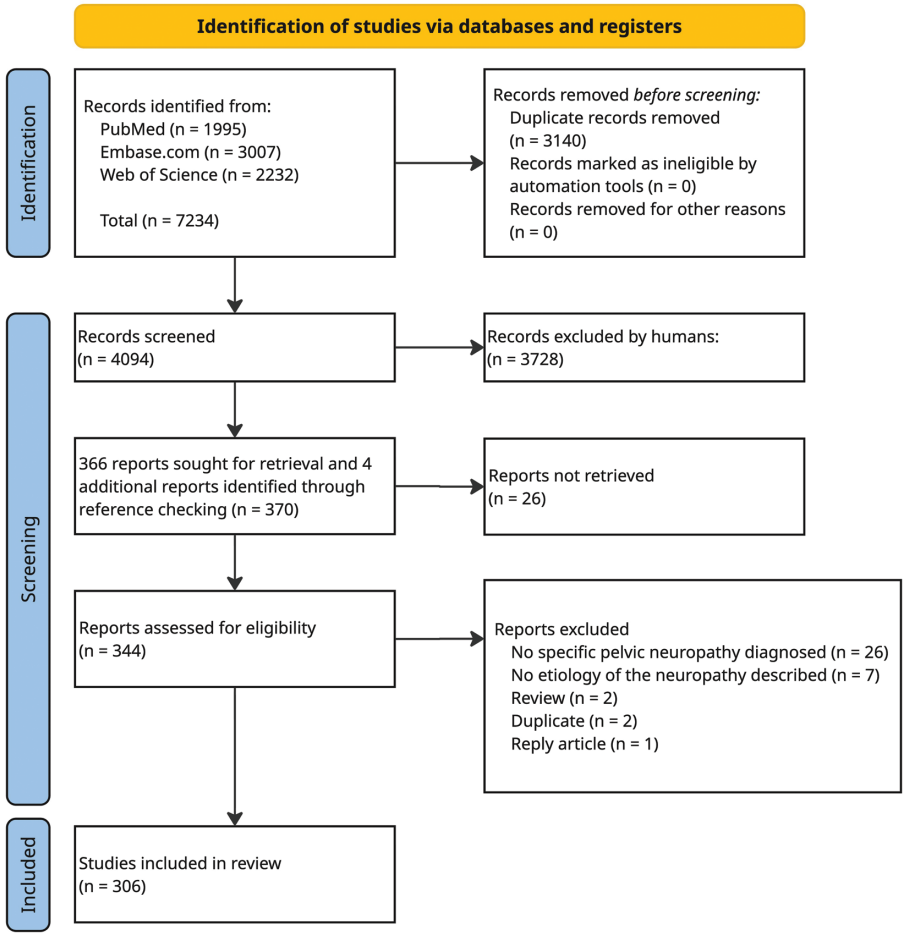

Figure 1. PRISMA flowchart.

Iatrogenic Causes

Most of the neuropathies were seen after surgery in the lower abdomen or pelvis and were mainly labelled as "not specified surgical complications" (n=955 patients, 40%). Only 11 of these 71 studies performed MRI and/or CT; however, most did perform an EMG and/or NCS to diagnose the neuropathy. Another 30 studies (n=85 patients, 4%) specifically indicate the aetiology of post-surgical neuropathy as compression. The compression was caused by the positioning of the patient or sandbag, oedema, haematoma, aneurysm or entrapment of the nerve by sutures.[3,7-9,12-14] Also, mesh or urethral sling for urinary incontinence or vaginal prolapse, an acetabular reconstruction ring, wear debris, granulomatous tissue, and intrapelvic cement or screws after total hip replacement were indicated as post-surgical compression aetiologies for pelvic neuropathies.[15-22] As seen in Appendix B, most neuropathies were reported after gynaecologic surgery, and femoral, lateral femoral cutaneous, obturator, ilioinguinal, iliohypogastric, genitofemoral, lumbosacral root, sciatic and pudendal neuropathies are described. After gynaecologic surgery, multiple cases of post-surgery pelvic neuropathies are also described for orthopaedic, urologic and general surgery. Only one study described a femoral neuropathy after abdominoplasty, a plastic surgery procedure.[23] Five studies reported a transection of the obturator nerve during gynaecologic surgery, which was intraoperatively recognised and repaired, of which only one patient had transient symptoms post-operatively.[24] Next to surgery, other descriptions of iatrogenic causes of pelvic neuropathies are radiation therapy, radiofrequency ablation, iliac bone graft and bone marrow harvesting, manoeuvre by an osteopath, local nerve block and a superior gluteal artery pseudoaneurysm after transvaginal follicle aspiration or needle biopsy.[25-32] The time between surgery, treatment or procedure and presentation was very divergent, as symptoms could start directly after intervention or had a long interval. Interval periods were described up to nine years after total hip arthroplasty,[33] 36 years after radiation therapy[34] or even 40 years after iliac bone grafting.[35]

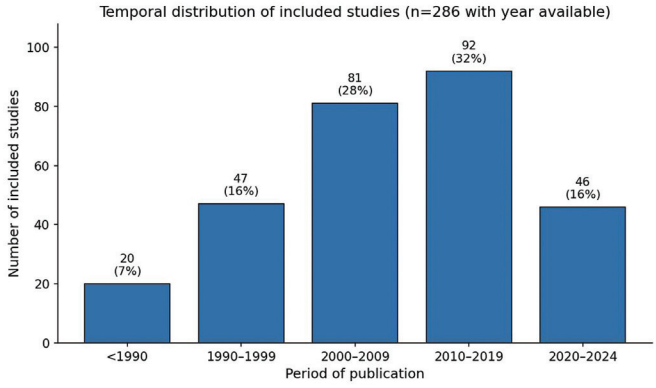

Figure 2. Temporal distribution of the included studies by period of publication.

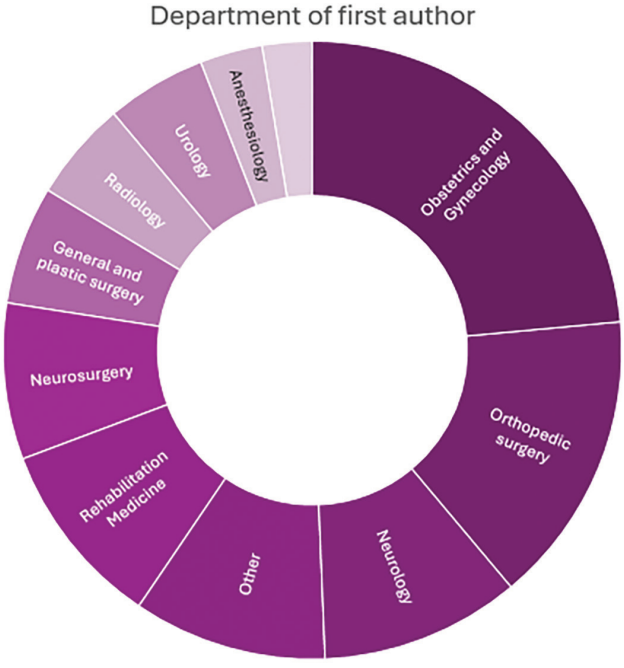

Figure 3. Diagram of the department of the included studies.

Nerve Invasion and Traction

The second largest group of patients demonstrated pelvic neuropathy due to nerve-invasive endometriosis (n=557 patients, 23%). This invasion and/or traction is mainly seen in the sciatic nerve, as sciatic neuropathy after invasion of endometriosis is described in 18 studies (18/23 studies). Femoral, lateral femoral cutaneous, pudendal, obturator and lumbosacral root neuropathies were also described due to endometriosis, as seen in Appendix B. Endometriotic infiltration most often involves the sacral plexus and the sciatic nerve, and the hallmark clinical clue is catamenial (cyclical) symptomatology, with pain, paraesthesia or motor deficit that intensifies in the perimenstrual period.[36] Symptoms may nonetheless become continuous once perineural fibrosis and axonal damage are established, and Possover[37] demonstrated progressive nerve destruction within two years in untreated cases. The diagnosis is frequently delayed because symptoms are attributed to lumbar disc disease; magnetic resonance neurography and laparoscopic

Table 1. Inclusion and exclusion criteria of the search.

	Inclusion criteria	Exclusion criteria
Population	• A located pelvic neuropathy that can occur in women	• Women with endometriosis, adhesions, MESH complications, nervous tumors or venous congestion syndromes without a pelvic neuropathy
Outcomes	• Etiologies for pelvic neuropathy	• Idiopathic neuropathy
Study design	• Randomized controlled trial • Case control • Cohort study • Case reports	• Reviews • Meta-analysis • Reply, editorials or opinion articles

Table 2. Aetiological categories of pelvic neuropathy, by mechanism and condition.

Mechanism	Condition
Iatrogenic	Surgical: Surgical complication, compression, post-repair Non-surgical
Nerve invasion	Neuritis Endometriosis Invasive nerve tumours
Pregnancy and birth	Pregnancy Birth Caesarean section
Trauma	Acute Chronic Haematoma e.g., due to clotting disorders
Compression	Muscular: Hypertrophy/nerve variation, rhabdomyolysis Vascular: Aneurysm; venous compression syndrome Tumour: Benign cysts; fibrosis; malignant tumours

exploration of the pelvic sidewall and sacral plexus are the most informative investigations. Laparoscopic neurolysis with excision of perineural endometriosis can relieve symptoms and, if performed before irreversible axonal loss, may allow neurological recovery—underscoring the value of early recognition by gynaecologists. Pelvic neuropathy caused by invasion of the nerve by a tumour is rare and only reported in case reports and small case series. The benign schwannoma is most documented and most often described in the sciatic nerve.[38] Other benign nerve tumours are intraneural perineuriomas, neurofibromas and lipomatosis of the nerves.[39-41] In addition, four case reports present a sciatic neuropathy by the invasion of a malignant tumour, namely malignant nerve sheath tumours, neurolymphomatosis, and extramedullary anaplastic ependymoma,[42-45] and one

Table 3. Categories of pelvic neuropathy aetiologies.

Etiology category-number of included patients	Subcategory (number of studies)-number of included patients
Iatrogenic-1094 patients (45%)	Surgery: Surgical complication (71)-955
	Surgery: Compression (30)-85
	Surgery: Post-repair (5)-5 Non-surgical (20)-49
Nerve invasion and traction-614 patients (25%)	Endometriosis (23)-557
	Neuritis (1)-1
	Nerve tumors (21)-56
Pregnancy and birth-93 patients (4%)	Pregnancy (4)-6
	Birth (12)-77
	Caesarean section (4)-10
Trauma-202 patients (8%)	Acute (16)-158
	Chronic (13)-28
	Hematoma (6)-9
	Clotting disorder (7)-7
Compression-409 patients (17%)	Muscular (18)-41
	Vascular (16)-220
	Tumor benign: Cysts (23)-29 Tumor benign: Fibrosis (1)-1
	Tumor benign: Other (40)-58
	Malignant tumors (17)-60

study reported a lumbosacral root neuropathy caused by malignant pelvic neuroblastoma.[46] Another rare presentation of nerve invasion is a case report by Lazaro and Butt[47] describing femoral neuropathy/neuritis caused by Lyme disease.

Pregnancy and Birth

Pelvic neuropathy after pregnancy or childbirth entails the smallest proportion of patients of all five main categories (n=95 patients, 4%). Sciatic, femoral, and lumbosacral

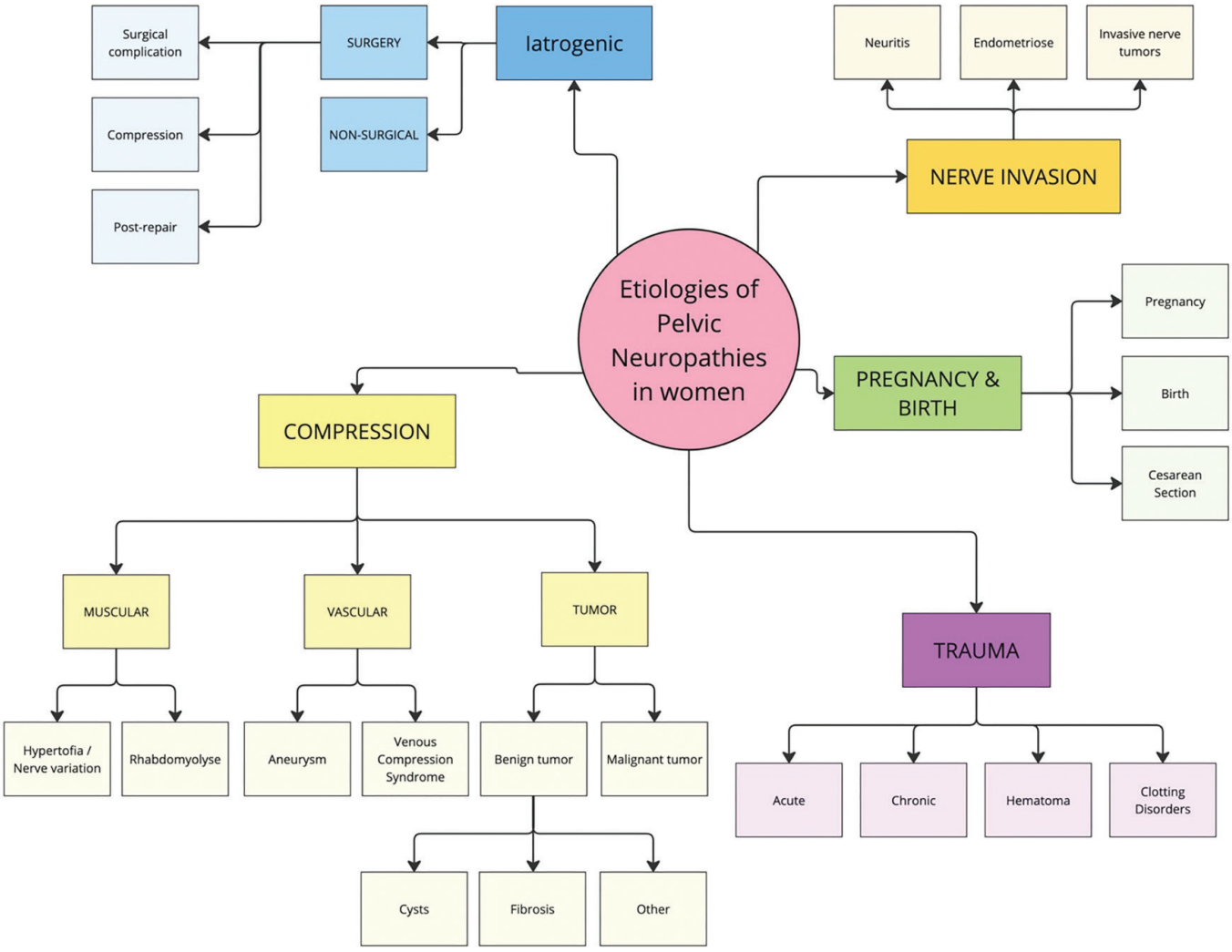

Figure 4. Aetiological categories of pelvic neuropathy.

root neuropathies were described during normal pregnancies of 30-34 weeks of gestation. Additionally, an obturator neuropathy was reported after an ectopic pregnancy.[48] Femoral and obturator neuropathy have been described both after uncomplicated vaginal birth and after prolonged vaginal birth.[49,50] As seen in Appendix B, pudendal and cluneal neuropathy were also reported after uncomplicated birth, and lumbosacral root neuropathies were seen after prolonged vaginal births. One case report describes an obturator neuropathy after forceps delivery.[51] Moreover, four studies report on cases of pelvic neuropathies after caesarean section.[52-55] Another rare observation after birth with massive postpartum haemorrhage is reported by Rohilla et al.,[56] describing lumbosacral root neuropathy because of uterine necrosis after pelvic vessel embolization.

Although the least frequent category, pregnancy- and childbirth-related neuropathies merit closer attention given their direct relevance to obstetric practice. Antepartum lesions are usually compressive, arising as the gravid uterus or the descending fetal head compresses the lumbosacral trunk against the pelvic brim, most commonly affecting the lumbosacral trunk, femoral and obturator nerves. Intrapartum mechanisms include prolonged lithotomy positioning, which stretches the femoral and common peroneal nerves, and instrumental (forceps) delivery. Recognised risk factors are nulliparity, a prolonged second stage of labour, cephalopelvic disproportion, and short maternal stature. Importantly, the great majority of obstetric neuropathies are demyelinating and self-limiting, resolving within weeks to a few months with conservative management; this probably explains both their low representation in the

literature and their frequent under-reporting. Persistent or progressive deficits, by contrast, should prompt imaging to exclude haematoma or another structural cause.

Trauma

Trauma was the aetiology in 8% (n=202 patients) of all included patients with pelvic neuropathies. We subdivided these patients into acute, chronic, haematoma and clotting disorders. Most studies in the acute trauma group described sciatic neuropathy after a fracture because of a fall, road traffic or sports accident (as displayed in Appendix B). In addition, blunt trauma, gunshot, stab wounds and lacerations were also described.[41,57] An avulsion fracture after a short run leading to sciatic or lateral femoral cutaneous neuropathy was reported by Dosani et al.[58] and Thanikachalam et al.,[59] respectively. More cases of neuropathies due to sport are described in the chronic category, for example a posterior femoral cutaneous neuropathy after a marathon[60] and a pudendal neuropathy after 2-3 hours of pilates exercises per day.[61] Other reported cases in the chronic trauma group mainly describe neuropathies after prolonged periods in certain positions. This could be in an altered state of consciousness due to medication, drugs, coma or by choice, like a sciatic neuropathy after hospitalisation with a pharmacologically altered state of consciousness[57] or after long-term gluteal pressure of a wallet.[62] All described haematomas after trauma led to femoral neuropathy specifically. We divided the cases with a haematoma and a clotting disorder into a separate group, possibly due to haemophilia or anticoagulants. Similarly, these haematomas caused femoral neuropathies, except for one case of sciatic neuropathy due to a pelvic haematoma as a first presentation of haemophilia.[63]

Compression-Muscular and Vascular

Neuropathy by muscle compression was only reported in case reports and small case series (n=41 patients, 2%). Hypertrophy of the muscle surrounding the pelvic nerves leading to neuropathy could be caused by muscle overuse or by imbalance from scoliosis or leg-length discrepancy.[62,64] Furthermore, abnormal piriformis muscle bundles or abnormal orientation of the nerve could lead to neuropathy in certain circumstances (described as piriformis syndrome). As seen in Appendix B, these included cases described neuropathies in the sciatic and pudendal nerves. In addition, five other studies described sciatic, femoral, obturator and lumbosacral root neuropathies due to rhabdomyolysis.[65,66] While neuropathy by compression of vessels was reported in fewer studies than muscular compression (16 studies vs. 18 studies), more patients were described (n=220, 9% vs. n=41, 2%). As displayed in Appendix B, compression of nerves by vessels could be due to aneurysms of arteries or dilation of veins. The largest two cohorts showed sciatic, pudendal, and lumbosacral root neuropathies by intrapelvic aberrant dilated veins.[67,68] Vascular entrapment syndromes are also described as a cause, but there was very little consensus about the definitions of the various pelvic congestion syndromes.[67-73]

Compression-Benign Tumours: Cysts, Fibrosis and Other

Only 1% of the included patients had pelvic neuropathy due to cysts (n=29). Most described were hydatid cysts (or echinococcosis), ganglion or synovial cysts, and Tarlov (also perineural or meningeal) cysts (as seen in Appendix B). One rare description of femoral neuropathy was presented in the case series of Ducic et al.[30] This neuropathy was idiopathic, as extensive workup had not yielded a direct cause or diagnosis; however, during neurolysis, tissues surrounding the femoral nerve appeared fibrotic and fixed to the inguinal ligament, which led to local swelling and hyperaemia of the nerve.[30] As displayed in Appendix B, other benign tumour aetiologies were very diverse, and 40 studies described 58 patients (2%). Multiple studies presented a patient with sciatic neuropathy after pyomyositis,[74] osteochondromas,[75] adenomyosis,[76] schwannomas[77] and lipomas.[75] Furthermore, next to sciatic neuropathy, uterine fibroids were described as the aetiology in obturator, lateral femoral cutaneous and lumbosacral root neuropathy.[78-81]

Compression-Malignant Tumours

In total, 17 case reports and series described 60 patients with pelvic neuropathies caused by malignant tumours (2%). Primary tumours or metastases mostly caused sciatic, femoral or lumbosacral root neuropathies (as seen in Appendix B). The largest case series, by Bickels et al.,[82] describes 18 cases with metastatic, bone or soft-tissue tumours, such as sarcomas. Another rare entity is described by Cucinella et al.,[83] presenting a case of sciatic neuropathy induced by retroperitoneal squamous cell carcinoma originating from endometriosis.

Specific Neuropathic Syndromes

Several aetiologies in this review are clinically grouped under named neuropathic syndromes, which deserve specific comment because their definitions are

inconsistent and they are frequently encountered—yet under-recognised—in gynaecological practice.

Piriformis syndrome denotes sciatic nerve irritation at the greater sciatic notch, attributed variously to piriformis hypertrophy, anatomical variants of the sciatic nerve, or post-traumatic scarring; as discussed below, the term is applied inconsistently and lacks agreed diagnostic criteria.

May-Thurner syndrome classically describes compression of the left common iliac vein by the overlying right common iliac artery. In the neuropelveological context it is implicated when the resulting venous congestion and dilated pelvic veins compress the lumbosacral plexus or sacral nerve roots, producing sciatic or pudendal symptoms.[68]

Pelvic congestion syndrome refers to CPP associated with dilated, refluxing pelvic veins. Its relationship to neuropathy is indirect—through perineural venous compression—and the literature shows little consensus on its definition or its distinction from other vascular entrapment syndromes.[67-73]

Alcock's canal (pudendal canal) syndrome is entrapment of the pudendal nerve within the fascial canal formed by the obturator internus fascia, and is probably the syndrome most relevant to—and most under-diagnosed by—gynaecologists. It produces neuropathic pain in the pudendal territory (clitoris, labia, perineum and anorectum), characteristically worsened by sitting and relieved by standing or by sitting on a toilet seat. Diagnosis rests on the clinical Nantes criteria,[88] supported where available by pudendal nerve motor latency testing and image-guided diagnostic nerve blocks. Management ranges from conservative measures and nerve blocks to laparoscopic or transgluteal pudendal nerve decompression. Greater familiarity with this syndrome would help gynaecologists distinguish pudendal neuralgia from vulvodynia and other causes of chronic perineal pain.

Discussion

Main Findings

This narrative review synthesises the published evidence on the identifiable aetiologies of pelvic neuropathy in women, encompassing 306 studies published between 1964 and 2024. Consistent with the literature, iatrogenic causes—particularly nerve injuries associated with pelvic surgery—were the most frequently reported mechanism of pelvic neuropathy. Other well-described mechanisms included nerve invasion or traction by pathological processes (including endometriosis and tumours), pregnancy- and childbirth-related injury, trauma, and extrinsic compression by benign or malignant structures.

Across the themes, a wide range of pelvic nerves was implicated, including the obturator, femoral, sciatic, pudendal, ilioinguinal, and lumbosacral plexus. Diagnostic approaches varied substantially, with many cases relying on clinical features alone, while others incorporated advanced imaging or neurophysiological testing. These themes provide a comprehensive framework for understanding the breadth of identifiable causes documented in the current literature.

Several included studies described a specific neuropathic syndrome, such as piriformis syndrome, May–Thurner syndrome, pelvic congestion syndrome and Alcock's canal syndrome. The problem with these terms is that there is very little consensus about their definitions, and the same syndrome label is used for patients with different aetiologies. For example, piriformis syndrome was used by Dere et al. (2009) to describe a patient with sciatic neuropathy because of hypertrophy of the unilateral piriformis muscle due to leg-length discrepancy.[84] However, Kraus et al. 2016 diagnosed piriformis syndrome in a patient with sciatic neuropathy because of variant anatomy of the left sciatic nerve,[85] while Siddiq et al. 2017 used the diagnosis of piriformis syndrome for patients with sciatic neuropathy after a fall or blunt trauma of the buttock.[62]

For the gynaecologist specifically, three messages stand out. First, nerve-invasive endometriosis should be considered in any woman with cyclical sciatic, gluteal or pudendal pain, since timely diagnosis can prevent irreversible nerve damage. Second, most obstetric neuropathies are self-limiting and can be managed conservatively with reassurance and physiotherapy, whereas persistent deficits warrant imaging. Third, pudendal (Alcock's canal) neuralgia is an under-recognised but treatable cause of chronic perineal pain that is frequently mislabelled as vulvodynia. Incorporating these considerations into the routine gynaecological assessment of CPP could reduce diagnostic delay and unnecessary symptom-only treatment.

Pelvic neuropathies in or after pregnancy were the smallest group of patients in our study (n=95 patients, 4%), possibly meaning that pelvic neuropathies are a rare complication of pregnancy and birth, perhaps because many pregnancy-related neuropathies are self-limiting.

Strengths and Limitations

A major strength of this review is its broad, inclusive approach, which allows us to map a large and heterogeneous body of evidence without excluding studies based on design or sample size. The use of inductive thematic synthesis enabled the identification of clinically meaningful groupings that reflect real-world mechanisms of nerve injury. The evidence base is, however, dominated by case reports and small case series, which provide only low-certainty evidence; we therefore refrained from a formal risk-of-bias assessment and instead interpret the findings descriptively. Several further limitations must be acknowledged. By design, the review included only studies in which a clear underlying aetiology of pelvic neuropathy was described, thereby excluding reports of idiopathic neuropathies or those lacking definitive diagnostic confirmation. This exclusion criterion—while enhancing specificity—may have led to underrepresentation of clinically relevant neuropathic presentations encountered in practice. Furthermore, diagnostic criteria were highly heterogeneous across studies, with many relying on clinical judgement without objective corroboration. Some case reports attributed causality based on temporal associations rather than definitive evidence, potentially introducing causal uncertainty. Finally, the predominance of case reports and small series limited the ability to derive robust epidemiological estimates.

Interpretation

The findings from this review confirm that pelvic neuropathy can result from a broad spectrum of identifiable causes, many of which remain under-recognised in clinical settings. The high prevalence of iatrogenic injuries emphasises the importance of surgical planning and anatomical awareness to prevent avoidable nerve damage. Other mechanisms, such as nerve invasion by disease, mechanical compression, and obstetric stretch injuries, underscore the need to consider neuropathic processes in patients presenting with chronic or atypical pelvic pain.

These results must be interpreted within the wider framework of CPP, a condition known to be multifactorial, involving nociceptive, myofascial, visceral, nociplastic, and central-sensitisation components alongside neuropathic contributions. Even when a neuropathic aetiology is confirmed, multidisciplinary management—including physical therapy, psychological support, pharmacological treatment, and interventional strategies—remains essential for optimal outcomes.[4,10]

This review also highlights a pressing need for standardised diagnostic frameworks. The lack of uniform imaging protocols and inconsistent application of neurophysiological testing limit diagnostic accuracy and reproducibility. Most studies relied on imaging (ultrasound, CT, or MRI), which often failed to detect neuropathy in the absence of a compressive lesion. EMG and NCSs were inconsistently used, and their diagnostic value remains uncertain. Pudendal neuropathy was occasionally assessed using the Nantes criteria,[88] but equivalent diagnostic frameworks are lacking for other pelvic nerves.

When a patient presents with suspected neuropathic pelvic pain, clinical reasoning should first distinguish between visceral and somatic pain, then localise the affected nerve and lesion site. The distinction between neurogenic (complete nerve lesion) and non-neurogenic (partial lesion) mechanisms, as described by Possover[5] can assist in refining the differential diagnosis. The aetiological categories synthesised in this review may support clinicians in structuring their diagnostic approach. However, the diagnostic process remains challenging. In addition to the absence of validated criteria, there is limited consensus on overlapping syndromes such as pelvic congestion or piriformis syndrome, further contributing to underdiagnosis.

Future research should prioritise the development of clear diagnostic criteria for pelvic neuropathies, enabling earlier recognition and facilitating access to structured, evidence-based care.

Conclusion

Pelvic neuropathy remains an under-recognised but clinically significant cause of CPP, with aetiologies ranging from iatrogenic injury to nerve invasion, trauma, compression, and obstetric complications. This review highlights the diversity of mechanisms and the diagnostic challenges associated with these conditions. Improved awareness, earlier recognition, and structured diagnostic pathways are essential to prevent misdiagnosis and enable timely, multidisciplinary intervention. Future research should aim to broaden the epidemiological understanding of pelvic neuropathies, including idiopathic cases, and evaluate treatment strategies that address both neuropathic and non-neuropathic contributors to pelvic pain.

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Appendix A Link: https://d2v96fxpocvxx.cloudfront.net/68ab204c-182b-49da-b227-bc7efe058632/content-images/99a04a40-6e9a-4799-85e2-9679a36663f7.pdf

Appendix B Link: https://d2v96fxpocvxx.cloudfront.net/bda9171a-fae8-4995-8276-2138323f1e16/content-images/721f9ea8-4ecd-4bc6-93a6-c021cb013baf.pdf

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