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Visceral pain and sensitisation in women with dysmenorrhea: a narrative review

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Abstract

Visceral pain (VP), from internal organs, is one of the main reasons for health consultations and represents a source of chronic pain. It can provoke referred musculoskeletal pain, viscero-somatic pain, hyperalgesia, and allodynia. The objective of this narrative review is to analyse the underlying mechanisms of somatic reflexed VP and viscero-visceral sensitisation processes that can occur in women with dysmenorrhea. The affectation of one viscera can lead to a sensitisation in those where metameric innervation is shared. In women with dysmenorrhea and painful menstrual periods, some studies find a concomitance increase in gastrointestinal pathologies. VP is a source of chronic pain, that favours the perception of referred pain in tissues where metameric innervation is shared. Women with dysmenorrhea have more possibilities to develop gastrointestinal pathologies like irritable bowel syndrome.

Key words: visceral pain, referred pain, dysmenorrhea

Introduction

Visceral pain (VP), which comes from internal organs, is one of the leading causes of medical consultation and represents a source of chronic pain (CP) [1, 2]. About 20% of adults have pain with a chest origin, 25% of them have abdominal intermittent pain, and between 16–25% of adult women have pelvic pain [3]. Clinical manifestations can be very different, from mild discomfort to acute pain, linked to renal colic or the menstrual cycle (dysmenorrhea) [4]. The most common manifestation of VP is functional gastrointestinal disorders (FGD). Irritable bowel syndrome (IBS) affects around 10–15% of the European population and the USA [4, 5]. It causes an overload of the healthcare system and an increase in costs [4, 6].

The objective of this paper is to develop a narrative review to provide the reader clarity on (1) VP as a source of CP, (2) to study its role in the perception of referred pain with a visceral origin in somatic tissues, and (3) to describe viscero-visceral sensitisations, especially in women with IBS and dysmenorrhea.

Theory

To develop this narrative review, we have consulted the databases PubMed and Cochrane, with the following keywords: visceral pain, dysmenorrhea, referred pain. The search strategy used is presented in Table 1.

Table 1. Keywords used

PubMed	Cochrane
– Pain, referred AND dysmenorrhea AND visceral pain – Dysmenorrhea AND visceral pain	– ((referred pain) AND dysmenorrhea) AND visceral pain – Dysmenorrhea AND visceral pain

The International Association for the Study of Pain (IASP) defined pain as "an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage" [7]. Pain and pain perception are different phenomena. Nociceptive stimulus is perceived by the neurons, and modulated by previous painful experiences and social and cultural factors. Pain has an adaptative role, that is learnt through life experiences and is linked to survival functions. It may alert the human body of homeostatic alterations [7, 8].

Chronic VP (CVP) is persistent or recurrent and originates in inner organs, or even from different regions like the head, neck, and thoracic, abdominal, or pelvic cavities [3]. It is imprecise and poorly localised. Usually, it provokes distant pain, referred pain to skin tissue, to muscles, or other visceras. It is usually perceived in somatic tissues (skin, subcutaneous cellular tissue, and muscles), in regions with the same innervation as the inner organ, where the symptom originated [1, 4, 9–11]. VP is supposed to be a maladaptation because it does not apport biological adaptative benefits. It's characterised by a spontaneous pain perception and referred pain as a response to a painful stimulus (hyperalgesia) or non-painful stimulus (allodynia) [12–14].

Visceral receptors are sensitive to thermal, mechanical, and chemical stimulus. Their depolarisation does not need to be linked to a real anatomic injury [3, 6]. The main causes of VP are persistent inflammation, vascular mechanisms (ischemia, thrombosis), mechanical (obstruction, distension, visceral traction, mesentery root traction, and compression), and emotional responses of the autonomous nervous system (ANS) [1, 2, 10, 15]. These mechanisms can even coexist. The viscera can react in different ways to a stimulus. Some studies indicate that VP is poorly studied [2, 9–11, 16].

Neural convergence and amplification

VP may cause changes in the excitability of sensory fibres [1]. It strengthens the local area of reflexed pain and the re-

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sponse to nociceptive stimulus [17]. In somatic areas of VP, it is possible to generate hyperalgesia, an increase of the sensibility to nociceptive stimulus and allodynia, perceived pain with stimuli that are usually not perceived as painful. The intensity of the pain perception is not usually related to tissue damage or the intensity of the visceral stimulus [1, 10, 18].

Nociceptive impulses that arrive at the spinal cord are vinculated through synapses with neurons that have direct connections with skin afferent neurons, somatic deep tissues, myotomes, and sclerotomes, that share the same spinal cord level as the affected viscera. These connections could facilitate pain perception in those localisations and mechanic hyperalgesia [9, 11, 19]. VP stimuli, through the contralateral ventrolateral tract and other upward tracts, project towards the brainstem, hypothalamus and other cortical areas [10]. They communicate in the spinal cord with second-order interneurons, which are under cortical control and can be inhibitory or excitatory [9–11].

All visceral afferents can be modulated between them. Consequently, there is a great network that contributes to pain perception [1, 9–11]. High-threshold nociceptors, usually silent, decrease their threshold under inflammatory conditions and can even contribute to amplifying the painful signals [1, 10]. Any stimulus that comes from tissues that share visceral metameric innervation, can facilitate somatic visceral referred pain perception, due to neural convergence [9, 11, 19].

Viscero-visceral sensitisation

Recurrent or intense VP facilitates somatic and visceral nociceptive processing. Viscero-visceral sensitisation is where the effects of one viscera can lead to an increase in pain perception in another, which shares metameric innervation [2, 5, 10, 20].

Visceral afferent innervation is defined as poor. Different convergent entrance impulses can arrive at the spinal cord, from other somatic structures where referred pain is radiated, or from another viscera [1, 10]. Visceral inflammation, distension, or visceral spasm can lead to changes in sensitive visceral pathways, amplifying the response to other irritative stimulus on the affected viscera and in those who share metameric innervation [1, 10, 18, 20].

Uterus-colon sensitisation in women with dysmenorrhea

Giamberardino [21] stated that chronic VP is produced mainly in women. That can occur in gynaecological VP and even in other types of VP. Between the uterus and sigmoid colon, a viscero-visceral sensitisation can happen. In women with dysmenorrhea and painful menstrual periods, they have found an increase in the sensitivity of the sigmoid colon and colonic hyperalgesia, compared to healthy women. Some studies have observed this phenomenon even in the absence of gastrointestinal symptomatology [5, 17]. They also are predisposed to develop IBS. Women with IBS, also show an increase of painful symptoms during menstruation [5, 22, 23].

Ayadilord et al. [24] studied IBS, premenstrual syndrome, and dysmenorrhea. They used validated questionnaires (Premenstrual Syndrome Screening Tool) about painful symptomatology and the Rome III criteria. They identified that women with concomitant pathologies, especially dysmenorrhea, suffered greater painful manifestations than those who did not have them [24]. Bahrami et al. [25] studied premenstrual pain and dysmenorrhea. They found that women with dysmenorrhea suffered more frequently from IBS compared to women with premenstrual syndrome [25]. They collected information about anthropometric measurements, demographic measurements, blood samples, dietary habits, and clinical characteristics of menstrual pain (signs, duration, severity).

Previous investigations, like Giamberardino et al. [22], studied pressure pain thresholds (PPT) for electrical skin stimulation during menstruation, in women with dysmenorrhea, healthy women, and men. They recorded measurements from the uterus viscerotome (abdomen and rectus abdominis), quadriceps, and deltoid. They measured pain intensity with a visual analogue scale (VAS) at different moments of the menstrual cycle. In women with dysmenorrhea, PPT increases during menstrual pain, during the menstrual phase, in the skin abdominal referred area, lower limb and subcutaneous cellular tissue. PPT in muscles and subcutaneous cellular tissue was reduced. Muscles were more vulnerable to menstrual influences than skin tissue of the thigh. In left abdominal muscles, they found a higher hyperalgesia. According to the study by Giamberardino et al. [22], in women with dysmenorrhea, pain perception in subcutaneous cellular tissue and muscles is facilitated. These changes lead to generalised hyperalgesia in muscles and subcutaneous cellular tissue, with no gender differences, according to the proximity of the stimulus to external genitals [22].

Brinkert et al. [5] identified how distension of the sigmoid colon generated colonic hyperalgesia in women with dysmenorrhea, compared to healthy women. They measured the referred pain area and the stimulus-response relationship between the sigmoid colon and rectum. They applied a distension in the sigmoid colon and rectum with a probe, in women with dysmenorrhea and healthy participants without gastrointestinal symptoms. They did not find hyperalgesia in somatic areas for colonic referred pain. Nevertheless, they found lower distension in women with dysmenorrhea, especially in the area related to the sigmoid colon. According to Brinkert et al. [5], those findings indicate that women with dysmenorrhea can develop intestinal hypersensitivity, even if they did not have gastrointestinal symptomatology beforehand. This phenomenon can be caused by hyperalgesia viscero-visceral, produced by dysmenorrhea [5].

Dysmenorrhea is also a leading risk factor for bladder pain syndrome. Oladosu et al. [26] found that patients with dysmenorrhea and bladder pain syndrome had an increased heart rate, increased diastolic blood pressure, and reduced heart rate variability, compared to controls. According to these authors, menstrual pain would be associated with abnormal autonomic activity and bladder sensitivity. Chung et al. [27] also found an association between bladder pain syndrome and dysmenorrhea.

More research is needed to verify these possible associations. However, different studies reinforce the idea of new strategies based on menstrual education, which improves the development of a supportive environment for students [28, 29].

Physiotherapy applies therapeutic strategies to reduce menstrual pain. Some research reflects the use of different physical agents for this purpose [30–34]. Physiotherapy can play a relevant role in addressing dysmenorrhea, acting on the pain of visceral and mechanical origins [33, 35–38]. Within these strategies, techniques such as therapeutic exercise [39], heat [33, 40] or TENS have already been studied, supporting their possible effectiveness in dysmenorrhea [33, 40]. Therapeutic exercise stands out as the technique with the largest number of studies [34, 36, 39, 41]. However, others, such as kinesiotapping [42, 43], present a smaller number of studies that address these symptoms.

Conclusions

Sensitisation of sigmoid colon happens in women with dysmenorrhea. It can trigger hyperalgesia, increasing the possibility of developing IBS, even if previously they had not had gastrointestinal symptoms. In women with IBS, it seems that painful clinical manifestations during menstruation are increased. More investigation is needed to clarify the underlying mechanisms of that sensitisation.

Ethical approval

The conducted research is not related to either human or animal use.

Informed consent

Informed consent has been obtained from all individuals included in this study.

Disclosure statement

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Conflict of interest

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References

[1] McMahon SB, Koltzenburg M. Wall and Melzack's Textbook of Pain [in Spain]. 5th ed. Madrid: Elsevier; 2007.
[2] Giamberardino M. Visceral Pain. Clinical, Pathophysiological and Therapeutic Aspects. Oxford: Oxford University Press; 2009.
[3] Aziz Q, Giamberardino MA, Barke A, Korwisi B, Baranowski AP, Wesselmann U, Rief W, Treede R-D; IASP Taskforce for the Classification of Chronic Pain. The IASP classification of chronic pain for ICD-11: chronic secondary visceral pain. Pain. 2019;160:69–76; doi: 10.1097/j.pain.0000000000001362.
[4] Sikandar S, Dickenson AH. Visceral pain: the ins and outs, the ups and downs. Curr Opin Support Palliat Care. 2012;6:17–26; doi: 10.1097/SPC.0b013e32834f6ec9.
[5] Brinkert W, Dimcevski G, Arendt-Nielsen L, Drewes AM, Wilder-Smith OHG. Dysmenorrhoea is associated with hypersensitivity in the sigmoid colon and rectum. Pain. 2007;132(Suppl 1):46–51; doi: 10.1016/j.pain.2006.12.011.
[6] Pacheco-Carroza EA. Visceral pain, mechanisms, and implications in musculoskeletal clinical practice. Med Hypotheses. 2021;153:110624; doi: 10.1016/j.mehy.2021.110624.
[7] Raja SN, Carr DB, Cohen M, Finnerup NM, Flor H, Gibson S, Keefe FJ, Mogil JS, Ringkamp M, Sluka KA, Song X-J, Stevens B, Sullivan MD, Tutelman PR, Takahiro U, Kyle V. The revised definition of pain by "International Association for the Study of Pain": concepts, challenges and compromises. Anaesth Pain Intensive Care. 2020;24(5):481–3; doi: 10.35975/APIC.V24I5.1352.
[8] Treede R-D. The International Association for the Study of Pain definition of pain: as valid in 2018 as in 1979, but in need of regularly updated footnotes. Pain Reports. 2018;3:e643; doi: 10.1097/PR9.0000000000000643.
[9] Elsenbruch S, Häuser W, Jänig W. Visceral pain [in German]. Schmerz. 2015;29(5):496–502; doi: 10.1007/s00482-015-0027-z.
[10] Jänig W. Neurobiology of visceral pain [in German]. Schmerz. 2014;28(3):233–51; doi: 10.1007/s00482-014-1402-x.
[11] Jänig W, Häbler HJ. Physiology and pathophysiology of visceral pain [in Geraman]. Schmerz. 2002;16(6):429–46; doi: 10.1007/s00482-002-0187-5.
[12] Arendt-Nielsen L. Central sensitisation in humans: assessment and pharmacology. Handb Exp Pharmacol. 2015;227:79–102; doi: 10.1007/978-3-662-46450-2_5.
[13] Ji RR, Nackley A, Huh Y, Terrando N, Maixner W. Neuroinflammation and central sensitisation in chronic and widespread pain. Anesthesiology. 2018;129(2):343–66; doi: 10.1097/ALN.0000000000002130.
[14] den Boer C, Dries L, Terluin B, van der Wouden JC, Blankenstein AH, van Wilgen CP, Lucassen P, van der Horst HE. Central sensitisation in chronic pain and medically unexplained symptom research: a systematic review of definitions, operationalisations and measurement instruments. J Psychosom Res. 2019;117:32–40; doi: 10.1016/j.jpsychores.2018.12.010.
[15] Dubin AE, Patapoutian A. Nociceptors: the sensors of the pain pathway. J Clin Invest. 2010;120(11):3760–72; doi: 10.1172/JCI42843.
[16] Van Oudenhove L, Kragel PA, Dupont P, Ly HG, Pazmany E, Enzlin P, Rubio A, Delon-Martin C, Bonaz B, Aziz Q, Tack J, Fukudo S, Kano M, Wager TD. Common and distinct neural representations of aversive somatic and visceral stimulation in healthy individuals. Nat Commun. 2020;11; doi: 10.1038/s41467-020-19688-8.
[17] Arendt-Nielsen L, Madsen H, Jarrell J, Gregersen H, Drewes AM. Pain evoked by distension of the uterine cervix in women with dysmenorrhea: evidence for central sensitisation. Acta Obstet Gynecol Scand. 2014;93(8):741–8; doi: 10.1111/aogs.12403.
[18] Montero-Homs J. Neuropathic pain patterns. Neuropathic pain. Definition, pathophysiology, semiology. Barcelona: Ars Medica; 2005.
[19] Hall EJ. Textbook of medical physiology. Guyton y Hall. 13th ed. Elsevier; 2016.
[20] Affaitati G, Costantini R, Tana C, Cipollone F, Giamberardino MA. Co-occurrence of pain syndromes. J Neural Transm. 2020;127(4):625–46; doi: 10.1007/s00702-019-02107-8.
[21] Giamberardino MA. Women and visceral pain: are the reproductive organs the main protagonists? Mini-review at the occasion of the "European Week Against Pain in Women 2007" Eur J Pain. 2008;12(3):257–60; doi: 10.1016/j.ejpain.2007.11.007.
[22] Giamberardino MA, Berkley KJ, Iezzi S, De Bigontina P, Vecchiet L. Pain threshold variations in somatic wall tissues as a function of menstrual cycle, segmental site and tissue depth in non-dysmenorrheic women, dysmenorrheic women and men. Pain. 1997;71(2):187–97; doi: 10.1016/S0304-3959(97)03362-9.
[23] Böttcher B, Gizewski ER, Siedentopf C, Steiger R, Verius M, Riedl D, Ischebeck A, Schmid J, Wildt L, Elsenbruch S. Behavioural and neural responses to aversive visceral stimuli in women with primary dysmenorrhoea. Eur J Pain. 2019;23(2):272–84; doi: 10.1002/ejp.1302.
[24] Ayadilord M, Mahmoudzadeh S, Hoseini ZS, Askari M, Rezapour H, Saharkhiz M, Abbaszadeh A, Karbasi S, Dashtebayaze NZ, Ferns GA, Bahrami A. Neuropsychological function is related to irritable bowel syndrome in women with premenstrual syndrome and dysmenorrhea. Arch Gynecol Obstet. 2020;302(4):915–23; doi: 10.1007/s00404-020-05659-3.

[25] Bahrami A, Gonoodi K, Khayyatzadeh SS, Tayefi M, Darroudi S, Bahrami-Taghanaki H, Eslami S, Jaberi N, Ferns GA, Farahmand K, Ghyour-Mobarhan M. The association of trace elements with premenstrual syndrome, dysmenorrhea and irritable bowel syndrome in adolescents. Eur J Obstet Gynecol Reprod Biol. 2019;233: 114–9; doi: 10.1016/j.ejogrb.2018.12.017.
[26] Oladosu FA, Hellman KM, Ham PJ, Kochlefl LE, Datta A, Garrison EF, Steiner ND, Roth GE, Tu FF. Persistent autonomic dysfunction and bladder sensitivity in primary dysmenorrhea. Sci Rep. 2019;9:2194; doi: 10.1038/s41598-019-38545-3.
[27] Chung S-D, Liu S-P, Lin H-C, Kang J-H. Association of dysmenorrhea with interstitial cystitis/bladder pain syndrome: a case-control study. Acta Obstet Gynecol Scand. 2014;93(9):921–5; doi: 10.1111/aogs.12437.
[28] Chéileachair FN, McGuire BE, Durand H. Coping with dysmenorrhea: a qualitative analysis of period pain management among students who menstruate. BMC Womens Health. 2022;22:1–11. doi: 10.1186/s12905-022-01988-4.
[29] Latthe P, Mignini L, Gray R, Hills R, Khan K. Factors predisposing women to chronic pelvic pain: systematic review. BMJ. 2006;332(7544):749–51; doi: 10.1136/bmj.38748.697465.55.
[30] Celenay ST, Kavalci B, Karakus A, Alkan A. Effects of kinesio tape application on pain, anxiety, and menstrual complaints in women with primary dysmenorrhea: a randomised sham-controlled trial. Complement Ther Clin Pract. 2020;39:101148; doi: 10.1016/j.ctcp.2020.101148.
[31] Azima S, Bakhshayesh HR, Kaviani M, Abbasnia K, Sayadi M. Comparison of the effect of massage therapy and isometric exercises on primary dysmenorrhea: a randomised controlled clinical trial. J Pediatr Adolesc Gynecol. 2015;28(6):486–91; doi: 10.1016/j.jpag.2015.02.003.
[32] Ortiz MI, Cortés-Márquez SK, Romero-Quezada LC, Murguía-Cánovas G, Jaramillo-Díaz AP. Effect of a physiotherapy program in women with primary dysmenorrhea. Eur J Obstet Gynecol Reprod Biol. 2015;194:24–9; doi: 10.1016/j.ejogrb.2015.08.008.
[33] Kannan P, Claydon LS. Some physiotherapy treatments may relieve menstrual pain in women with primary dysmenorrhea: a systematic review. J Physiother 2014;60: 13–21; doi: 10.1016/j.jphys.2013.12.003.
[34] Matthewman G, Lee A, Kaur JG, Daley AJ. Physical activity for primary dysmenorrhea: a systematic review and meta-analysis of randomised controlled trials. Am J Obstet Gynecol. 2018;219(3):255.e1–255.e20; doi: 10.1016/j.ajog.2018.04.001.
[35] Lefebvre G, Pinsonneault O, Antao O, Black A, Burnett M, Feldman K, Lea R, Robert M. Primary dysmenhorrea consensus guideline. J Obstet Gynaecol Can. 2005: 49–57.
[36] Kannan P, Claydon LS, Miller D, Chapple CM. Vigorous exercises in the management of primary dysmenorrhea: a feasibility study. Disabil Rehabil. 2015;37(15):1334–9; doi: 10.3109/09638288.2014.962108.
[37] Barassi G, Bellomo RG, Porreca A, Di Felice PA, Prosperi L, Saggini R. Somato-visceral effects in the treatment of dysmenorrhea: neuromuscular manual therapy and standard pharmacological treatment. J Altern Complement Med. 2018;24(3):291–9; doi: 10.1089/acm.2017.0182.
[38] Lauretti GR, Oliveira R, Parada F, Mattos AL. The new portable transcutaneous electrical nerve stimulation device was efficacious in the control of primary dysmenorrhea cramp pain. Neuromodulation 2015;18(6):522–6; doi: 10.1111/ner.12269.
[39] Armour M, Ee CC, Naidoo D, Ayati Z, Chalmers KJ, Steel KA, de Manincor MJ, Delshad E. Exercise for dysmenorrhoea. Cochrane Database Syst Rev. 2019;9(9): CD004142; doi: 10.1002/14651858.CD004142.pub4.
[40] Perez Machado AF, Perracini MR, de Morais ACS, da Silva BO, Driusso P, Liebano RE. Microwave diathermy and transcutaneous electrical nerve stimulation effects in primary dysmenorrhea: clinical trial protocol. Pain Manag. 2017;7(5):359–66; doi: 10.2217/pmt-2017-0021.
[41] Kannan P, Cheung K-K, Lau BW-M. Does aerobic exercise induced-analgesia occur through hormone and inflammatory cytokine-mediated mechanisms in primary dysmenorrhea? Med Hypotheses. 2019;123:50–4; doi: 10.1016/j.mehy.2018.12.011.
[42] Pawalia A, Panihar U, Yadav VS, Joshi S. Can taping reduce menstrual pain, distress, and anxiety? A systematic review and meta-analysis. Adv Rehabil. 2022;36(3): 49–59; doi: 10.5114/areh.2022.119587.
[43] Bandara EMIA, Kularathne WNI, Brain K, Weerasekara I. Safety and efficacy of therapeutic taping in primary dysmenorrhea: a systematic review and meta-analysis. Sci Rep. 2021: 12(1):7146; doi: 10.1038/s41598-022-11034-w.