

Review on Implementing Patient-Centered Care in Primary Health Practice in Qatar

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Abstract

Patient-centered care is a critical component of health-care delivery, emphasizing the involvement of patients in their care and the consideration of their preferences and needs. In Qatar, a country dedicated to providing high-quality healthcare, the implementation of patient-centered care in primary health practices is of paramount importance. This literature review examines the existing literature on patient-centered care implementation in primary health practices in Qatar, focusing on strategies, challenges, and outcomes. The review underscores the significance of cultural considerations, patient engagement, provider training, health information technology integration, quality improvement initiatives, multidisciplinary collaboration, and care coordination. Findings highlight the need for cultural competency training, effective communication, shared decision-making, provider education, health information technology integration, patient feedback mechanisms, multidisciplinary teamwork, and patient involvement in quality improvement efforts. While limited research specifically focuses on Qatar, international evidence supports the positive outcomes of patient-centered care, including improved patient satisfaction, treatment adherence, health outcomes, and potentially reduced healthcare costs. Further research is needed to evaluate the outcomes and impacts of implementing patient-centered care in primary health practices in Qatar and develop tailored guidelines and strategies for successful implementation.

Keywords: Patient centered care, Primary health practice, Qatar

Introduction

Early in its formation, the FMAHealth Board recognized the importance of including a patient advocate voice on the board to inform policy decisions (Stollenwerk et al., 2019). Patient-centered care is a fundamental aspect of healthcare delivery that places the patient at the center of their care and is a core principle in healthcare that focuses on involving patients as active participants in their care, considering their values, preferences, and needs. In Qatar, a country that is committed to providing high-quality healthcare services, implementing patient-centered care in primary health care settings is crucial. This literature review examines the existing literature on the implementation of patient-centered care in primary healthcare settings in Qatar, exploring key strategies, challenges, and outcomes.

Methods

A comprehensive search was conducted using electronic databases, including PubMed, Medline, and Google Scholar, to identify relevant articles published in English between 2013 and 2023. The Studies and review articles focusing specifically on patient-centered care in primary health practices in Qatar were included in this review.

Key words: “patient-centered care,” “primary health care,” “Qatar,” “implementation,” and related keywords.

Results

Strategies for Implementing Patient-Centered Care:

In addition to adding a patient representative to the board and in pursuit of obtaining a greater understanding of how best to approach transformation of primary care delivery to promote patient-centeredness, the board also created and charged an Engagement Tactic Team with two primary objectives:

1. To engage patients as partners in transforming primary care practices and the health care system at large in order to enhance the patient experience, improve community health, and reduce costs; and
2. To strengthen working alliances with other primary care professions and other stakeholders in order help all speak with a unified voice for primary care (Stollenwerk et al., 2019).

Several strategies have been proposed to promote patient-centered care in Qatar. These include improving healthcare provider communication skills, fostering a collaborative and empathetic patient-provider relationship, involving patients in decision-making through shared decision-making approaches, and integrating patient perspectives into quality improvement initiatives. Features implemented to demonstrate PFCC in Qatar’s health centers include access to a physician whenever required, and automated confirmation of an appointment to hospital outpatients by Short Message Service (SMS) (Verjee & Robertson-Malt, 2013).

1. Cultural Considerations:

Patient-centered care cultural transformation is a complex and long-term endeavor (Bokhour et al., 2018). Qatar has a diverse population with varying cultural backgrounds. Implementing patient-centered care requires understanding and respecting cultural values, beliefs, and preferences. Studies emphasize the importance of cultural competency training for healthcare providers to effectively communicate and provide care that aligns with patients’ cultural expectations.

2. Patient Engagement and Shared Decision-Making:

Such signs orienting both staff and patients to the innovations driving PCC cultural change were present throughout the facility (Bokhour et al., 2018). Patient engagement and shared decision-making are fundamental to patient-centered care. Studies in Qatar highlight the importance of empowering patients to actively participate in their healthcare decisions. Strategies such as friends of the health center, effective communication, patient education, and decision aids are identified as effective tools for facilitating shared decision-making in primary health practices.

3. Provider Training and Education:

It was critical that staff see PCC as essential to care, not as another fleeting VA initiative (Bokhour et al., 2018). Healthcare provider training and education play a crucial role in implementing patient-centered care. Studies underscore the need for training programs that enhance communication skills, empathy, cultural competency, and shared decision-making among healthcare providers in Qatar. Continuing education programs and workshops are recommended to support providers in delivering patient-centered care.

4. Health Information Technology (HIT) Integration:

The abrupt onset of the coronavirus disease 2019 (COVID-19) pandemic required a rapid implementation of telemedicine—the synchronous delivery of health care in an audio-plus-video or audio-only modality—in primary care (Rabinowitz et al., 2023). The integration of health information technology (HIT) can support patient-centered care in primary health practices. Studies highlight the importance of implementing electronic health records (EHRs), patient portals, and telemedicine platforms in Qatar. Unique benefits of telemedicine over in-person visits, ranging from reduced concerns regarding transmission of infections to improved access for patients with limited mobility (Rabinowitz et al., 2023). HIT integration can improve communication, enhance care coordination, and facilitate patient access to medical information by My health mobile application or Sehaty.

5. Quality Improvement Initiatives:

Quality improvement initiatives are essential for implementing patient-centered care. Capturing the patients’ voices, obtaining patient perspectives, and finding out what matters most to patients and families were essential to selecting, planning, and implementing PCC initiatives (Bokhour et al., 2018). Studies emphasize the need for collecting patient feedback, conducting

patient satisfaction surveys, and involving patients in quality improvement efforts in primary health practices in Qatar. Feedback mechanisms and patient engagement in quality improvement initiatives contribute to the delivery of patient-centered care.

6. Multidisciplinary Collaboration and Care Coordination:

Collaboration with other people-centered organizations such as government ministries, academic institutions, and civil society associations locally, regionally and internationally, will raise awareness of PFCC (Verjee & Robertson-Malt, 2013). Patient-centered care necessitates collaboration among healthcare professionals in primary health practices. Studies in Qatar highlight the importance of multidisciplinary teamwork and care coordination to provide comprehensive and coordinated care. Effective communication channels, regular team meetings, and care planning are identified as facilitators of patient-centered care.

7. Recognizing the Outcomes and Impacts:

Limited studies directly investigating the outcomes and impacts of implementing patient-centered care in primary health practices in Qatar were found. However, international evidence suggests that patient-centered care leads to improved patient satisfaction, better adherence to treatment plans, enhanced health outcomes, and potentially reduced healthcare costs and improved healthcare provider-patient relationships.

Conclusion

The literature on implementing patient-centered care in primary health practices in Qatar highlights the significance of cultural considerations, patient engagement, provider training, health information technology integration, quality improvement initiatives, multidisciplinary collaboration, and care coordination. It also highlights the conceptual framework, benefits, and strategies for implementing patient-centered care in clinical practice. By embracing patient-centered care principles, Qatar can enhance the patient experience, improve health outcomes, and strengthen its primary health care system to meet the evolving needs of its diverse population.

While there is limited research specifically focusing on Qatar, the existing evidence underscores the importance of these factors in delivering patient-centered care. Further research is needed to evaluate the outcomes and impacts of implementing patient-centered care in primary health practices in Qatar and to develop tailored guidelines and strategies for successful implementation.

References

- Bokhour, B. G., Fix, G. M., Mueller, N. M., Barker, A. M., LaVela, S. L., Hill, J. N., Solomon, J. L., & Lukas, C. V. (2018). How can healthcare organizations implement patient-centered care? Examining a large-scale cultural transformation. *BMC Health Services Research*, 18(1). <https://doi.org/10.1186/s12913-018-2949-5>
- Rabinowitz, G., Cho, L. D., Benda, N. C., Goytia, C., Andreadis, K., Lin, J. J., Horowitz, C., Kaushal, R., Ancker, J. S., & Poeran, J. (2023). The telemedicine experience in primary care practices in the United States: Insights from practice leaders. *Annals of Family Medicine*, 21(3), 207–212. <https://doi.org/10.1370/afm.2967>
- Stollenwerk, D., Kennedy, L. B., Hughes, L. S., & O'Connor, M. (2019). A systematic approach to understanding and implementing patient-centered care. *Family Medicine*, 51(2), 173–178. <https://doi.org/10.22454/FamMed.2019.320829>
- Verjee, M. A., & Robertson-Malt, S. (2013). Patient- and family-centered care in Qatar: A primary care perspective. *Avicenna*, 2013(1). <https://doi.org/10.5339/avi.2013.1>

Graves Eye Disease Medical and Surgical Management: A Review

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Abstract

Graves' disease (GD) is the most frequent cause of hyperthyroidism, where iodine levels are abundant.

One of the extrathyroidal symptoms is Graves' ophthalmopathy (GO) which presents with ophthalmic symptoms that can range from minor (e.g., dry eye) to sight-threatening (e.g., corneal ulceration and compressive optic neuropathy) features.

About 79% of Graves' disease cases can be attributed to genetic predispositions, while the remaining 21% are due to environmental factors. Acute stress, active or passive smoking, and past radioactive iodine therapy have all been linked to the development or aggravation of thyroid eye disease (TED).

The devastating effects of GO or TED might include diplopia, ocular hypertension, optic nerve degeneration, and glaucoma.

A low basal serum Thyroid Stimulating hormone (TSH) level has the highest sensitivity and specificity for diagnosing hyperthyroidism. Moreover, the appearance

of Thyroid Stimulating hormone receptors (TSHR) autoantibodies (TRAbs) is presumed to be highly specific for the diagnosis of Graves' disease.

Imaging studies of the orbit that use ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI), for example, can confirm the diagnosis of TED.

In order to treat Graves's eye disease optimally, a multidisciplinary approach must be applied involving primary care physicians, ophthalmologists, internists and endocrinologists. Therefore, it is essential to restore the euthyroid state and this can be obtained by either antithyroid medications, radioactive iodine or surgical thyroidectomy.

Treatment of GO ranges from supportive treatment (lubricants and moisturizer drops), to medical intervention, preferably corticosteroid, and variable surgical interventions.

Key words: Graves' disease, Medical and surgical management

List of abbreviations:

GD: Graves disease

TED: thyroid eye disease

GO: Graves ophthalmopathy

TSH: thyroid stimulating hormone

TSHR: thyroid stimulating hormone receptors

TRAbs: Thyroid receptors autoantibodies

CT: computed tomography

MRI: magnetic resonance imaging

RAI: radioactive iodine

ATD: Antithyroid drugs

HLA: human leukocyte antigen

Introduction

Graves' disease (GD) is the most frequent cause of hyperthyroidism, where iodine levels are abundant. The existence of antibodies against the TSH receptor is termed TSH receptor antibodies (TRAb) [1]. The incidence of GD is approximately 40/100,000 per year [2]. Women are more likely than men to have GD, and individuals aged between 30 and 50 years are most commonly affected. Extrathyroidal symptoms such as Graves' ophthalmopathy (GO), thyroid dermopathy, and acropachy may also be present in addition to hyperthyroidism [3].

The development of this autoimmune illness is influenced by both genetic and environmental factors. Antithyroid drugs (ATDs) are the mainstay of medical treatment for GD [4,5]. However, thyroid ablation, either thyroidectomy or radioiodine (RAI) treatment, is necessary in around half of cases due to the high recurrence rate of hyperthyroidism. The devastating effects of GO or thyroid eye disease (TED) might include diplopia, ocular hypertension, optic nerve degeneration, and glaucoma. A patient's quality of life may be negatively impacted by even mild TED [7]. Although TED is more common in younger women, studies suggest that men and older people are at a higher risk of developing this serious illness [8]. Those with unstable thyroid function or specific anatomical features of the orbit, such as a larger lateral orbital wall angle, are also more likely to develop TED [9]. Acute stress, active or passive smoking, and past radioactive iodine therapy have all been linked to the development or aggravation of TED [10,11].

The current review article aims to summarize recent advances in our understanding of the pathophysiology of GD and clinical considerations for diagnosing, prognosticating, and treating GD patients [6].

Methodology

A review of the literature was performed to find scholarly publications about TED through a systematic web search. Multiple keywords, including epidemiology, etiology, pathophysiology, clinical features, diagnosis, medical and surgical interventions of TED, were used to search in research databases Google Scholar and PubMed. Among 84 articles retrieved (published between 1988 and 2021), 48 articles were included in the study. Articles were excluded if they are not directly linked to the research topic. Duplicates were also removed after the final retrieving process.

The Review of Literature

Graves' Disease Epidemiology

Graves' disease is the leading cause of hyperthyroidism. Many studies have examined the incidence of hyperthyroidism, but only a few assessed Graves' disease as a cause of hyperthyroidism. Graves' disease is caused by an immune system malfunction, which fights diseases

in the body. About 79% of Graves' disease cases can be attributed to genetic predispositions, while the remaining 21% are due to environmental factors [12]. Common environmental risk factors include vitamin D and selenium deficiency, smoking, and changes in iodine levels. The depletion of iodine in the body can significantly increase its incidence, but the long-term changes in iodine level are not considered a risk factor. It is also believed that stress and pregnancy may increase the risk of developing Graves' disease [12].

Statistics indicate that Graves' disease affects about 40 in every 100,000 people yearly, with an estimated prevalence of 0.4%. However, these statistics are from the retrogressive analysis of available medical records. Hence, these figures may be underestimated and not representative, as patients with mild symptoms are often undiagnosed. The prevalence of Graves' disease is higher in women than men. Its prevalence in the United States was about 0.4% in the 1970s. A United States survey found Graves' disease to be more common among Caucasians than other races [13]. Research conducted in the United Kingdom showed a prevalence rate of about 1.1% to 1.6%. As far as demographics are concerned, people can be affected at any age, but its prevalence is higher between the ages of 30 and 50 years.

The prevalence of Graves' disease is fairly evenly distributed across the globe; however, its incidence is higher in areas with rich iodine consumption, such as India. A recent population study in India indicated that 16.7% of the population suffered from Graves' disease, with those with metabolic syndrome accounting for more than 40%. The same study also indicated that the prevalence was higher in women than in men [14].

Epidemiology of Thyroid Eye Disease (TED)

Graves' ophthalmopathy is a complex inflammation disease of the orbit. Most patients with TED have a biochemical indication of hyperthyroidism, with Graves' disease being the most common. Thyroid eye disease affects about 16 in 100,000 people among women and 3 in 100,000 men, with an average prevalence of about 0.25% [15]. There is no defined ethnic predisposition of TED. The high incidence of TED among women can be attributed to the higher incidence of hyperthyroidism disease among women; however, the disease severity is more pronounced among men [15].

Common risk factors of TED include the female gender, smoking, young age, and hyperthyroidism. The treatment of hyperthyroidism using radioiodine is also a risk factor. The presence of other autoimmune thyroid illnesses can account for up to 15% of the total TED diseases; however, genetic factors are the main risk factors, especially for people with susceptibility alleles [15]. TED usually manifests itself at the beginning of hyperthyroidism and could take five years of treatment. A significantly small proportion of patients have no history of hyperthyroidism. Research shows a decrease in the prevalence in the last two decades, but little justification exists [16]. It is challenging

to determine the definitive prevalence of the TED disease due to insufficient data. However, a study conducted in Olmstead County in the United States showed a bimodal peak for men and women aged between 40 and 44 years and 60 and 64 years. The same study also indicated that about 50% of the patients with Graves' disease have clinically apparent TED [17].

More than 66% of the patients will experience TED either six months before the onset of thyroid disease, or thyroid dysfunction. The natural history of TED consists of two phases; the active inflammatory stage and the static stage. The active inflammatory stage is the first phase; the static stage follows. Only about 5% of TED patients have a late reactivation. Despite TED having no ethical depositions, people of Asian origin tend to have mild manifestations compared to Caucasians [18].

Pathophysiology of GD and GO:

It is widely acknowledged that GD has a substantial hereditary component, with genetic factors playing a key role. Several investigations have established that the main genes causing GD include human leukocyte antigen (HLA), CD40, CTLA-4, PTPN22, Tg, and TSHR. On chromosome 6, the HLA complex contains sequences that code for genes important in controlling the immune response [6]. The involvement of the central tolerance, which is impacted by the production of self-antigens (such as TSHR) within the thymus for negative selection of autoreactive T cell clones, is another factor in the genesis and pathophysiology of GD. Polymorphisms of certain tissue-restricted genes that encode autoantigenes might affect their degree of expression in the thymus, becoming a risk factor for autoimmunity [19,20].

As a component of GD, Graves' ophthalmopathy (GO, often referred to as Graves' orbitopathy) is an autoimmune inflammatory disorder [21]. There are several risk factors for GD-related GO. GO is more prevalent in women than men, and the risk of developing severe GO seems to be higher in males with GD [22]. Moreover, there are ethnic disparities in the frequency of GO, with Asians being less likely than Caucasians to contract the disease [23]. Moreover, the aforementioned hereditary variables are relevant, and smoking is a significant additional risk factor [24]. The activation of autoantibodies to thyroid stimulating hormone (TSH, thyrotropin) receptors (TSHR) appears to be the triggering event in thyroid eye disease, despite the complex underlying molecular mechanisms [25,26]. TSHR is overexpressed in the retrobulbar tissue of Graves' and hyperthyroidism patients compared to controls, especially in orbital fibroblasts, which are crucial to the pathophysiology of thyroid eye disease [21,25,27,28].

Orbital fibroblasts multiply and produce pro-inflammatory cytokines and hydrophilic hyaluronan in the interstitial space when activated [21,25,27]. These mechanisms cause a high osmotic pressure gradient in the orbit, causing greater fluid collection between the muscle fibers. Moreover, some orbital fibroblasts develop into mature adipocytes, resulting in orbital adipose tissue growth [26–28]. This cycle continues, and orbital congestion may result

[21,27]. Long-lasting edema causes fibrosis, sclerosis, and the extraocular muscles to atrophy, resulting in restricted strabismus [29].

Clinical Features

Clinical symptoms are linked to both the autoimmune and hyperthyroidism processes. The signs and symptoms of GD can vary greatly and significantly impact general health since excess thyroid hormones affect many different body systems. Tremors, heat sensitivity and warmth, weight loss despite regular eating habits, anxiety and irritability, goiter, and changes in menstrual cycles are common symptoms [30]. Ophthalmic symptoms can range from minor (e.g., dry eye) to sight-threatening (e.g., corneal ulceration and compressive optic neuropathy) problems, and treatment can range from supportive (e.g., lubrication of the ocular surface) to surgical (e.g., orbital decompression) approaches. Due to various clinical presentations, various disorders, such as allergic conjunctivitis and orbital tumors, are included in the differential diagnosis [31].

The devastating effects of GO and TED might include diplopia, ocular hypertension, optic nerve degeneration, and glaucoma [7]. Lid retraction, proptosis, soft tissue edema, strabismus, and compressive optic neuropathy are some of the clinical signs and symptoms of GO. The globe is pushed forward by the enlarged soft tissues inside the bony orbit, which also prevents venous outflow from the orbit. The adipogenesis and glycosaminoglycan buildup that follows the local fibroblasts' activation due to inflammation, causes enlarged soft tissues. Furthermore, lymphocyte infiltration and tissue remodeling are seen in the GD orbital symptoms, which might lead to fibrosis [32].

Diagnosing Graves' Disease

A low basal serum TSH level has the highest sensitivity and specificity for diagnosing hyperthyroidism and should, therefore, be used as an initial screening parameter. However, if Graves' disease is strongly suspected, diagnostic accuracy improves when serum TSH, free T4, and free T3 are also assessed [33]. Moreover, the appearance of TSHR autoantibodies (TRAbs) is presumed to be highly specific for the diagnosis of Graves' disease. Therefore, the diagnosis is usually confirmed by demonstrating elevated TRAbs [34].

Imaging studies of the orbit that use ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI), for example, can confirm the diagnosis of TED while excluding other diagnoses such as orbital tumor and idiopathic orbital inflammation (previously known as orbital pseudotumor). Computerized tomography without contrast remains the standard radiographic technique because of its ability to display the bony anatomy of the orbit and its low cost relative to MRI [35].

In ultrasound research, Graves' disease is usually characterized by hypoechoic and heterogeneous parenchyma, diffusely enlarged, and hypervascularity. In

contrast, Technetium-99 (99Tc) scanning of the thyroid is limited in diagnosing GD due to the high sensitivity and specificity of TRAb measurement [36,37].

Diagnosing Graves' Ophthalmopathy

The diagnosis of TED is straightforward based on the clinical history and physical examination of patients. Ophthalmic manifestations are present in up to 50% of Graves' hyperthyroidism patients [25]. TED follows a biphasic course: a progressive or active phase lasting up to three years, followed by a stable or inactive phase [26]. Ophthalmic manifestations can vary from mild (for example, dry eye) to sight-threatening (for example, corneal ulceration and compressive optic neuropathy) problems.

There is no single clinical finding or laboratory test that can diagnose TED. Frequently, the presenting symptoms are non-specific dry eye complaints, such as foreign body sensation, redness, blurring of vision, photophobia, glare, or excessive tearing [3,21]. However, there are many additional symptoms, including concern about cosmesis, retrobulbar discomfort, swelling of the eyelids worse in the morning, diplopia, and uncommon loss of vision [38, 39]. Common clinical signs are upper eyelid retraction, conjunctival and caruncle injection and/or edema, eyelid edema and/or erythema with diurnal variation, ocular motility disruption, or strabismus and proptosis [38,40]. Paradoxically, upper eyelid ptosis can also be a presenting sign of TED [41]. The clinical evaluation for TED focuses on determining clinical activity and severity by assessing visual acuity, pupils, color vision, extraocular movements, visual field, exophthalmometry, external eyelid evaluation, slit-lamp examination, and dilated fundus examination.

Medical and surgical treatment of Graves eye disease

Managing thyroid disease:

In order to treat Graves's eye disease optimally, a multidisciplinary approach must be applied involving primary care physicians, ophthalmologists, internists and endocrinologists [42, 43]. Therefore, it is essential to restore the euthyroid state and this can be obtained by either antithyroid medications, radioactive iodine or surgical thyroidectomy[40]. Some reports have shown that Radioactive iodine may result in the development or aggravation of thyroid eye disease by 15-20% in those who are smokers [44]. These adverse effects could be minimized by using oral corticosteroids post-radioactive iodine [43,45]. Smoking is a known risk factor for the progression of thyroid eye diseases [46,11]. Smoking cessation is considered one of the most important modifiable risk factors in the prevention of thyroid eye disease [47]. Therefore, it is advised for the patient with thyroid disease to stop smoking [42,48].

Graves eye disease treatment

Supportive treatment:

Some mild cases of graves eye diseases can be managed conservatively, for patients with dry eye manifestation, lubricants and moisturizer drops could be used [26]. Sunglasses are recommended to minimize photosensitivity and glare. For eyelids retraction, botulinum toxin injections could be used on the levator palpebrae superioris and Muller's muscles [31].

Medical treatment:

For patients with moderate to severe Graves' eye disease, corticosteroid is the mainstay treatment option. Almost 80% of patients on high intravenous corticosteroid show improvement in their condition in comparison to oral steroid which is less effective and with more side effects [31]. An immunosuppressive agent such as rituximab has shown some potential in the treatment of thyroid eye disease. However, some studies reported conflicting results [31]. Selenium supplementation has been shown to have the potential to improve the quality of life and reduces the severity and progression of thyroid eye disease [31].

Surgical treatment

When there is a significant impact on visual function or quality of life, individuals with moderate-to-severe inactive thyroid eye disease may consider surgical rehabilitation [42, 31]. In general, orbital decompression is done first, then extraocular muscle surgery, and finally eyelid procedures are done while treating inactive thyroid eye illness [26].

Several methods of orbital bone decompression and the amount of removed orbital walls have been researched. One approach hasn't proven itself to be better than the others up to this point [26].

For the purpose of reducing proptosis and improving diplopia, orbital fat decompression can be done either in conjunction with bone decompression or on its own [31]. Temporary tarsorrhaphy can be used to treat exposure keratopathy while waiting for orbital decompression [26]. In order to maintain enough corneal covering, eyelid surgery is only done for symptomatic eyelid retraction or asymmetric lid position [26].

Complications of thyroid eye diseases

Thyroid eye disease may lead to diplopia, ocular hypertension, glaucoma and optic nerve damage. Even mild thyroid eye disease could have a significant effect on the patient's quality of life [7].

Conclusion

Graves' disease is a common condition that can be associated with ocular manifestations that range from mild symptoms like dry eye to severe ones like corneal ulceration and compression to optic disc. Presentation in the eye could be devastating to the patient hence affecting the quality of life. Management of TED can range from medical options to a variety of surgical interventions. Therefore, healthcare providers must be aware of its clinical presentations and treatment modalities.

References

- 1- Bartalena L, Chiovato L, Vitti P. Management of hyperthyroidism due to Graves' disease: frequently asked questions and answers (if any). *Journal of endocrinological investigation*. 2016 Oct;39:1105-14
- 2- Weetman AP. Graves' Disease 1835–2002. *Hormone Research in Paediatrics*. 2003;59(Suppl. 1):114-8.
- 3-Bartalena L, Fatourechi V. Extrathyroidal manifestations of Graves' disease: a 2014 update. *Journal of endocrinological investigation*. 2014 Aug;37:691-700.
- 4-Burch HB, Cooper DS. Management of Graves disease: a review. *Jama*. 2015 Dec 15;314(23):2544-54.
- 5-Marinò M, Latrofa F, Menconi F, Chiovato L, Vitti P. An update on the medical treatment of Graves' hyperthyroidism. *Journal of Endocrinological Investigation*. 2014 Nov;37:1041-8.
- 6-Ehlers M, Schott M, Allelein S. Graves, disease in clinical perspective. *Frontiers in Bioscience-Landmark*. 2019 Jan 1;24(1):33-45.
- 7-Choi YJ, Lim HT, Lee SJ, Yoon JS. Assessing Graves' ophthalmopathy-specific quality of life in Korean patients. *Eye*. 2012 Apr;26(4):544-51.
- 8-Kendler DL, Lippa J, Rootman J. The initial clinical characteristics of Graves' orbitopathy vary with age and sex. *Archives of ophthalmology*. 1993 Feb 1;111(2):197-201.
- 9-Stan MN, Bahn RS. Risk factors for development or deterioration of Graves' ophthalmopathy. *Thyroid*. 2010 Jul 1;20(7):777-83.
- 10-Traisk F, Tallstedt L, Abraham-Nordling M, Andersson T, Berg G, Calissendorff J, Hallengren B, Hedner P, Lantz M, Nystrom E, Ponjavic V. Thyroid-associated ophthalmopathy after treatment for Graves' hyperthyroidism with antithyroid drugs or iodine-131. *The Journal of Clinical Endocrinology & Metabolism*. 2009 Oct 1;94(10):3700-7.
- 11- Vestergaard P. Smoking and thyroid disorders—a meta-analysis. *European Journal of Endocrinology*. 2002 Feb;146(2):153-61.
- 12- Smith, T. J., & Hegedüs, L. (2016). Graves' disease. *New England Journal of Medicine*, 375(16), 1552-1565.
- 13- Antonelli, A., Ferrari, S. M., Ragusa, F., Elia, G., Paparo, S. R., Ruffilli, I., ... & Fallahi, P. (2020). Graves' disease: Epidemiology, genetic and environmental risk factors and viruses. *Best Practice & Research Clinical Endocrinology & Metabolism*, 34(1), 101387.
- 14- Wémeau, J. L., Klein, M., Sadoul, J. L., Briet, C., & Vélayoudom-Céphise, F. L. (2018, December). Graves' disease: introduction, epidemiology, endogenous and environmental pathogenic factors. In *Annales d'endocrinologie* (Vol. 79, No. 6, pp. 599-607). Elsevier Masson.
- 15- Chin, Y. H., Ng, C. H., Lee, M. H., Koh, J. W. H., Kiew, J., Yang, S. P., ... & Khoo, C. M. (2020). Prevalence of thyroid eye disease in Graves' disease: A meta-analysis and systematic review. *Clinical endocrinology*, 93(4), 363-374.
- 16- Hiromatsu, Y., Eguchi, H., Tani, J., Kasaoka, M., & Teshima, Y. (2014). Graves' ophthalmopathy: epidemiology and natural history. *Internal Medicine*, 53(5), 353-360.
- 17- Lazarus, J. H. (2012). Epidemiology of Graves' orbitopathy (GO) and relationship with thyroid disease. *Best Practice & Research Clinical Endocrinology & Metabolism*, 26(3), 273-279.
- 18- Debnam, J. M., Koka, K., & Esmaeli, B. (2021). Extrathyroidal manifestations of thyroid disease: graves eye disease. *Neuroimaging Clinics*, 31(3), 367-378.
- 19- Colobran R, Armengol Mdel P, Faner R, Gärtner M, Tykocinski LO, Lucas A, Ruiz M, Juan M, Kyewski B, Pujol-Borrell R. Association of an SNP with intrathymic transcription of TSHR and Graves' disease: a role for defective thymic tolerance. *Hum Mol Genet*. 2011 Sep 1;20(17):3415-23. doi: 10.1093/hmg/ddr247. Epub 2011 Jun 3. PMID: 21642385.
- 20- Stefan M, Wei C, Lombardi A, Li CW, Concepcion ES, Inabnet WB 3rd, Owen R, Zhang W, Tomer Y. Genetic-epigenetic dysregulation of thymic TSH receptor gene expression triggers thyroid autoimmunity. *Proc Natl Acad Sci U S A*. 2014 Aug 26;111(34):12562-7. doi: 10.1073/pnas.1408821111. Epub 2014 Aug 13. PMID: 25122677; PMCID: PMC4151767.
- 21- Bahn RS. Graves' ophthalmopathy. *N Engl J Med*. 2010 Feb 25;362(8):726-38. doi: 10.1056/NEJMra0905750. PMID: 20181974; PMCID: PMC3902010.
- 22- Burch HB, Wartofsky L. Graves' ophthalmopathy: current concepts regarding pathogenesis and management. *Endocr Rev*. 1993 Dec;14(6):747-93. doi: 10.1210/edrv-14-6-747. PMID: 8119236.
- 23- Tellez M, Cooper J, Edmonds C. Graves' ophthalmopathy in relation to cigarette smoking and ethnic origin. *Clin Endocrinol (Oxf)*. 1992 Mar;36(3):291-4. doi: 10.1111/j.1365-2265.1992.tb01445.x. PMID: 1563082.
- 24- Wiersinga WM, Bartalena L. Epidemiology and prevention of Graves' ophthalmopathy. *Thyroid*. 2002 Oct;12(10):855-60. doi: 10.1089/105072502761016476. PMID: 12487767.
- 25- Douglas RS, Gupta S. The pathophysiology of thyroid eye disease: implications for immunotherapy. *Curr Opin Ophthalmol*. 2011 Sep;22(5):385-90. doi: 10.1097/ICU.0b013e3283499446. PMID: 21730841; PMCID: PMC3512192.
- 26- Stan MN, Garrity JA, Bahn RS. The evaluation and treatment of graves ophthalmopathy. *Med Clin North Am*. 2012 Mar;96(2):311-28. doi: 10.1016/j.mcna.2012.01.014. Epub 2012 Feb 22. PMID: 22443978; PMCID: PMC3898790.
- 27- Shan SJ, Douglas RS. The pathophysiology of thyroid eye disease. *J Neuroophthalmol*. 2014 Jun;34(2):177-85. doi: 10.1097/WNO.000000000000132. PMID: 24821101.

- 28- Papageorgiou KI, Hwang CJ, Chang SH, Jarullazada I, Chokron Garneau H, Ang MJ, King AJ, Mancini R, Douglas RS, Goldberg RA. Thyroid-associated periorbitopathy: eyebrow fat and soft tissue expansion in patients with thyroid-associated orbitopathy. *Arch Ophthalmol*. 2012 Mar;130(3):319-28. doi: 10.1001/archophthalmol.2011.1271. PMID: 22411661.
- 29- Barrio-Barrio J, Sabater AL, Bonet-Farriol E, Velázquez-Villoria Á, Galofré JC. Graves' Ophthalmopathy: VISA versus EUGOGO Classification, Assessment, and Management. *J Ophthalmol*. 2015;2015:249125. doi: 10.1155/2015/249125. Epub 2015 Aug 17. PMID: 26351570; PMCID: PMC4553342.
- 30- Antonelli A, Fallahi P, Elia G, Ragusa F, Paparo SR, Ruffilli I, Patrizio A, Gonnella D, Giusti C, Virili C, Centanni M. Graves' disease: Clinical manifestations, immune pathogenesis (cytokines and chemokines) and therapy. *Best Practice & Research Clinical Endocrinology & Metabolism*. 2020 Jan 1;34(1):101388.
- 31- Weiler DL. Thyroid eye disease: a review. *Clinical and experimental optometry*. 2017 Jan 1;100(1):20-5.
- 32- Li H, Wang T. The autoimmunity in Graves's disease. *Front Biosci (Landmark Ed)*. 2013 Jan 1;18:782-33-
- 33- Edith T, Starich GH, Mazzaferri EL. Sensitivity, specificity, and cost-effectiveness of the sensitive thyrotropin assay in the diagnosis of thyroid disease in ambulatory patients. *Archives of internal medicine*. 1989 Mar 1;149(3):526-32.
- 34- Furmaniak J, Sanders J, Miguel RN, Smith BR. Mechanisms of action of TSHR autoantibodies. *Hormone and Metabolic Research*. 2015 Sep;47(10):735-52.
- 35- Bradley EA. Graves ophthalmopathy. *Current Opinion in Ophthalmology*. 2001 Oct 1;12(5):347-51.
- 36- Ralls PW, Mayekawa DS, Lee KP, Colletti PM, Radin DR, Boswell WD, Halls JM. Color-flow Doppler sonography in Graves disease: "thyroid inferno". *American Journal of Roentgenology*. 1988 Apr 1;150(4):781-4.
- 37- Ruchała M, Szczepanek E. Thyroid ultrasound—a piece of cake? *Endokrynologia Polska*. 2010;61(3):330-44.
- 38- Dolman PJ. Evaluating Graves' orbitopathy. *Best Pract Res Clin Endocrinol Metabol* 2012; 26: 229–248.
- 39- Dolman PJ, Rootman J. VISA Classification for Graves orbitopathy. *Ophthal Plast Reconstruct Surg* 2006; 22: 319–324.
- 40- Menconi F, Marcocci C, Marino M. Diagnosis and classification of Graves' disease. *Autoimmun Rev* 2014; 13: 398–402.
- 41- Scruggs RT, Black EH. Thyroid eye disease with significant levator involvement and ptosis: a case report. *Ophthal Plast Reconstruct Surg* 2015; 31: e153-e154.
- 42- Bartalena L, Baldeschi L, Boboridis K et al. The 2016 European Thyroid Association/European Group on Graves' Orbitopathy Guidelines for the Management of Graves' Orbitopathy. *Eur Thyroid J* 2016; 5: 9–26.
- 43- Bhatti MT, Dutton JJ. Thyroid eye disease: therapy in the active phase. *J Neuroophthalmol* 2014; 34: 186–197.
- 44- Bartalena L. Diagnosis and management of Graves disease: a global overview. *Nat Rev Endocrinol* 2013; 9: 724–734.
- 45- Bartalena L. Diagnosis and management of Graves disease: a global overview. *Nat Rev Endocrinol* 2013; 9: 724–734.
- 46- Bartalena L, Marcocci C, Bogazzi F et al. Relation between therapy for hyperthyroidism and the course of Graves' ophthalmopathy. *New Engl J Med* 1998; 338: 73–78.
- 47- Thornton J, Kelly SP, Harrison RA et al. Cigarette smoking and thyroid eye disease: a systematic review. *Eye (Lond)* 2007; 21: 1135–1145.
- 48- Bartalena L. Prevention of Graves' ophthalmopathy. *Best Pract Res Clin Endocrinol Metab* 2012; 26: 371–379.

Fibromyalgia : A review

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Abstract

This paper reviews guidelines of fibromyalgia. Fibromyalgia is a common medical condition which is still misdiagnosed with other rheumatological disorders. It can be complex and brings many challenges. Presentation can vary from patient to patient. It is estimated that around up to 5% of population may have fibromyalgia with more cases in women. Although there is no cure for this condition, more understanding of Fibromyalgia can contribute to a well-rounded effective treatment and therapy options via a multidisciplinary approach to help in relieving the symptoms.

Keywords : fibromyalgia, challenges, multidisciplinary approach, therapy, rheumatological disorders.

Definitions and epidemiology

Fibromyalgia is a medical condition presenting with widespread musculoskeletal pain, tiredness, poor sleeping patterns and tenderness in different points in the body (1). It can be associated with other symptoms such as headaches, brain fog and increased sensitivity to sensations such as light, noise, temperature and touch (2). The cause is still unclear and undiagnosed with a high number of patients. There is no evidence of muscle inflammation and it is thought to be a disorder of pain processing (3). The prevalence of fibromyalgia is more in females than males and the percentage can be increased by age. The number of consultations of fibromyalgia is increasing over time due to increased public awareness (4).

Aetiology

There is still no clear aetiology found that can cause fibromyalgia but some theories suggest there could be factors contributing in developing fibromyalgia such as:

- Genetic factors: this can run in families which can increase risk of fibromyalgia (5).
- Change in pain perception and threshold: Abnormal pain processing in the nervous system and hypersensitivity. However, the mechanism is still not clear. It seems that pain in fibromyalgia can mimic neuropathic pain and analgesia can be ineffective (6).
- Poor sleeping patterns which can affect healing process of muscles.
- Patients with history of rheumatic disorders such as arthritis, rheumatoid arthritis and ankylosing spondylitis.
- Triggers such as physical injuries, infections and emotional trauma.
- Mental health illnesses such as depression.

Symptoms of fibromyalgia

(see Figure 1):

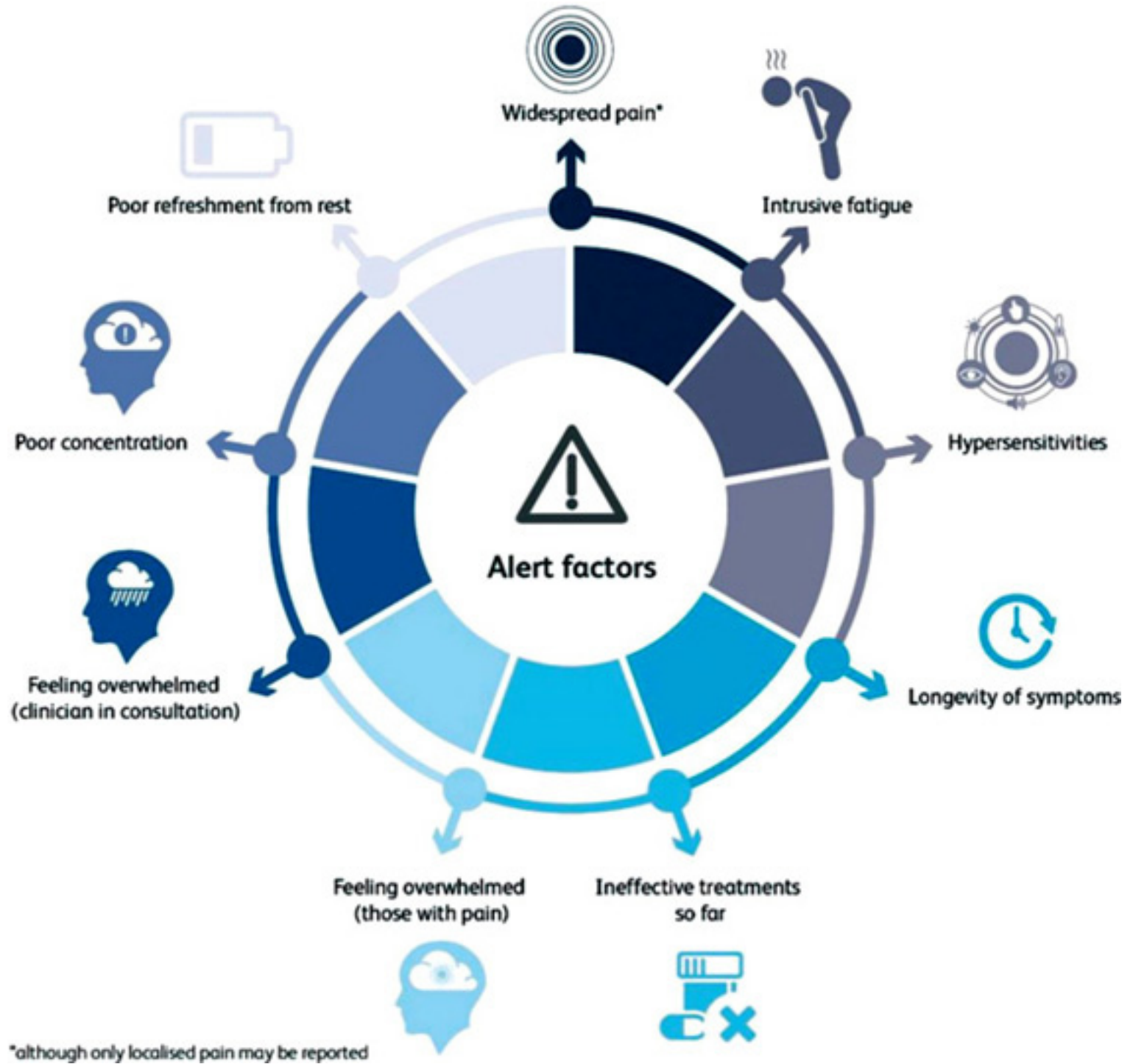
- Widespread pain which can vary in intensity. It can be worse in certain areas such as back and neck. It can flare up and get worse at various times.
- Fatigue.
- Poor sleeping pattern and may not feel refreshed after sleeping all night.
- Headache, dizziness, forgetfulness and poor concentration.
- Achiness and stiffness.
- Stress, anxiety and low mood.
- Constipation, diarrhoea or stomach cramps. It can be diagnosed as Irritable Bowel Syndrome IBS.
- Irritable or overactive bladder.

Diagnosis

Diagnosis of fibromyalgia remains challenging due to the absence of definitive biochemical markers or imaging studies (8). However, physicians rely on the patient's reported symptoms by taking a detailed medical history and clinical assessment. The subjective nature of pain perception can make the diagnostic process complicated. The second challenge is that fibromyalgia shares symptoms with other medical conditions such as hypothyroidism, ankylosing spondylitis, systemic lupus erythematosus SLE, rheumatoid arthritis, multiple sclerosis, sleep apnea, chronic fatigue syndrome, depression and medications (high dose opioids, statins, letrozole). The diagnostic approach involves ruling out other medical conditions via tests such as urine, blood tests includes (FBC, ESR, CRP, CK, LFT, TFT, Glucose, U&E), x-rays and other scans. The American College of Rheumatology (ACR) has set up a criteria for fibromyalgia diagnosis (See Figure 2). This criteria included identifying duration and sites of pain with associated symptoms then giving a total score to aid diagnosis. Patients play an important aspect of diagnostic approach. Effective communication with patients can help build a full picture. Also, a symptoms diary can be very effective in tracking patterns to make a diagnosis.

Fibromyalgia is a common condition in primary care. However, it may need a holistic approach and involve other specialties when there is doubt in diagnosis, such as rheumatologists, pain management team and neurologists.

Figure 1 – Symptoms of fibromyalgia (7)



Fibromyalgia syndrome diagnostic worksheet

Symptom severity index (SSI)

Have your problems with the symptoms below been present for 3 months or more?

 Yes

 No

If yes, using the following scale, indicate the severity of each symptom over the past week by circling the appropriate number.

	No problem	Mild	Moderate	Severe
Fatigue	0	1	2	3
Trouble thinking or remembering	0	1	2	3
Waking up tired (unrefreshed)	0	1	2	3

During the past 6 months, have you had any of the following symptoms?

Pain or cramps in lower abdomen

 Yes

 No

Depression

 Yes

 No

Headache

 Yes

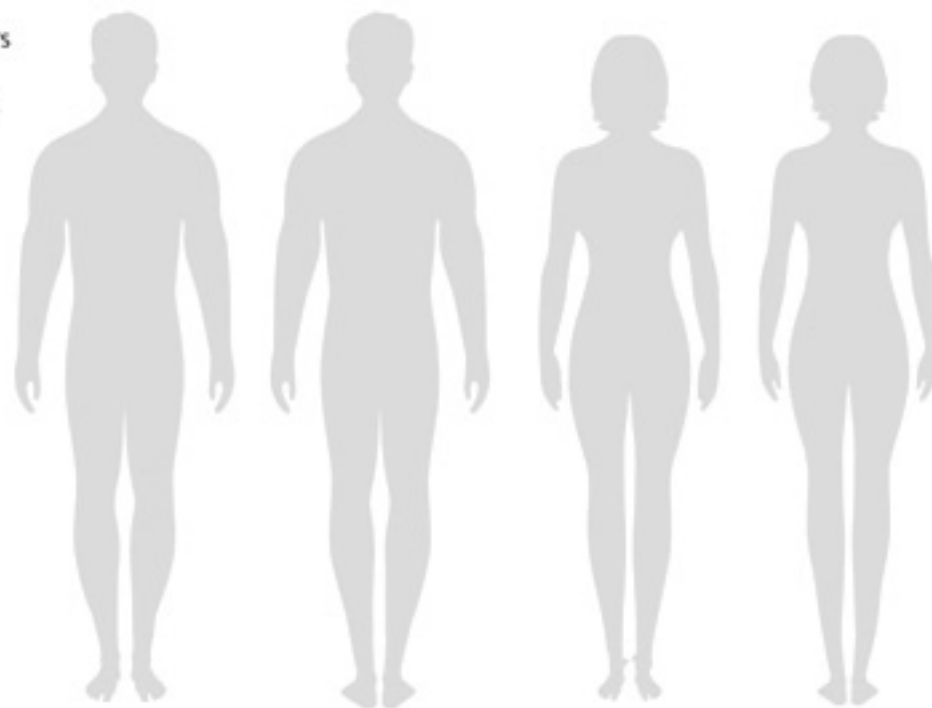
 No

Total score* for the SSI _____

*The sum of the three scaled symptoms plus one point each for the other symptoms (pain or cramps, depression, headache). The total will be between 0 and 12.

Body map

Use the figures to record where pain occurs in detail. Shade the areas of your body where you have felt persistent or recurrent pain for the past 3 months or longer (chronic pain).



Calculating the WPI score

Use this checklist to calculate the widespread pain index (WPI) score. Tick the areas where you have had chronic pain for 3 months or longer.

Region 1: left upper

- L jaw
- L shoulder girdle
- L upper arm
- L lower arm and/or L wrist/hand, L elbow

Region 2: right upper

- R jaw
- R shoulder girdle
- R upper arm
- R lower arm and/or R wrist/hand, R elbow

Region 3: left lower

- L hip and/or L buttock
- L upper leg and/or L groin
- L lower leg and/or L ankle/foot, L knee

Region 4: right lower

- R hip and/or R buttock
- R upper leg and/or R groin
- R lower leg and/or R ankle/foot, R knee

Region 5: axial

- Neck
- Upper back
- Lower back
- Chest (L and/or R)
- Abdomen

Total score* for the WPI _____

*The total will be between 0 and 19.

L=left; R=right

A diagnosis requires widespread pain > 3 months duration with currently either
 i) widespread pain index (WPI) ≥ 7 and symptom severity scale (SSS) score ≥ 5 , or
 ii) WPI 4–6 and SSS score ≥ 9 , with pain in 4/5 body regions.