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Possible relations between arterial hypertension and cervical spine fibromyalgias (literature review)

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Abstract: *The literature review analyses and systematises currently available information on fibromyalgia etiology and pathogenesis, hypertension classification and causes, and considers possible relationships between cervical fibromyalgia and hypertension. Statistical data on mortality in Ukraine related to cardiovascular diseases, including hypertension, are highlighted. A concise classification of arterial hypertension according to the International Classification of Diseases-10 (ICD-10) and the International Classification of Diseases-11 (ICD-11) is provided, and its pathogenesis (essential hypertension, resistance hypertension and hypervolemia) is outlined. Physiological relations between cervical spine and cardiovascular system are considered. The main diagnostic criteria for fibromyalgia classification and its development stages are described. Prevalence of fibromyalgia is identified and its forms are classified in accordance with ICD-11. The available data on the likely causes of fibromyalgia pain and any established mechanisms of fibromyalgia pathogenesis, including, but not limited to peripheral and central sensitisation, neurogenic inflammatory processes occurring in the disease-associated peripheral tissues, spinal cord and brain, as well as potential involvement of genetic, endocrine, psychopathological factors and sleep disorders in fibromyalgia development are discussed. The current formal criteria for fibromyalgia classification (revised in 2016), together with assessment of the main pharmacological and non-pharmacological fibromyalgia treatment methods and approaches based upon available published clinical trials outcomes are described. The main directions of fibromyalgia research are identified, and further prospective studies covering different relationship aspects between hypertension and cervical fibromyalgia are evaluated. Among the available literature sources, the only study of patients with comorbid fibromyalgia (fibromyalgia was diagnosed using 1990 American College of Rheumatology criteria and evaluated by Revised fibromyalgia impact questionnaire (FIQR)) and hypertension (blood pressure levels were assessed using the WHO protocol) has been reported. However, this group has only studied the dynamics of changes in cortisol, melatonin and serotonin blood levels, which accompany these pathologies, and these authors have found that fibromyalgia tender points' quantity and FIQR scores are much higher in hypertensive patients with fibromyalgia in comparison with the rest, which has led them to the conclusion that hypertension prevalence in patients with fibromyalgia could be related to fibromyalgia*

severity. Thus, this thorough literature review demonstrates that clinical studies examining possible relationships between hypertension and cervical spine fibromyalgia are at lack and require further considerations.

Key words: [cardiovascular system](#), [cervical vertebrae](#), [comorbidity](#), [fibromyalgia](#), [hypertension](#), [international classification of diseases](#), [pain](#).

Introduction

According to the official Ukrainian Ministry of Health statistics, every third adult in Ukraine suffers from hypertension. Every year, over a million people are diagnosed with hypertension among patients assessed in medical institutions for the first time. High blood pressure (BP) is the main cause of atherosclerosis, the key cardiovascular risk factor which significantly (3-4 times) increases incidence of coronary heart disease (CHD) and stroke, heart and kidney failure, as well as elevates overall occurrence of various cerebrovascular disorders by 7 times. When analysing the total mortality structure of the Ukrainian population, it becomes apparent that 66.3% of deaths result from circulatory system diseases. Therefore, reduction of arterial hypertension is the vital factor in lowering cardiovascular disease risks and improving the overall population health (Karel et al., 2019). Treatment of cervical spine pathologies can improve hypertension course (Zharova and Shevtcova, 2011), so it is reasonable to assume that hypertension development and progression might be associated with cervical spine disorders, in particular with neck fibromyalgia.

Aim

Evaluate possible links between hypertension and cervical spine fibromyalgia by assessing all relevant published research data.

Methods

Theoretical analysis and synthesis of modern scientific literature sources

Results and discussion

According to the "Health Index Ukraine – 2019" national study, more than 80% of all deaths in Ukraine are due to cardiovascular disease, diabetes, cancer, chronic obstructive pulmonary disease, and mental health disorders (Barska et al., 2020). Nationwide, cardiovascular mortality has increased by almost 8% in 29 years, from 350,605 deaths in 1990 (56.5% of total deaths) to 449,376 deaths (64.3% of total deaths) in 2019 despite

the overall reduction in population headcount. In 2020, taking into account the COVID-19 pandemic, it amounted to 408,721 deaths (33.13% of the total number of deaths) (State Statistics Service of Ukraine, 2020).

1. Aetiology, pathophysiology and epidemiology of hypertension

Today, hypertension is defined worldwide as a social problem that affects public health and life expectancy. According to the current classification (Abanto and Abanto, 2020), the diagnosis of hypertension is established in the case of persistently elevated systolic blood pressure (SBP) up to 140 mm Hg, and / or diastolic blood pressure (DBP) up to 90 mm Hg. Moreover, there are essential hypertension (hypertension disease or primary hypertension) – high blood pressure in absence of an obvious disease cause, and secondary (symptomatic) hypertension the cause of which can be clearly identified. In May 2020, the hypertension classification was updated. According to the updated version, blood pressure is considered normal if it is within the SBP <130 – DBP <85 mm Hg range; high-normal – SBP 130-139 – DBP 85-89 mm Hg; increase of SBP to 140-159 mm Hg and DBP to 90-99 mm Hg indicates I degree hypertension; SBP \geq 160 and DBP \geq 100 mm Hg – II degree hypertension (Unger et al., 2020).

In Ukraine, the 10th revision (ICD-10) of the International Classification of Diseases has been used for disease statistics coding since the 1st of January, 1999 (Ukrainian Ministry of Health, 1998; WHO, 2004). According to this classification, circulatory system disorders are classified as class IX. Diseases characterised by high blood pressure have codes I10-I15. The following concepts / clarifications of hypertension are included in the I10 section: hypertension, essential, primary, systemic, benign, malignant hypertension.

Secondary (symptomatic) hypertension is classified under the I15 section. Renovascular

hypertension has the I15.0 code; hypertension of nephrogenic origin (associated with other kidney diseases, such as pyelonephritis, glomerulonephritis, etc.) – I15.1; hypertension of endocrine genesis (due to endocrine disorders) – I15.2; secondary hypertension of other origin (for example, haemodynamic – in aortic coarctation, aortic valve insufficiency; neurogenic – in diseases or injuries of the brain or spinal cord; exogenous – in lead, thallium poisoning, etc.) – I15.8; secondary hypertension is unspecified and has I15.9 code (Association of Cardiologists of Ukraine, 2017; WHO, 2004).

There are 3 generalised links in hypertension pathogenesis: emission hypertension, resistance hypertension and hypervolemia. Thus, at the beginning of hypertension development increased SBP, tachycardia and autonomic dysfunction are prevalent, suggesting that the increased heart rate – ejection hypertension plays the key role. As the disease progresses, SAP increase becomes accompanied by that of a DBP, i.e. in the arteries and arterioles tone, which is expressed by an increase in total peripheral vascular resistance (TPVR) and pulmonary vascular resistance (PVR) pointing at the hypertension of resistance. At the same time, the body increases production of antidiuretic hormone (ADH) causing Na^+ and fluid retention which leads to further CBP increase in some patients, and steady increase in DBP in other patients, or simultaneous elevation of both CBP and DBP in other patient groups. In rare cases, patients' blood pressure levels are almost unchanged. Eventually, heart failure (HF) begins to progress as hypervolemic hypertension develops (Berezyn, 2017).

2. Aetiology, pathophysiology and epidemiology of neck fibromyalgia

One of the most common cervical spine pathologies is fibromyalgia (FM): a complex chronic pain condition characterised by widespread pain, fatigue, sleep disturbances, cognitive problems, anxiety, depression, and loss of functional productivity reaching 75%. FM is also characterised by chronic diffuse generalised musculoskeletal pain, muscle tension, paresthesias, sleep disorders and general weakness, with the appearance of multiple painful points symmetrically spread throughout the body (Casale et al., 2019).

FM syndrome was first described back in the XIX century. Initially, this pathological condition was referred to as fibrositis or “psychogenic rheumatism”, as it was considered a psychosomatic disease. In 1972, formal diagnostic criteria for FM were proposed by H. Smythe (Smythe, 1979) for the first time. Later on (1990), F. Wolfe, H. Smythe and M. Yunus et al. have developed the first diagnostic criteria for FM adopted by the American College of Rheumatology (ACR) (Arnett et al., 2019; Krakov et al., 2021; Wolfe et al., 1990), which had two revisions suggested by F. Wolfe in 2010-2011 (Wolfe et al., 2011) and in 2016 (Wolfe et al., 2016), and continue to attract scientists attention. Also, Burckhardt et al. have designed a patient questionnaire for activity and mental functions disorders assessment (Fibromyalgia Impact Questionnaire) (Burckhardt et al., 1991).

According to recent epidemiological studies, the prevalence of FM is from 2 to 8% of the total population. Despite high FM prevalence, its aetiology and pathophysiological mechanisms are still not fully understood. The overall prevalence of FM in the United States is from 6 to 15%, and it is five times higher among females (Paschali et al., 2021). In Ukraine, FM is rarely diagnosed, which may be due to identified difficulties in separating FM and non-FM patients.

In the 11th revision of the International Classification of Diseases, FM is defined as a syndrome that, according to the criteria published by the ACR in 2010 (ICD-11, 2020), can be both primary (in the absence of any associated disorders) and secondary (associated with rheumatic or other organic diseases) while having the same course. However, several authors argue that the question of primary and secondary FM equivalence is no longer valid since FM has established associations with numerous disorders and most patients with “primary” FM have diagnosed comorbidities (Bennett and Friend, 2019).

The aetiology and pathogenesis of FM are complex and not fully known. Most experts agree that FM is a syndrome of central pain hypersensitivity due to impaired control and perception of pain in the central nervous system (Giral et al., 2021). It is believed that FM pain usually occurs spontaneously but can result from a number of

reasons. Such causes include genetic, environmental, hormonal, neural and immunological, as well as certain infections such as Epstein-Barr virus, parvovirus, brucellosis and Lyme disease, although for the vast majority of patients no specific cause is found (Borg-Stein et al., 2020).

According to the current analytical FM pathogenesis reviews (Sarzi-Puttini et al., 2020), its appearance can be explained by the following possible mechanisms: peripheral and central sensitization (hyperalgesia and/or allodynia, indicating issues with pain amplification or sensory processing in the CNS with neurotrophic brain factor, p-CREB protein, substance P, and possible nerve growth factor involvement, as well as hypersensitivity to various external stimuli, including sound or light), neurogenic inflammatory processes occurring in peripheral tissues, spinal cord and brain (with release of chemokines and cytokines, including IL-6, IL-8, IL-1 β , TNF- α , antinuclear antibodies), leading to edema and dysesthesia, cognitive impairment and fatigue, and emotional distress; numerous clinical studies conducted in FM patients indicated that small unmyelinated C fibres and myelinated A fibres are involved in peripheral neuroinflammation, and increase in nociceptive activity in muscles and other tissues after neurogenic inflammation may further contribute to central sensitisation (O'Mahony et al., 2021). The potential involvement of genetic factors in FM emergence relates to about 100 genes that regulate pain and are important for pain sensitivity or analgesia, in particular it applies to genes encoding potential-dependent sodium channels, GABA-ergic signalling pathways, mu-opioid receptors, catechol-O-methyltransferase (COMT) and GTP cyclohydrolase 1, serotonin transporter gene (SLC6A4), and the vanillin transitory receptor potential gene (TRPV2) which are responsible for pain perception in FM (Polli et al., 2020). The endocrine FM factors involve the hypothalamic-pituitary-adrenal axis as being central to the stress response, in particular including cortisol levels, their circadian variation and increased cortisol secretion in response to adrenocorticotrophic hormone which were studied in FM patients' blood plasma; the relationship between corticotropin-releasing factor levels in cerebrospinal fluid, heart rate variabil-

ity, and pain-related symptoms (e.g., fatigue and depression); low levels of growth hormone and insulin-like growth factor 1 were also established in patients with FM (Lightman et al., 2020). Finally, psychopathological factors and sleep disorders also play a distinct role: the presence of concomitant mental illnesses such as anxiety and depression among patients with FM reached 60% in certain subpopulations, depressive patterns correlated with reports of greater pain severity and duration, as well as greater degree of hyperalgesia / allodynia. Serotonin and norepinephrine imbalance, and sleep deprivation in FM patients have been shown to promote hyperalgesia and increase spontaneous changes in pain and mood, especially in relation to anxiety and depression (Broadbent et al., 2021).

According to the current criteria of the ACR classification after the 2016 revision (Wolfe et al., 2016), the diagnosis of FM is established if the following three conditions are present: 1) widespread pain index (WPI) ≥ 7 and symptom severity index (symptom severity scale – SSS) ≥ 5 or WPI 3-6 and SSS ≥ 9 ; 2) generalised pain, defined as pain in at least 4 of the 5 parts of the body (excluding the lower jaw, chest and abdomen); 3) symptoms of such severity persist for at least ≥ 3 months; 4) the FM diagnosis is valid regardless of other diagnoses and FM the diagnosis does not exclude the presence of other clinically important diseases. FM is determined by WPI and SSS. When determining WPI, the number of body areas in which the patient had pain during the last week (from 0 to 19) is recorded: left upper area (Region 1) includes the left lower jaw, left shoulder girdle, left shoulder, left forearm; right upper part (Region 2) – right part of the lower jaw, right part of the shoulder girdle, right shoulder, right forearm; lower left area (Region 3) – left pelvis (buttocks or large swivel), left thigh, left leg; right lower area (Region 4) – right pelvis (buttocks or large swivel), right thigh, right leg; axial area – neck, upper back, lower back, chest, abdomen. When determining the SSS on a tribal scale (0 = no violations, 1 = minor or mild disorders, mostly mild, or occurring periodically, 2 = moderate significant disorders, frequent and / or moderate, 3 = severe disorders that significantly pronounced, persistent, aggravating) the severity

of fatigue, restlessness, and cognitive symptoms during the last week, as well as the presence / absence of a headache (0-1), pain or spasms in the epigastric region (0-1), and depression (0-1) during the last 6 months are assessed. The final SSS result (0 to 12) is calculated as the sum of the 3 symptoms severity (fatigue, awakening without rest, cognitive symptoms) (0-9), and three other symptoms (headache, pain or cramps in the epigastric region and depression) (0-3). The sum of WPI and SSS is the fibromyalgia severity index (FS) (Wolfe et al., 2016). Also, a short patient questionnaire (6 questions) for FM screening (FiRST) was developed (Fan et al., 2016).

Many factors contribute to FM development in a unique way: genetic predisposition, personal experience, emotional and cognitive factors, the relationship between mind and body, and biopsychological ability to cope with stress. In this sense, FM can be considered as a state of brain/mind and body desynchronization (Perrot, 2019). Therefore, FM treatment should be holistic and comprehensive, being characterised by integration and multidisciplinary interventions (Giusti et al., 2017), and include not only pharmacological intervention, but also patient education, good health keeping, and psychotherapy (Aksoy et al., 2021). The revised European League Against Rheumatism (EULAR) guidelines for the FM treatment have assessed the main pharmacological and non-pharmacological treatments for FM based on available published clinical trials. Among the pharmacological agents considered were amitriptyline (“weak for”), anti-convulsants (pregabalin – “weak for”, gabapentin – only study), cyclobenzaprine (“weak for”), growth hormone (“strong against”), monoamine oxidase inhibitors (“weak against”), nonsteroidal anti-inflammatory drugs (“weak against”), serotonin-norepinephrine reuptake inhibitors (duloxetine and milnacipran – “weak for”), selective serotonin reuptake inhibitors (“weak against”), sodium oxybutyrate (“strong against”), tramadol (“weak for”), strong opioids and corticosteroids (“strong against”). Among the non-pharmacological agents considered were acupuncture (“weak for”), biological feedback (“weak against”), capsaicin (“weak against”), chiropractics (“strong

against”), cognitive-behavioural psychotherapy (“weak for”), physical exercise (“strong for”), hydro/spa therapy (“weak for”), hypnotherapy (“weak against”), massage (“weak against”), meditative movement (“weak for”), invaluable conscious observation/psychosomatic therapy (“weak for”), multicomponent therapy (“weak for”), s-adenosylmethionine (“weak against”), other additional and alternative therapies (controlled mental images, homoeopathy – “strong against”).

Modern scientific studies consider the need for immediate pharmacological treatment initiation, antidepressants and structural GABA analogues including, as most diagnoses of FM are established years after the symptoms onset (Pearson et al., 2020). Standard FM treatments also include antioxidants and vitamins that alter FM symptoms severity (Miranda-Díaz and Rodríguez-Lara, 2017). Certain groups of bioactive compounds and derivatives from medicinal plants have also demonstrated usefulness in FM due to their analgesic activity and antioxidant properties (Bhardwaj et al., 2021; Siqueira-Lima et al., 2019).

Thus, the complexity of its course, significant impact on quality of life, and lack of clearly defined aetiology and pathogenesis make FM an attractive disease for further research, as numerous studies available (Bağcıer et al., 2021) provide only general ideas in regard to FM pathophysiology. Oxidative stress, mitochondrial dysfunction, multivitamin deficiency, and a mismatch between oxidants and antioxidants are typically cited in relation to FM status, development, and course. The lack of specific objective tests or biomarkers with sufficient diagnostic accuracy, as well as availability and limitations of the current tests which can only indicate basic predisposition to FM encourage scientists to consider novel proteomics studies and gene expression analysis. Pharmacological treatment on its own is insufficient for most FM syndrome patients. Given the different pain sensitivity mechanisms, its treatment should include multidisciplinary programs (Illescas-Montes et al., 2021; Pătru et al., 2021) focusing on peripheral, central, cognitive-emotional and interpersonal causes of chronic pain which characterises the FM pathophysiology.

3. *The relationship between hypertension and neck fibromyalgia*

In the hypertension course, cerebrovascular disorders and negative cerebrospinal fluid dynamics which provoke and aggravate the disease, and can also lead to serious complications such as heart attacks and strokes leading to mortality or disability are commonly observed. Recent studies indicate that amongst the 40-59 years old adult group more than 70% of patients with hypertension have musculoskeletal system and connective tissue pathologies (Sevostyanova et al., 2019). Hypertension formation may be associated with cervical spine pathology, including neck fibromyalgia. According to the WHO, back pain of vertebrogenic origin is prevalent at different ages in 70-80% of the population reaching 90-95% in the elderly (Zharova and Shevtcova, 2011). There are close physiological links between the cervical spine and cardiovascular system: the spinal sympathetic heart innervation centres are at the 7th cervical to 5-6th thoracic vertebrae level, the same segments also host sympathetic innervation centres of the head, neck, and chest. The nerves extending from the 3rd cervical and 5-6th upper thoracic sympathetic ganglia are directly involved in heart innervation. The sympathetic nervous system's ability to broadly irradiate and generalise excitation in cervical lesions causes dysfunction of the centres that regulate blood pressure (Korchynskyi and Ponomarenko, 2018).

While hypertension in cervical spine pathology is considered a transient reaction, which is a part of the "vertebral artery" or "vertebrobasilar insufficiency" syndrome (Vikulova et al., 2020), degenerative cervical spine changes eventually lead to brachiocephalic arteries deformity causing brain blood supply deterioration. Therefore, hypertension in such patients develops due to a compensatory increase in blood pressure in response to progressing brain hypoxia. Clinical studies have shown the presence of widespread cerebral blood flow dysfunction in hypertension, especially in patients not receiving any antihypertensive treatment or with inadequate antihypertensive therapy applied (Ripp and Rebrova, 2021). On the other hand, analysis of 509 patients with FM revealed that 187 of them

(36.7%) were diagnosed with cardiovascular disease (Bilge et al., 2017); in a different study in 941 patients aged 18-87 years (with an established FM diagnosis) circulatory disorders were revealed and observed in the surveyed age groups as follows: 18-29 years old – 28 people (3%), 30-39 years old – 39 people (4.2%), 40-49 years old – 92 people (9.8%), 50-59 years old – 101 people (10.8%), 60-69 years old – 33 people (3.5%), 70-87 years old – 6 people (0.6%), which indicates predominance of such disorders in adults suffering from FM (Häuser et al., 2019). Chronic headache in FM has also been reported (Kleykamp et al., 2021), with suggestions that FM may be a manifestation of idiopathic intracranial hypertension (Altaraqji et al., 2021). Possible pathophysiological links between idiopathic intracranial hypertension, fibromyalgia, and chronic fatigue syndrome were discussed by Hulens et al. (2018). Moreover, animal experiments demonstrated that angiotensin I inhibitors can cause FM-like symptoms in mice (Brusco et al., 2021).

Among all publicly available literature sources, we have found only one dedicated study of patients with FM (FM was diagnosed using 1990 American College of Rheumatology criteria and evaluated by Revised fibromyalgia impact questionnaire (FIQR)) and arterial hypertension comorbidity (blood pressure levels were assessed using the WHO protocol) (Ghizal et al., 2018), however it was limited to assessing the levels of cortisol, melatonin and serotonin in these patients, and these authors have found that fibromyalgia tender points' quantity and FIQR scores are much higher in hypertensive patients with fibromyalgia in comparison with the rest, which has led them to the conclusion that hypertension prevalence in patients with fibromyalgia could be related to fibromyalgia severity.

Conclusion

All assessed scientific publications have only contained indirect indications of a possible positive relationship between FM and arterial hypertension. Despite these studies findings, there is no established proof of a direct clinical and pathophysiological relationship between these pathologies yet, even though such comorbidity is common. Further dedicated scientific research

and clinical studies of links between FM and arterial hypertension in comorbid patients of different gender and age groups are clearly needed.

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Consent to publication

All authors have read and approved the final version of the manuscript. All authors have agreed to publish this manuscript.

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Можливі взаємозв'язки між артеріальною гіпертензією та фіброміалгіями шийного відділу хребта (огляд літератури)

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Анотація: В літературному огляді проаналізовано і систематизовано наявні сучасні відомості щодо етіології і патогенезу фіброміалгій, класифікації і причини виникнення артеріальної гіпертензії і розглянуто можливі взаємозв'язки фіброміалгій шийного відділу хребта з артеріальною гіпертензією. Виокремлено статистичні відомості щодо смертності в Україні, пов'язаної із серцево-судинними захворюваннями, зокрема артеріальною гіпертензією. Надано стислу класифікацію артеріальної гіпертензії відповідно до МКБ-10 і МКБ-11 і охарактеризовано узагальнені ланки її патогенезу (гіпертонію викиду, гіпертонію опору і гіперволемію). Розглянуто фізіологічні зв'язки між шийним відділом хребта і серцево-судинною системою. Описано основні діагностичні критерії класифікації фіброміалгій і стадії їх розробки. Відповідно до сучасних досліджень виділено ступінь поширеності фіброміалгій і надано їй класифікацію відповідно до МКБ-11, а також наявні відомості щодо можливих причин болю за фіброміалгій і механізмів формування фіброміалгії як такої, серед яких розглядаються периферична і центральна сенситизація, нейрогенні запальні процеси, що протікають у периферичних тканинах, спинному та головному мозку, потенційна участь генетичних факторів в появі фіброміалгій, ендокринні фактори, психопатологічні фактори і порушення сну. Надано сучасні критерії класифікації фіброміалгій перегляду 2016 р., а також оцінку основних фармакологічних і нефармакологічних методів лікування фіброміалгій на підставі наявних опублікованих клінічних досліджень. Також виокремлено основні напрями досліджень фіброміалгій і проведено оцінку подальших перспективних досліджень стосовно взаємозв'язків між артеріальною гіпертензією і фіброміалгіями шийного відділу хребта. Серед доступних літературних джерел було знайдено єдине дослідження стану пацієнтів при коморбідності фіброміалгій (фіброміалгії діагностувалися з використанням критеріїв Американського коледжу ревматології 1990 р. і оцінювалися за допомогою переглянутого опитувальника впливу фіброміалгій) і артеріальної гіпертензії (рівні артеріального тиску оцінювались за протоколом ВООЗ), однак в ньому вивчалися лише рівні кортизолу, мелатоніну і серотоніну при цих патологіях, але автори виявили, що кількість болючих точок і балів за опитувальником значно вищі у осіб з артеріальною гіпертензією і фіброміалгією у порівнянні з іншими хворими, що привело їх до висновку, що переважає артеріальної гіпертензії у пацієнтів з фіброміалгією може бути пов'язане зі ступенем вираженості фіброміалгії. Таким чином, ретельний науковий пошук показав, що досліджень, в яких розглядаються взаємозв'язки між артеріальною гіпертензією і фіброміалгіями шийного відділу хребта, відсутні і потребують подальшого розгляду.

Ключові слова: біль, гіпертензія, коморбідність, Міжнародна класифікація хвороб, серцево-судинна система, фіброміалгія, шийний хребець



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