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PERIPHERAL NEUROPATHIES IN PATIENTS WITH CHRONIC KIDNEY DISEASE

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Patients with chronic kidney disease often suffer from peripheral neuropathies, which have a significant impact on morbidity and mortality. Therefore, it is important to understand their pathophysiological mechanism to enable an effective management strategy for these neurological complications. This review aimed to highlight the importance of detecting clinical parameters that enable the diagnosis and early treatment of peripheral neuropathy (MRI).

To achieve the goal of this review paper, we used PubMed and MEDLINE databases. The information presented in this work is gathered from the literature with the keywords peripheral neuropathy, chronic renal failure, hemodialysis, clinical picture, and treatment. At the beginning of 2024, we conducted research in the PubMed and Medline databases.

Based on the established criteria for searching the literature related to peripheral neuropathy in patients with end-stage renal disease, in the last ten years, as well as some earlier works that were of interest to the issue of peripheral neuropathy, out of a total of 22 works that met the inclusion criteria, there were 8 review works, 9 original works, 4 case reports, and 1 monograph.

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Key words: *peripheral neuropathy, chronic renal failure, hemodialysis, clinical picture, treatment*

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Introduction

The prevalence of chronic kidney disease is increasing worldwide. About 850 million people in the world have kidney failure, which is currently the third leading cause of death. Neurological complications associated with renal insufficiency significantly increase morbidity, mortality and decrease the quality of life of patients with renal insufficiency, and among them, peripheral neuropathy occupies a significant place. Peripheral neuropathy, one of the commonest neurological complications of chronic kidney disease, its frequency among the population of patients with chronic renal failure, in the terminal stage, is between 60–100% (1, 2).

Methodology

At the beginning of 2024, we conducted research in the PubMed and Medline databases. The criteria for including studies in the research were keywords: peripheral neuropathy, chronic renal failure, hemodialysis, clinical picture, and treatment.

The literature search covered the period of the last ten years, except for some works that we included in the reference literature from an earlier period, which were significant for the assessment of the problem of peripheral neuropathy in hemodialysis patients (Figure 1).

All works with these keywords were reviewed and selected to remove duplicates. Selected publications are organized and summarized for each form of peripheral neuropathies separately with a general description of study characteristics and findings organized by subtopics derived from the data. Of the selected publications, the largest number were original and review works, a smaller part was monographs. Most of the selected papers were written in

English, except the monograph, which was written in Serbian.

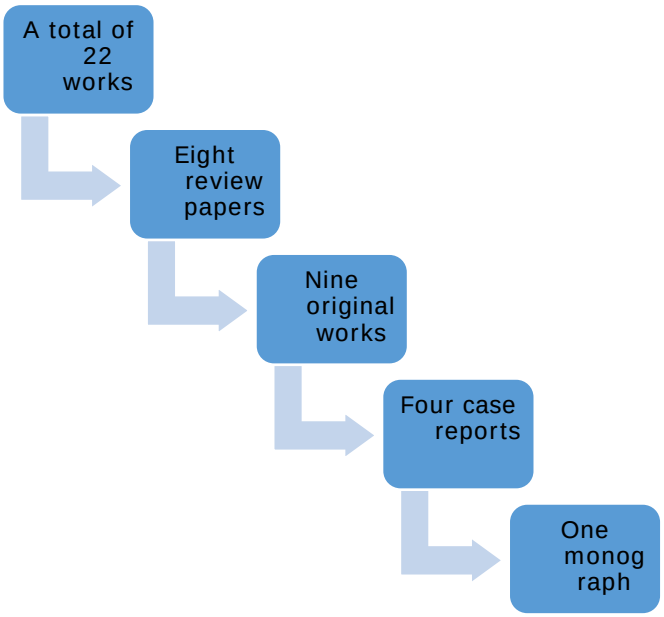

Figure 1. Selection of articles included in the work

Results

Based on the established criteria for searching the literature related to peripheral neuropathy in patients with end-stage renal disease, in the last ten years, as well as some earlier works that were of interest to the issue of peripheral neuropathy, out of a total of 22 works that met the inclusion criteria, there were 8 review works, 9 original works, 4 case reports, and 1 monograph.

Peripheral neuropathies

Current data indicate that the prevalence of neuropathy in patients with end-stage renal failure is between 60% and 100% and that it is clinically manifested when the glomerular filtration rate falls below 12 ml/min. The systemic effects of uremia on the peripheral nerves are emphasized by the generalized nature of the slowing of nerve conduction in the extremities and sensorimotor nerves. Structural changes that occur in peripheral neuropathy depend on the duration of uremia, which in itself can have a universal neurotoxic effect. Creatinine, urea, methylguanidine, phenols, guanidine, uric acid, aromatic hydroxy acids, oxalic acid, guanidinosuccinic acid, myoinositol, indican, amines, "intermediate molecules", parathormone, β-2 microglobulin have shown their negative influence on peripheral nerve endings, most often on median, ulnar and femoral nerves. In the pre-dialysis period, that is, in the pre-terminal phase of chronic renal failure, the nerve endings are in a depolarized state, but the membrane potential is normalized after dialysis. It must be emphasized that the degree of depolarization is correlated with the concentration of potassium, which indicates that hyperkalemic depolarization has a significant role in the development of nerve dysfunction (3).

Peripheral neuropathy caused by chronic renal failure results from various mechanisms. Intracellular calcium accumulation due to hyperkalemia in chronic renal failure and reversal of the K/Ca pump can cause axonal damage and significant axonal depolarization in the early stages of neuropathy. Other factors causing neuropathy in patients with chronic renal failure include the neurotoxic effects of small molecules (eg, myoinositol and methyl guanidine) that accumulate in body fluids, elevated parathyroid hormone levels, and vitamin B1 deficiency. Likewise, many drugs used to treat chronic renal failure can cause peripheral neuropathy (4).

In the early stages of uremic neuropathy, there is a distal loss of needle prick sensation for fistula puncture, as well as a reduction or absence of tendon reflexes in the lower limbs. As the disease progresses, this sensory loss spreads proximally in the lower limb, and then when the motor nerves are damaged, weakness and atrophy may occur in the distal muscles of the lower limb. In addition to the damage to large motor and sensory fibers in uremic neuropathy, neuropathy of small fibers also occurs. In patients with diabetic nephropathy, small fiber neuropathy can lead to clinical manifestations of burning and sharp pain, as well as changes in temperature perception. Advanced uremic neuropathy can be clinically presented by atrophy of the musculature of the distal part of the lower extremities with the appearance of ulcerations and consequent limb amputations (5).

Polyneuropathy, in patients with chronic renal failure, can be a consequence of glomerulonephritis, diabetes, hypertension, as well as multisystem accompanying disorders. Signs and symptoms of uremic polyneuropathy begin distally in the feet, slowly progress along the legs and are symmetrical, also sensory disturbances manifesting as cramps, pain and burning, and restless legs syndrome, while a smaller number of patients report discomfort, pain and hyperalgesia in the feet. Uremic polyneuropathy continues to significantly limit the quality of life of patients with uremia. It must be emphasized that the symptoms of damage to small-diameter nerve fibers are manifested by pain, hyperalgesia, and dysesthesia, while damage to large-diameter fibers is clinically manifested by paresthesias and numbness (6, 7).

The accumulation of uremic toxins associated with oxidative stress-related free radical activity causes motor, sensory and autonomic nerve damage which leads to peripheric neuropathy. Although this exact mechanism remains unknown, hypotheses are supporting the role of electrolytes in this process. Hyperkalemia and hyperphosphatemia cause chronic uremic depolarization of nerves, contributing to the development of peripheric neuropathy. This occurs because potassium disrupts the normal ionic gradient and therefore activates calcium-mediated processes leading to axonal death (5, 8).

Clinical diagnosis of peripheral neuropathies, in patients with chronic kidney diseases, implies, in addition to eliminating other causes of neuropathy (glucose metabolism disorder and connective tissue disease), involves a neurological examination and a nerve conduction test, by which it is determined conduction velocity and amplitude and confirms axonal neuropathy. It should be known that in the initial stages of the disease, axonal neuropathy is characterized by reduced sensory, and in the later stages of the disease, motor amplitudes are reduced, while conduction velocity is minimally impaired (5).

Hemodiafiltration, as a dialysis procedure in the treatment of patients with end-stage kidney diseases, can have a positive effect on nerve function. Nevertheless, there is ample evidence that the most effective treatment for peripheral neuropathy in patients with end-stage kidney disease is kidney transplantation. Unfortunately, some drugs used after a kidney transplant, such as calcineurin inhibitors, can contribute to nerve damage. Likewise, it should be emphasized that maintaining normal serum potassium homeostasis can have a positive effect on the prevention of peripheral nerve damage, which can be a potential strategy for the prevention and treatment of neuropathy in chronic kidney disease. Interesting are the findings of recent studies that confirmed the potential benefit of limiting potassium intake in preventing the progression of neuropathy in stage 3−4 chronic renal failure. Drugs used against neuropathic pain belong to the group of tricyclic antidepressants and anticonvulsants and they are avoided due to numerous side effects, although they belong to the first line of drugs, of which amitriptyline is the best known. Also, extra care is needed when using anticonvulsant drugs such as pregabalin or gabapentin, because the doses are limited and they are dependent on creatinine clearance. There is some research that emphasizes the importance of an exercise program for improving neuromuscular function. Despite the significant share and breadth of the therapeutic range, apart from the control of potential risk factors, unfortunately, there is no specific treatment for uremic myopathy (5, 9, 10).

Ischemic neuropathy

Ischemic neuropathy is the most common clinical manifestation among diabetics who have serious damage to peripheral arteries, especially when the brachial artery is used to create an arteriovenous fistula immediately after arteriovenous anastomosis (usually within a few hours). Clinically, it is manifested by severe pain, weakness in the hands and paresthesia. A neurological examination shows weakness of the distal muscles innervated by the median nerve. The incidence of ischemic neuropathy ranges between 1 and 10% (11). The mechanism of ischemic neuropathy is not clear enough, but it is known that it is a form of the phenomenon of blood theft after the creation of an arteriovenous anastomosis, which contributes to the loss of axons in the distal part of the limb. Ischemic monomyelopathy in the lower extremities is more sensitive to ischemic monomyelopathy due to greater loss of axons, compared to the ulnar nerve. In these circumstances of arteriovenous anastomosis, there is an inversion of a small amount of blood from the distal blood vessels into the arteriovenous fistula or arteriovenous graft, which is sufficient for the consequent ischemia and the appearance of ischemic neuropathy, most likely due to the lack of collateral blood flow (12, 13). Ischemic monomyelopathy involves dysfunction of multiple peripheral nerves of the upper extremities. Symptoms are usually immediate, predominantly neurological, and often without significant ischemia of the hand. There is also paresthesia and numbness in the area of innervation of all three nerves of the forearm, as well as diffuse motor weakness or paralysis. The main diagnostic feature of ischemic monomyelopathy is the lack of findings indicating reduction or redirection of arterial flow. One of the factors that creates a dilemma in the setting of diagnostic criteria is the high percentage of diabetics in the population of patients with chronic kidney disease (14, 15).

When the neurological examination is not enough to make a diagnosis, an electrodiagnostic test can be of clinical importance. Dominant, clinically significant signs of ischemic monomyelopathy are sensorimotor symptoms in

the area of the hands or feet, in the sense of unpleasant, deep and burning pain, which lasts continuously for months. The expressiveness of the sensory, in relation to the physical findings, can introduce confusion and lead the clinician to think about a psychogenic or radiculopathic etiology. Clinical manifestations of motor symptoms manifest as weakness or paralysis of the muscles innervated by the three forearm nerves and include impairment of wrist and finger extension (radial nerve), ulnar abduction and adduction of extended fingers (ulnar nerve), and thumb opposition, flexion, and abduction (medial nerve). Ischemic monomyelopathy is a consequence of reduced blood flow in the nerve trunk, and due to the extreme sensitivity of nerve tissue to ischemia, there may be a significant risk of permanent damage. It has been described in the literature that, regardless of the establishment of radial pulses, when the blood flow is reduced 6 weeks after the creation of an arteriovenous fistula, there is no neurological improvement. From the meager literature data, it can be concluded that ischemic neuropathy can be considered a severe, focal neurological consequence of arterial theft syndrome. Electromyographic nerve conduction studies indicate the loss of motor and sensory nerve axons in the distal part of the limb and a decrease in the amplitude of nerve conduction, especially of sensory fibers in the forearm and hand (15, 16).

The therapeutic approach to the treatment of ischemic monomyelopathy involves reducing the flow in the vascular access, which will consequently lead to an increase in perfusion in the extremities and thus reduce the symptoms of neuropathy. Reduction of blood flow or complete shutdown improves blood flow to the extremities, which can lead to recovery, but unfortunately without convincing results in practice, because the majority of patients will remain with neurological damage. Further treatment is mostly symptomatic and includes drugs for pain control (anticonvulsants, antidepressants and narcotics) (13).

However, the most important treatment for ischemic neuropathy is immediate closure of the access, which increases the probability of recovery and leads to partial or full restoration of the sensory and motor function. That's why it would better education and awareness on the part of the surgeon and nephrologist should lead to an early diagnosis and the proper management of ischemic neuropathy (17).

Uremic and diabetic polyneuropathy

Uremic and diabetic polyneuropathy is also a type of peripheral neurological dysfunction, characterized by acute onset and bilateral symmetry. It worsens with the duration of the primary condition that caused it, and distal sensory symmetry is a common denominator for both diabetic and uremic etiology. If they occur simultaneously, the deterioration of the clinical condition can be even more progressive, with gradual progression as time goes on, creating even greater disability, making patients unable to stand or walk. The effect of uremic or diabetic peripheral polyneuropathy on the manifestation of symptoms is without significant differences, except that the clinical symptoms of diabetic neuropathy are most often the result of the influence of symptomatic autonomic neuropathy, and the severity of symptoms is positively correlated with the duration of diabetes and chronic renal insufficiency that is dependent on dialysis (16).

Compressive neuropathy

Carpal tunnel syndrome occurs in about 10% of the uremic population with a prevalence of about 3% and is the most common form of compressive neuropathy. It occurs as a result of damage to the median nerve in the area of the carpal tunnel of the hand joint, and all as a result of ischemia and compression of this nerve. Carpal tunnel syndrome can occur acutely, after a wrist fracture, but most often occurs as a result of chronic accumulation of multiple elements that increase pressure in the carpal tunnel, edema occurs, fibrosis, collagen proliferation, amyloid deposition, and thickening of blood vessel walls. However, in addition to the usual risk factors, genetic predisposition, obesity, hypothyroidism, and inflammatory arthropathies, such as rheumatoid arthritis and diabetes, should also be considered. The presence of an arteriovenous fistula can cause symptoms of an already existing asymptomatic lesion of the carpal tunnel through additional microischemia, as well as an independent increase of inflammatory deposits inside the carpal tunnel. There is a thought that the development of carpal tunnel syndrome, after the formation of an arteriovenous fistula, is a consequence of venous hypertension, due to compression of the median nerve. On the other hand, the deposition of β2-microglobulin in the carpal tunnel is a consequence of amyloidosis-induced carpal tunnel syndrome, which represents a serious complication of hemodialysis, independent of the influence of arteriovenous fistula. In dialysis patients, compared to the general population, compressive nerve entrapment syndrome occurs more often. Ischemic monomyelopathy and complex regional pain syndrome represent complications due to nerve damage during the surgical procedure of creating vascular access. The etiology of compressive neuropathy is diverse and knowledge of the anatomy of the vascular and nerve supply is essential for diagnosis and intervention to prevent complications. The anatomical proximity of the brachial artery and the median nerve may be the reason for potential compression due to the development of an eventual (pseudo)aneurysm or hematoma that occurs after an unsuccessful

puncture of the fistula. Clinical manifestations of median nerve entrapment reduce wrist flexion, and pronation and make fine hand movements difficult, which significantly affects the patient's functional ability. Cubital tunnel syndrome is a phenomenon that occurs as a result of pinching of the ulnar nerve in the cubital fossa, it is also called Guyon's syndrome, it is manifested by pain in the hand, paresthesia and dominant weakness of the muscles of the hand, where created arteriovenous fistula. This manifestation indicates a gradual compression syndrome of the medial and ulnar nerve. The differential diagnosis in relation to ischemic syndrome due to distal hypoperfusion is the absence of a temperature difference between the arms, as well as good pulses of the radial artery. There are conflicting views in the literature on whether an arteriovenous fistula increases the risk of developing carpal tunnel syndrome (3, 7, 18–22).

Carpal tunnel syndrome, as a compressive neuropathy, is clinically manifested by numbness, and tingling pain in the distribution region of the median nerve, most often as a result of compression of the median nerve in the area of the carpal tunnel, it can be progressive due to various sensory and motor disorders. It occurs in 2-4% of the adult population and women are more often affected than men. The incidence of carpal tunnel syndrome in the general population is approximately one to five cases per 1000 people per year, more often in the dominant hand (18–22).

The incidence of carpal tunnel syndrome in patients with end-stage renal disease ranges between 8% and 31%, with a tendency to increase in relation to the duration of hemodialysis. Some data indicate indicate that carpal tunnel syndrome occurs in up to 30.5% of the extremities where the arteriovenous fistula was created, while the frequency in the opposite extremity is 12.2%. Therefore, arteriovenous fistula, due to the diversion of blood from the distal limbs, is cited as one of the causative factors contributing to the occurrence of carpal tunnel syndrome, but still without clear evidence. Perhaps one of the reasons is that there are no studies that used electrophysiological tests to confirm the diagnosis (3, 7, 18–22).

In the general population, carpal tunnel syndrome usually occurs in the dominant hand, while in patients with arteriovenous fistula, it occurs in the hand with the fistula (usually the non-dominant limb). In patients with arteriovenous fistulas, swelling of the forearm and hand may occur, most likely as a result of destruction of the valve of the superficial veins distal to the arteriovenous anastomosis. The swelling is more pronounced in post-puncture fistula vein compression for mechanical hemostasis when venous pressure can reach the level of systemic arterial pressure. Amyloid deposits of β2-microglobulin are another proposed etiological factor that, by provoking inflammation, leads to the formation of adhesions and edema, thus compressing the median nerve, regardless of the type of vascular access, thus accumulating β2-microglobulin in the synovium and tendon area, which can be the reason for the establishment of carpal tunnel symptoms in patients who do not have arteriovenous fistulas (19).

Median nerve entrapment is clinically manifested in the area of innervation of the medial nerve, that is, in the palmar region, with pain, numbness and a burning sensation, mostly during dialysis and at night. Muscular changes are manifested by weakness and atrophy of the thenar muscles, which is clinically reflected in the patient's inability to join the thumb and index finger. When in the later period, motor dysfunction occurs, the recovery is prolonged and uncertain. In the general population without chronic kidney disease approximately 15% of patients have relevant complaints, but only 2 to 3% of them have a confirmed diagnosis of median nerve entrapment and that, unfortunately, is only based on the clinical picture. The research results indicate a significant increase in the incidence median nerve entrapment (up to ten times), with the dominance of men, with an uncertain outcome, and frequent recurrences in the postoperative period. Symptoms of compressive neuropathy most often appear after 6 to 8 years of hemodialysis treatment, which supports the view that compressive neuropathy increases with longer time spent on dialysis (16).

The diagnosis of carpal tunnel syndrome is usually based on clinical findings, but recently ultrasonography has been proposed as a valuable test for the diagnosis and evaluation of carpal tunnel syndrome, that is, the measurement of the cross-sectional area of the median nerve (15). Many comorbidities and various conditions contribute to the severity of the clinical picture of carpal tunnel syndrome, such as diabetes, pregnancy, gout, acromegaly, rheumatoid arthritis, amyloidosis, hypothyroidism, use of corticosteroids and estrogen, etc. This form of compressive peripheral neuropathy is more common in some occupations that require more activity of the hand joints, such as construction workers and typists. It is believed that the etiology of carpal tunnel syndrome, in hemodialysis patients, is the deposition of amyloid protein in the area of the carpal tunnel, elevated venous pressure distal to the site, nerve ischemia, edema, damage to the median nerve by the uremic toxin, as well as some still insufficiently known reasons. It is still not clear enough which is the predominant causative factor, whether mechanical (due to the thickening of surrounding tissues) or vascular (due to arteriovenous fistula). Some studies, however, favor compression of the median nerve as a consequence of amyloid protein deposition. When there is a strong suspicion of carpal tunnel syndrome and electrodiagnostic tests do not confirm the diagnosis, the so-called provocative tests, namely Phalen's and Tinel's

tests, of which Phalen's test showed more reliable results. Therefore, the validity of electrodiagnostic results is increasingly being questioned, especially in patients with arteriovenous fistula who are treated with chronic hemodialysis. In patients with chronic kidney disease, distal motor and sensory polyneuropathy is caused by segmental demyelination and degeneration of axons, on the other hand, the presence of central-peripheral axonopathy is indicated by a reduced conduction velocity of sensory and motor nerves, which is why doubts have been expressed about the reliability of electrodiagnostic tests in patients on hemodialysis (16).

The treatment strategy of compressive neuropathy recommends decompressive operations in the region of the median nerve. This type of intervention, in the general population, relieves pain in more than 70% of patients, and they are completely satisfied with the intervention. However, the return of muscle strength occurs gradually, months later. In hemodialysis patients, the results are less satisfactory. Nevertheless, improvement was reported by 76% of those operated on, but it turned out that patients with preoperative symptoms longer than 2 years had the least improvement, especially motor functions. Operative intervention involves resection of the calcified medial artery, however, if a tourniquet is used for postpunctal hemostasis, it must be released to assess whether the resection leads to circulatory disturbances in the hand. If the artery is thrombosed, resection is uncomplicated and necessary to allow adequate tissue perfusion of the index and middle fingers (18).

It is clear that the pathophysiology of carpal tunnel syndrome in patients dialyzing with arteriovenous fistulas is currently unknown, and there is controversy as to whether arteriovenous fistula increases the risk of carpal tunnel syndrome developing. Correct and timely interventions are required for these different conditions and hence, the need to review the known literature to identify an evidence-based approach (22).

Conclusion

Due to the high incidence of peripheral neuropathies in patients with chronic kidney disease and the significant impact on the morbidity and mortality of these patients, early recognition of clinical symptoms and signs of peripheral neuropathy is extremely important. It is also important to understand the pathophysiological mechanisms of peripheral neuropathies in order to approach an effective strategy of diagnostic and therapeutic planning for these neurological complications.

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PERIFERNE NEUROPATIJE KOD BOLESNIKA SA HRONIČNOM BUBREŽNOM INSUFICIJENCIJOM

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Pacijenti sa hroničnom bolešću bubrega često pate od perifernih neuropatija, koje značajno utiču na morbiditet i mortalitet. Dakle, važno je razumeti patofiziološki mehanizam ovih neuroloških komplikacija da bi se mogla osmisliti efikasna strategija upravljanja ovim poremećajima. Cilj ovog preglednog rada bio je da ukaže na važnost otkrivanja kliničkih parametara koji omogućavaju dijagnozu i rano lečenje periferne neuropatije.

U te svrhe korišćene su baze podataka *PubMed* i *MEDLINE*. Podaci predstavljeni u ovom radu prikupljeni su iz literature na koju je uputila pretraga sledećih ključnih reči: periferna neuropatija, hronična bubrežna insuficijencija, hemodijaliza, klinička slika, lečenje.

Pronađeno je ukupno dvadeset radova koji su ispunili pomenute kriterijume – osam preglednih radova, sedam originalnih radova, četiri prikaza slučaja i jedna monografija.

Budući da kliničke manifestacije periferne neuropatije nisu specifične, veoma je važno da se rano otkriju kako bi se sprečilo njihovo napredovanje i postigao uspeh u njihovom lečenju.

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***Ključne reči**: periferna neuropatija, hronična bubrežna insuficijencija, hemodijaliza, klinička slika, lečenje*