Journal of Society Medicine
Research & Review Articles on Diseases
Journal of Society Medicine. 2026; 5 (10)

Hypertension as a Risk Factor for Diabetic Peripheral Neuropathy in Patients with Type 2 Diabetes Mellitus: A Narrative Review

Widha Zhafira Dewi [1*], Yudhisman Imran [2]

[1] *Faculty of Medicine, Universitas Trisakti, Jakarta, Indonesia*
[2] *Department of Neurology, Faculty of Medicine, Universitas Trisakti, Jakarta, Indonesia*

***Corresponding Author**: Yudhisman Imran, Email: yudhisman.imran@trisakti.ac.id

ARTICLE INFO

Article history:
Received
20 July 2026

Revised
12 September 2026

Accepted
31 October 2026

Manuscript ID:
JSOCMED-20072026-510-4

Checked for Plagiarism: Yes

Language Editor:
Rebecca

Editor-Chief:
Prof. Aznan Lelo, PhD

ABSTRACT

Introduction: Type 2 diabetes mellitus (T2DM) is a chronic metabolic disease frequently complicated by diabetic peripheral neuropathy (DPN), which substantially impairs the quality of life and predisposes patients to foot ulceration and amputation. Hypertension, a highly prevalent comorbidity in T2DM, is an important modifiable risk factor for DPN. This review aims to synthesize the current evidence on the association between hypertension and DPN in patients with T2DM and elucidate the underlying pathophysiological mechanisms.

Methods: A narrative literature review was conducted using electronic databases, including PubMed and Google Scholar, to identify clinical, epidemiological, and experimental studies that examined the relationship between blood pressure and DPN in patients with diabetes.

Results: Observational studies across diverse populations consistently demonstrated that coexisting hypertension is associated with a higher prevalence and greater severity of DPN, and multivariate analyses identified hypertension as an independent risk factor after adjusting for age, diabetes duration, and glycemic control. Mechanistically, hypertension acts synergistically with chronic hyperglycemia to aggravate endothelial dysfunction, oxidative stress, inflammation, and structural remodeling of the vasa nervorum, resulting in endoneurial hypoxia, demyelination, and accelerated nerve degeneration. Experimental and imaging studies have corroborated more severe nerve injury when both conditions coexist.

Conclusion: Hypertension is a modifiable risk factor for the development and progression of DPN in patients with T2DM. Blood pressure control, along with glycemic management, should be integrated into DPN prevention strategies, and interventional studies are warranted.

Keywords

Type 2 Diabetes Mellitus, Diabetic Peripheral Neuropathy, Hypertension, Microvascular Dysfunction, Modifiable Risk Factor

How to cite: Dewi WZ, Imran Y. Hypertension as a Risk Factor for Diabetic Peripheral Neuropathy in Patients with Type 2 Diabetes Mellitus: A Narrative Review. *Journal of Society Medicine*. 2026; 5 (10): 407-414. **DOI: https://doi.org/10.71197/jsocmed.v5i10.316**

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder with a steadily increasing global prevalence and has become one of the major public health challenges of the 21st century. According to the International Diabetes Federation (IDF), approximately 537 million adults, representing 10.9% of the global adult population, were living with diabetes in 2021, and Indonesia ranked among the five countries with the highest number of people with diabetes [1]. Beyond its metabolic disturbances, T2DM leads to long-term complications that adversely affect quality of life, increase morbidity, and contribute substantially to disability and mortality [2].

© 2026 Dewi and Imran. This work is published by CoinReads Media Prima Ltd. The full terms of this license are available at https://www.coinreads.com/terms.php and attribution 4.0 International (https://creativecommons.org/licenses/by/4.0/). The article has been reviewed and approved by the author before being submitted for publication. Journals, editor in chief and editorial board have no right or obligation to correct or be responsible for inaccurate and misleading data if any. It is the responsibility of the author.

In parallel with the rising prevalence of T2DM, chronic complications have become increasingly common, particularly microvascular complications such as diabetic peripheral neuropathy (DPN), diabetic nephropathy, and diabetic retinopathy [3]. DPN is one of the most frequently encountered complications [4]. DPN is a chronic complication characterized by peripheral nerve damage resulting from prolonged hyperglycemia in the absence of other identifiable causes of neuropathy, and it typically presents with tingling, numbness, pain, burning sensations, and sensory loss in the distal extremities in a characteristic "stocking-and-glove" distribution [5]. Pfannkuche et al. reported that approximately 42.2% of patients with T2DM were affected by DPN [6]. DPN substantially impairs quality of life, as neuropathic pain and sensory deficits interfere with daily activities [7]. Moreover, loss of protective sensation in the lower extremities increases the risk of unnoticed injuries, which may progress to diabetic foot ulcers and severe infections [8]. Several factors contribute to the development of DPN, including a longer duration of diabetes, poor glycemic control, advanced age, obesity, dyslipidemia, smoking, and comorbid cardiovascular disease [9]. Chronic exposure to hyperglycemia and dyslipidemia induces metabolic disturbances that activate multiple oxidative stress pathways, ultimately leading to peripheral nerve dysfunction and degeneration [10]. In addition to these factors, hypertension is a highly prevalent comorbidity in patients with diabetes and has been increasingly recognized as a "missed" modifiable risk factor that accelerates the development of DPN [11]. Elevated blood pressure contributes to microvascular damage by increasing pressure within the small vessels supplying peripheral nerves, thereby reducing endoneurial blood flow and tissue oxygenation and impairing glucose metabolism within neural tissue [12].

In patients with diabetes, the coexistence of hypertension further increases the risk of DPN through synergistic effects of chronic hyperglycemia and elevated blood pressure on oxidative stress, inflammation, and endothelial dysfunction. Consequently, hypertension should be regarded not merely as a common comorbidity but as an independent contributor to the onset and progression of DPN [11]. Although several studies have demonstrated a significant association between hypertension and DPN in patients with T2DM, hypertension remains underemphasized in routine DPN prevention compared with glycemic control. Therefore, this narrative review aims to synthesize current evidence on the role of hypertension in the development of DPN in patients with T2DM and to elucidate the underlying mechanisms of peripheral nerve injury in this setting.

METHODS

This review searched PubMed and Google Scholar for publications on hypertension, T2DM, and DPN. Terms included "type 2 diabetes mellitus," "diabetic peripheral neuropathy," "diabetic neuropathy," "hypertension," "blood pressure," and "risk factor." Original articles (cross-sectional, case–control, and cohort studies), animal studies, systematic and narrative reviews, and guidelines in English or Indonesian were included. Studies selected for epidemiology, risk factors, and pathophysiological mechanisms linking hypertension and DPN were included. The findings were synthesized thematically.

DISCUSSION

Type 2 Diabetes Mellitus

T2DM is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from reduced tissue responsiveness to insulin (insulin resistance), which may eventually lead to impaired pancreatic β-cell function and inadequate insulin secretion [13]. As the disease progresses, chronic hyperglycemia gradually damages multiple organ systems through injury to the microvasculature, leading to microvascular complications, including diabetic nephropathy, diabetic retinopathy, and diabetic neuropathy [3].

Diabetic Peripheral Neuropathy: Definition and Clinical Spectrum

DPN is one of the most common long-term complications of diabetes and is characterized by damage to the peripheral nervous system as a consequence of chronic hyperglycemia, which induces progressive injury to peripheral nerve fibers. Clinically, DPN may present with sensory disturbances (numbness, tingling, and

neuropathic pain), motor dysfunction (muscle weakness and impaired coordination), and autonomic abnormalities [14]. Distal symmetric polyneuropathy is the most common clinical subtype [5].

Epidemiology of Diabetic Peripheral Neuropathy

DPN is among the most prevalent complications in individuals with T2DM, and it is estimated that approximately 50%–66% of individuals with diabetes will develop DPN during their lifetime. Although neuropathy may occur in all types of diabetes, its prevalence is generally higher in T2DM, and the risk increases with longer disease duration and the presence of comorbidities [2]. In Indonesia, the reported prevalence of DPN among patients with T2DM ranges from 30% to 50% [15].

Pathophysiology of Diabetic Peripheral Neuropathy

Chronic hyperglycemia initiates multiple interconnected pathophysiological mechanisms that contribute to DPN. Activation of the polyol pathway increases the conversion of glucose to sorbitol, resulting in nicotinamide adenine dinucleotide phosphate (NADPH) depletion. This reduces cellular antioxidant capacity and generates oxidative stress that damages both neurons and the microvasculature supplying peripheral nerves. Oxidative stress is further amplified by the activation of the hexosamine pathway and the formation of advanced glycation end products (AGEs), which promote inflammation and neuronal injury [16-18].

In addition, chronic hyperglycemia induces microvascular alterations, including thickening of the vascular basement membrane, endothelial dysfunction, and impaired regulation of vasodilation and vasoconstriction. These changes reduce nerve perfusion, resulting in endoneurial hypoxia and ischemia. Neural injury is accompanied by altered ion channel expression, neuronal hyperexcitability, and abnormal nerve fiber sprouting, which enhance pain signal transmission. Persistent nociceptive input eventually leads to central sensitization through increased glutamatergic activity and N-methyl-D-aspartate (NMDA) receptor activation, along with reduced γ-aminobutyric acid (GABA)-mediated inhibition within the central nervous system. Collectively, these mechanisms give rise to the clinical manifestations of neuropathic pain, including burning sensations, tingling, electric shock-like pain, diminished pain and temperature sensation, and motor weakness [19].

Clinical Manifestations

Patients with DPN commonly present with tingling, numbness, burning sensation, pain, and/or muscle weakness. These symptoms typically begin in the feet and progress proximally in a length-dependent “stocking-and-glove” pattern. Manifestations are usually symmetrical, with sensory deficits predominating over motor impairments. Some patients experience numbness while simultaneously exhibiting heightened sensitivity to normally nonpainful stimuli, a phenomenon known as allodynia. In severe cases, DPN leads to chronic, non-healing foot ulcers; approximately 15% of patients develop foot ulceration during the course of the disease, and a substantial proportion eventually require lower limb amputation [19].

In addition to distal symmetric polyneuropathy, patients with T2DM may develop other forms of neuropathy. Small-fiber neuropathy is often an early manifestation and may remain asymptomatic and undetected in its initial stages. Autonomic neuropathy may present with urinary dysfunction, gastrointestinal symptoms, erectile dysfunction, heat intolerance, dysphagia, cardiac arrhythmias, orthostatic hypotension, and syncope. Diabetic radiculoplexus neuropathy involves the major nerve plexuses and is characterized by pain, muscle weakness, and impaired balance, whereas mononeuropathy affects a single peripheral nerve, such as the median or peroneal nerve, with manifestations depending on the nerve involved [14].

Risk Factors for Diabetic Peripheral Neuropathy

The development of DPN in T2DM is influenced by multiple interacting risk factors, which may be classified as modifiable or non-modifiable. Modifiable risk factors include hypertension, dyslipidemia, smoking, poor glycemic control, being overweight or obese, and excessive alcohol consumption, whereas non-modifiable risk factors include older age, longer duration of diabetes, and genetic predisposition [12].

A longer duration of diabetes increases the risk of DPN due to prolonged exposure to chronic hyperglycemia. Rahmi et al. reported a significant correlation between the duration of T2DM and the occurrence of DPN [20]. Similarly, Liu et al. demonstrated that compared with patients with diabetes for less than 5 years, those with a disease duration of 10–15 years had a significantly higher risk of DPN (OR = 1.629; 95% CI: 1.261–2.106; $p < 0.001$), with the risk increasing further among those with a duration of ≥15 years (OR = 1.898; 95% CI: 1.483–2.429; $p < 0.001$) [21].

Hypertension: Definition and Classification

Hypertension is defined as persistently elevated systolic blood pressure (SBP) of ≥140 mmHg and/or diastolic blood pressure (DBP) of ≥90 mmHg [22]. Based on etiology, hypertension is classified as primary (essential) or secondary. Primary hypertension accounts for approximately 80%–95% of cases and results from complex interactions between genetic and environmental factors, whereas secondary hypertension arises from identifiable underlying conditions, such as renal or endocrine disorders. Because hypertension is frequently asymptomatic, many individuals remain undiagnosed until elevated blood pressure is detected during routine evaluation [23].

Epidemiology of Hypertension in Type 2 Diabetes Mellitus

Hypertension is one of the most common comorbidities in patients with T2DM, and the two conditions frequently coexist and synergistically drive both micro-and macrovascular complications. Naseri et al. reported that hypertension was present in 70.5% of patients with T2DM, with a higher prevalence among women than men [24]. In contrast, Al-Hadi et al. found hypertension in 46.8% of patients with T2DM in an Indonesian hospital setting [25]. Globally, the prevalence of hypertension among individuals with T2DM is estimated to be 50%–80%, and patients who are hypertensive at the time of diabetes diagnosis exhibit higher mortality and an increased risk of cardiovascular complications compared with normotensive patients [26].

Table 1. Summary of key clinical and experimental studies evaluating the association between hypertension and diabetic peripheral neuropathy (DPN).

Study	Design / population	Key findings
Jarmuzewska et al., 2007 [34]	Clinical study; patients with T2DM	Hypertension independently associated with sensorimotor neuropathy after multivariable adjustment ($R^2 = 0.17$, $p = 0.023$)
Sanada et al., 2015 [32]	Experimental animal model; sural nerve morphometry	Combined hypertension and diabetes produced more severe axonal atrophy, demyelination, small-fiber loss, and microvascular abnormalities
Rahmawati et al., 2018 [29]	Cross-sectional; patients with T2DM, Indonesia	History of hypertension significantly associated with DPN ($p = 0.03$)
Esmat et al., 2022 [27]	High-resolution ultrasound of the median nerve; hypertensive patients with and without diabetes	Higher neuropathy prevalence in hypertension plus diabetes vs. diabetes alone (34% vs. 22%)
Hasyim et al., 2023 [30]	Observational; outpatients with T2DM, Indonesia	Significant relationship between hypertension and DPN ($p = 0.001$)
Asram et al., 2024 [28]	Case–control; 124 patients with diabetes, Indonesia	Significant association between hypertension and DPN ($p = 0.0011$)
Gebreziher et al., 2024 [33]	Multicenter cross-sectional; patients with T2DM, Ethiopia	69.6% had hypertension >5 years; neuropathy frequently observed in this group

DPN, diabetic peripheral neuropathy; T2DM, type 2 diabetes mellitus.

Association Between Hypertension and Diabetic Peripheral Neuropathy

Hypertension may influence the development of DPN through mechanisms involving microvascular injury [26]. Elevated blood pressure induces microvascular dysfunction, which impairs microcirculation that supplies oxygen and nutrients to peripheral nerves, reducing nerve conduction and compromising axonal integrity [27]. Clinical and experimental studies investigating this relationship are summarized in Table 1. In a case–control study of 124 patients with diabetes, Asram et al. demonstrated a significant association between hypertension

and DPN in T2DM (p = 0.0011) [28]. These findings are consistent with those of a cross-sectional study by Rahmawati et al., which identified a history of hypertension as significantly associated with DPN (p = 0.03) [29]. and with those of Hasyim et al., who reported a significant relationship between hypertension and DPN in T2DM (p = 0.001) [30].

Using high-resolution ultrasound, Esmat et al. demonstrated that patients with both hypertension and diabetes exhibited a moderate positive correlation between the vibration perception threshold and median nerve parameters (r = 0.39, p = 0.005), indicating a significant relationship between functional and structural nerve impairment. The study also reported a higher prevalence of neuropathy in patients with coexisting hypertension and diabetes than in those with diabetes alone (34% vs. 22%), suggesting that this combination accelerates peripheral nerve damage [27]. In a narrative review, Bondar et al. identified hypertension as an independent risk factor for DPN, attributable to impaired impulse conduction secondary to myelin sheath thinning accompanied by compensatory Schwann cell activation [31]. Consistently, a systematic review concluded that hypertension is a consistent, yet frequently overlooked, modifiable risk factor for diabetic neuropathy [11]. Beyond elevated blood pressure per se, prolonged hypertension contributes to peripheral neuropathy through chronic microvascular injury and nerve ischemia, and these effects appear to be synergistic with chronic hyperglycemia. Sanada et al. investigated this interaction in an experimental animal model comprising control, hypertension, diabetes, and combined hypertension and diabetes groups. Histopathological and morphometric analyses of the sural nerve have demonstrated that the combination of hypertension and diabetes produces more severe nerve damage, including axonal atrophy, demyelination, loss of small nerve fibers, and microvascular abnormalities [32]. In a multicenter study from Ethiopia, Gebreziher et al. reported that 69.6% of patients with T2DM had a history of hypertension lasting more than five years and that diabetic neuropathy was frequently observed in this group, suggesting a potential relationship between hypertension duration and DPN [33]. Furthermore, Jarmuzewska et al. demonstrated that hypertension remained independently associated with sensorimotor peripheral neuropathy in T2DM after adjusting for age, sex, diabetes duration, HbA1c, and serum cholesterol ($R^2 = 0.17$, p = 0.023) [34].

Pathophysiological Mechanisms Linking Hypertension and DPN

The mechanisms linking hypertension to DPN have not been fully elucidated. Chronic hyperglycemia initiates multiple pathways that promote oxidative stress and endothelial dysfunction, leading to increased arterial stiffness, progressive microvascular injury, and hypertension further amplifies these deleterious effects. Reduced Na^+/K^+-ATPase activity has been reported in hypertensive states, which may aggravate hyperglycemia-induced polyol pathway dysfunction [11]. Patients with diabetes also exhibit impaired myogenic tone and vascular hypertrophy, resulting in narrowing of the microvascular lumen; these alterations compromise endoneurial blood flow and contribute to progressive nerve injury [35]. Reduced luminal diameter may additionally impair nutrient and glucose transport, further disrupting neural glucose homeostasis [29]. Moreover, the loss of unmyelinated small nerve fibers in the skin has been shown to progress more rapidly under hypertensive conditions [36].

Inflammation is a potential link between these two conditions. Putri et al. found that poor glycemic control, reflected by elevated HbA1c levels, was linked to increased inflammation, indicated by higher hs-CRP concentrations, and that systolic blood pressure correlated positively with hs-CRP in patients with T2DM (r = 0.277, p = 0.044) [37]. Therefore, hypertension may exacerbate endothelial dysfunction and accelerate microvascular injury through inflammatory pathways. Higher blood pressure is consistently associated with severe neuropathic symptoms in patients with T2DM [38].

Clinical Implications

These findings have important implications for clinical practice. Because hypertension is a modifiable risk factor, systematic blood pressure control should be regarded as an integral component of DPN prevention, alongside glycemic management, rather than solely as a cardiovascular intervention [11]. Experimental data further suggest that therapy targeting the renin–angiotensin system, as part of combination treatment, may

improve neuropathy-related endpoints in diabetic models [36]. In practice, patients with T2DM and coexisting hypertension should be regarded as a high-risk group warranting regular neuropathy screening and structured foot care education to prevent ulceration and amputation [8].

Limitations of the Current Evidence

Several limitations of the available evidence must be acknowledged. Most clinical studies are cross-sectional or case–control in design, precluding causal inference, and many were conducted in single centers with modest sample sizes. The definitions of hypertension and DPN, as well as the diagnostic methods for neuropathy, vary considerably across studies, limiting comparability. Furthermore, important confounders such as antihypertensive drug classes, blood pressure variability, and the duration of hypertension have been inconsistently reported. Large prospective cohorts and randomized trials are needed to evaluate specific blood pressure targets and antihypertensive classes for DPN prevention.

CONCLUSION

DPN, a T2DM complication, impairs quality of life. Hypertension, a modifiable factor, is independently associated with DPN occurrence and severity. This involves microvascular damage, endothelial dysfunction, inflammation, and perfusion and oxygen delivery, synergizing with chronic hyperglycemia to accelerate nerve degeneration. Blood pressure control, glycemic management, and neuropathy screening should be integrated into care.

DECLARATIONS

None

CONSENT FOR PUBLICATION

The Authors agree to be published in the Journal of Society Medicine.

FUNDING

None

COMPETING INTERESTS

The authors declare no conflicts of interest in this case report.

AUTHORS' CONTRIBUTIONS

W. Z. D. contributed to the conceptualization of the review, literature search, data extraction and synthesis, and drafting of the manuscript. Y. I. contributed to the conceptualization, supervision, critical interpretation of the evidence, and critical revision of the manuscript for important intellectual content. Both authors have read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

ACKNOWLEDGMENTS

The authors gratefully acknowledge the Faculty of Medicine, Universitas Trisakti, Jakarta, and the Department of Neurology for their academic support and access to scientific resources that made this review possible.

REFERENCE

1. International Diabetes Federation (IDF). IDF Diabetes Atlas. 10th ed. Brussels: International Diabetes Federation; 2021.
2. Bodman MA, Dreyer MA, Varacallo MA. Diabetic peripheral neuropathy. In: StatPearls. Treasure Island (FL): StatPearls. 2025.
3. Sapra A, Bhandari P. Diabetes. In: StatPearls.Treasure Island (FL): StatPearls Publishing; 2023.

4. Amelia R, Wahyuni AS, Yunanda Y. Diabetic neuropathy among type 2 diabetes mellitus patients at Amplas primary health care in Medan city. Open Access Maced J Med Sci. 2019;7(20):3-3400.
5. Pop-Busui R, Ang L, Boulton AJM. Diagnosis and treatment of painful diabetic peripheral neuropathy. Arlington (VA): American Diabetes Association; 2022.
6. Pfannkuche A, Alhajjar A, Ming A. Prevalence and risk factors of diabetic peripheral neuropathy in a diabetic's cohort: register initiative "diabetes and nerves." Endocr Metab Sci. 2020;1(2).
7. Olmez N, Degirmenci Y, Kececi H. Effects of pain and disability on quality of life in patients with diabetic polyneuropathy. Neurosci Med. 2015;6(3):98-106.
8. McMorrow R, Nube VL, Manski-Nankervis JA. Preventing diabetes-related foot ulcers through early detection of peripheral neuropathy. Aust J Gen Pract. 2022;51(11):8-833.
9. Papanas N, Ziegler D. Risk factors and comorbidities in diabetic neuropathy: an update 2015. Rev Diabet Stud. 2015;12(2):48-62.
10. Zhu J, Hu Z, Luo Y. Diabetic peripheral neuropathy: pathogenetic mechanisms and treatment. Front Endocrinol (Lausanne). 2024;14.
11. Sethi Y, Uniyal N, Vora V. Hypertension the 'missed modifiable risk factor' for diabetic neuropathy: a systematic review. Curr Probl Cardiol. 2023;48(4):101581.
12. Triulandari KD, Zulkarnaini A. Characteristics of risk factors for diabetic neuropathy in type 2 diabetes mellitus patients at RSI Siti Rahmah Padang, 2019–2020. Scientific Journal. 2022;1(2):96-102.
13. Goyal R, Singhal M, Jialal I. Type 2 diabetes. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023.
14. Anandhanarayanan A, Teh K, Goonoo M. Diabetic neuropathy. In: Feingold KR, Ahmed SF, Anawalt B. Endotext. South Dartmouth (MA): MDText.com, Inc.; 2022.
15. Nurjannah N, Saputra B, Erianti S. Description of the severity of peripheral neuropathy in type 2 diabetes mellitus patients. Prima Medika Sains Journal. 2023;5(1):16-20.
16. Armstrong D, Bley K, Simpson D, Staats P, Allen S, Carnevale A, et al. Diabetic peripheral neuropathy: pathophysiology and new insights into the mechanism of action of high-concentration topical capsaicin. J Exp Pharmacol. 2025;17:65-651.
17. Galiero R, Caturano A, Vetrano E. Peripheral neuropathy in diabetes mellitus: pathogenetic mechanisms and diagnostic options. Int J Mol Sci. 2023;24(4):3554.
18. Lin Q, Li K, Chen Y. Oxidative stress in diabetic peripheral neuropathy: pathway and mechanism-based treatment. Mol Neurobiol. 2023;60(8):94-4574.
19. Schreiber AK, Nones CF, Reis RC. Diabetic neuropathic pain: physiopathology and treatment. World J Diabetes. 2015;6(3):432.
20. Rahmi AS, Syafrita Y, Susanti R. The relationship between the duration of type 2 diabetes mellitus and the incidence of diabetic neuropathy. Jambi Medical Journal. 2022;10(1):5-20.
21. Liu J, Yuan X, Liu J, Yuan G, Sun Y, Zhang D, et al. Risk factors for diabetic peripheral neuropathy, peripheral artery disease, and foot deformity among the population with diabetes in Beijing, China: a multicenter, cross-sectional study. Front Endocrinol (Lausanne). 2022;13.
22. Page MR. The JNC 8 hypertension guidelines: an in-depth guide. Am J Manag Care. 2014;20(1):E8. PMID: 25618230
23. Adrian SJ, Tommy. Essential hypertension: latest diagnosis and management in adults. Mirror of the Medical World. 2019;46(3):8-172.
24. Naseri MW, Esmat HA, Bahee MD. Prevalence of hypertension in type-2 diabetes mellitus. Ann Med Surg (Lond). 2022;78.
25. Al-Hadi H, Zurriyani Z, Saida SA. Prevalensi diabetes melitus tipe 2 with the incidence of hypertension at the Internal Medicine Outpatient Clinic of Petramedika Ummi Rosnati Hospital. Jurnal Medika Malahayati. 2020;4(4).
26. Jia G, Sowers JR. Hypertension in diabetes: an update of basic mechanisms and clinical disease. Hypertension. 2021;78(5):205-1197.

27. Esmat A, Elshamy MI, Zakaria DM. Median nerve affection in hypertensive patients with and without diabetes: high-resolution ultrasound assessment. Diabetes Metab Syndr Obes. 2022;15:8-183.
28. Asram A, Hidayati PH, Mulyadi FE. The relationship between hypertension and diabetic neuropathy at Ibnu Sina Hospital, Makassar. Journal of Public Health (Prepotif). 2024;8(2):44-3338.
29. Rahmawati A, Hargono A. Dominant factor of diabetic neuropathy on diabetes mellitus type 2 patients. Jurnal Berkala Epidemiologi. 2018;6(1):8-60.
30. Hasyim H, Wangi H, Sartika SD. Risk factors for diabetic neuropathy in type 2 diabetes mellitus patients receiving outpatient care at the Internal Medicine Clinic of Hikmah Hospital, Makassar. Bosowa Medical Journal. 2023;1(2):77-82.
31. Bondar A, Popa A, Papanas N. Diabetic neuropathy: a narrative review of risk factors, classification, screening and current pathogenic treatment options (Review). Exp Ther Med. 2021;22(1):690.
32. Sanada LS, Tavares MR, Sato KL. Association of chronic diabetes and hypertension in sural nerve morphometry: an experimental study. Diabetol Metab Syndr. 2015;7(1):9.
33. Gebreziher LH, Beyene MG, Mekonnen D. Predictors of uncontrolled hypertension among type 2 diabetic patients in Ethiopia: multicenter cross-sectional study. Int J Cardiol Cardiovasc Risk Prev. 2024;22.
34. Jarmuzewska EA, Ghidoni A, Mangoni AA. Hypertension and sensorimotor peripheral neuropathy in type 2 diabetes. Eur Neurol. 2007;57(2):5-91.
35. Cheng Y, Cao W, Zhang J, Wang J, Liu X, Wu Q, et al. Determinants of diabetic peripheral neuropathy and their clinical significance: a retrospective cohort study. Front Endocrinol (Lausanne). 2022;13.
36. Davidson EP, Holmes A, Coppey LJ. Effect of combination therapy consisting of enalapril, α-lipoic acid, and menhaden oil on diabetic neuropathy in a high fat/low dose streptozotocin treated rat. Eur J Pharmacol. 2015;765:67-258.
37. Putri MA, Amani P, Adriani D. Glycemic control and cardiovascular risk assessment: a study on HbA1c and hs-CRP levels in type 2 diabetes mellitus. Journal of Biomedicine and Health. 2025;8(2):50-142.
38. Putu AGP, Govindha PA, Putri LI, Artana WI. Relationship of hypertension and the severity of neuropathy symptoms in type II diabetes mellitus patients at Sidhi Sai Medical Clinic. Int J Educ Soc Sci Res. 2019;2(6).