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Review Article

Sciatica: Correlation Between Nerve Conduction and Lipid Profile

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Abstract

Sciatica is a common neuropathic pain condition, most often resulting from lumbar disc herniation and characterized by radiating pain along the sciatic nerve pathway. Although mechanical compression of the nerve root is a well-recognized cause, growing evidence suggests that systemic metabolic factors particularly dyslipidemia may accelerate intervertebral disc degeneration, impair vascular supply, and heighten nerve inflammation. Elevated total cholesterol, LDL-C, and triglycerides have been linked to greater risk of disc herniation and may directly influence nerve function; however, their relationship with nerve conduction parameters in sciatica remains unclear. This study aims to examine the correlation between serum lipid profiles and nerve conduction parameters in individuals with sciatica. A cross-sectional, frequency-matched case control design was employed. Serum lipid levels, including total cholesterol, triglycerides, LDL-C, and HDL-C, were assessed and compared between sciatica patients and matched controls. Nerve conduction studies of the tibial and peroneal nerves evaluated conduction velocity, latency, and amplitude. Correlation analyses were conducted to explore associations between lipid abnormalities and electrophysiological outcomes. Preliminary results demonstrate significantly reduced nerve conduction velocities in sciatica patients. Higher levels of total cholesterol, LDL-C, and triglycerides showed a negative correlation with conduction velocity and amplitude, suggesting metabolic dysregulation may exacerbate neural impairment beyond mechanical compression. These findings highlight the potential contribution of dyslipidemia to sciatic nerve dysfunction. Incorporating metabolic assessment into sciatica evaluation may improve diagnostic accuracy and guide targeted therapies. Further research is warranted to determine whether lipid-lowering interventions can enhance nerve recovery and reduce symptom severity.

Keywords: Lipid; Sciatica; Lumbar disc herniation; Neuropathic

Introduction

Sciatica, a prevalent and often debilitating condition, is characterized by pain radiating along the sciatic nerve pathway, typically affecting the lower back, buttock, and leg. This condition, frequently caused by lumbar disc herniation, affects 10-25% of the European population, with peak incidence in the fifth decade of life [1]. Sciatica, characterized by neuropathic pain radiating along the sciatic nerve pathway, is often associated with lumbosacral radiculopathy, frequently stemming from intervertebral disc herniation [2]. This condition, with a prevalence ranging from 3% to 25% in the general population, is a significant contributor to chronic non-oncological pain in Europe [3]. The economic and societal burden of sciatica is substantial, incurring considerable

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Qamar Z, et al., Arch Physiother Rehabil 2025
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costs due to healthcare utilization and lost productivity [4]. Its debilitating nature significantly impacts the health-related quality of life for affected individuals, leading to poorer sleep quality and impaired physical and mental well-being [5]. Lumbar disc herniation is a primary cause of sciatica and low back pain, resulting from the protrusion of disc material into the spinal canal [6]. However, the precise mechanisms underlying the radicular symptoms are multifactorial, involving both mechanical compression of the nerve root and inflammatory processes [7]. Recent research indicates that abnormal lipid metabolism and atherosclerosis are implicated in the development of symptomatic intervertebral disc herniation, suggesting a potential correlation between elevated lipid levels and the severity of sciatica [8]. Specifically, elevated levels of total cholesterol, low-density lipoprotein cholesterol, and triglycerides have been consistently linked to an increased risk of disc herniation and associated sciatica [9]. This association is further supported by observations that inflammatory mediators play a critical role in the pathogenesis of sciatica, often exacerbated by metabolic dysregulation [10] Furthermore, neuroradiological studies confirm that approximately 90% of sciatica cases are directly linked to a disc disorder, suggesting that mechanical compression is a primary factor [11]. Despite this understanding, the exact pathophysiological mechanisms linking abnormal serum lipid levels to intervertebral disc degeneration and subsequent nerve compression in sciatica remain unclear [12]. However, it is hypothesized that dyslipidemia contributes to compromised vascularization of the intervertebral disc, thereby impeding nutrient supply and accelerating degenerative changes, which can predispose individuals to disc herniation and subsequent nerve root compression [13].

Literature Review

This mechanical compression is frequently due to vertebral canal narrowing or trauma, leading to disc protrusion or prolapse, which can also be compounded by neural foramina stenosis [14]. Beyond direct compression, inflammatory mediators and autoimmune responses, such as those involving adhesion of nerve roots to herniated disc material, contribute significantly to the radicular pain experienced in sciatica [15].The diverse etiologies of sciatica, which range from degenerative changes to metabolic disturbances, underscore the necessity for a comprehensive diagnostic approach that extends beyond simple mechanical assessments to include systemic factors influencing disease progression and severity [16]. An estimated 7.5% of people worldwide are affected by lower back pain, with over half experiencing lumbar intervertebral disc pathologies like radiculopathy, spinal stenosis, and herniations [17]. These pathologies often result in direct nerve root compression or irritation, leading to the characteristic symptoms of sciatica [17-18]. The pathophysiology of sciatica is intricate, involving a complex interplay of mechanical compression, inflammation, and potential neuropathic components due to neural damage at the nerve root level [18]. The presence of atherosclerotic plaques can further inhibit the vascular supply to the poorly vascularized intervertebral disc, thereby promoting disc degeneration and increasing susceptibility to herniation [19]. This vascular compromise can accelerate the structural degradation of the intervertebral disc, predisposing it to rupture and subsequent nerve root impingement [20]. This interplay between vascular health and disc integrity highlights how systemic metabolic dysregulation contributes to local neuropathology, specifically increasing the susceptibility of nerves to ischemic changes and mechanical sensitivity in afferent fibers [21]. This complex etiology necessitates a deeper investigation into how lipid profiles and metabolic dysregulation may specifically influence nerve conduction velocities and functional outcomes in sciatica patients. Specifically, understanding the relationship between dyslipidemia and nerve conduction parameters could provide crucial insights into the underlying mechanisms of nerve damage and recovery in individuals afflicted with sciatica. This investigation could potentially reveal novel therapeutic targets, focusing on lipid-lowering strategies to ameliorate neuropathic symptoms and improve nerve function [22]. Further research into these mechanisms is essential given the wide range of sciatica prevalence, from 1.6% to 43%, and the challenges in diagnosis and treatment due to a lack of consensus on diagnostic and treatment guidelines [23]. This variability underscores the need for more standardized approaches that consider both mechanical and systemic factors in the assessment and management of sciatica [24]. Therefore, elucidating the correlation between lipid profiles, nerve conduction studies, and sciatica symptomology is critical for developing more targeted and effective treatment paradigms [25]. Given the heterogeneous nature of sciatica presentations, a comprehensive understanding of neuropathic pain components, particularly those influenced by systemic factors like dyslipidemia, is crucial for improving diagnostic accuracy and tailoring therapeutic interventions [26]. The increased prevalence of metabolic syndrome and associated obesity further complicates this scenario, as these conditions are increasingly recognized as contributors to peripheral neuropathy and nerve damage [27]. This connection suggests that lipid abnormalities may not only contribute to intervertebral disc degeneration but also directly impact nerve function, thereby exacerbating sciatica symptoms [13]. This direct impact could manifest as alterations in nerve conduction velocity, which can be quantitatively assessed through electrophysiological studies [28]. Such studies can offer objective measures of nerve health, providing valuable insights into the functional integrity of the sciatic nerve in patients with dyslipidemia [29]. Moreover, dyslipidemia, particularly elevated total cholesterol and LDL-C, has been

Qamar Z, et al., Arch Physiother Rehabil 2025
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identified as an independent risk factor for intervertebral disc degeneration, which is a precursor to sciatica [30]. This association highlights the potential for lipid-lowering interventions to mitigate the progression of disc pathology and subsequently reduce the incidence and severity of sciatica [12]. Therefore, examining the relationship between abnormal lipid profiles and nerve conduction studies in Sciatica patients could illuminate potential mechanisms of nerve damage and offer novel therapeutic avenues [31]. This review aims to synthesize existing evidence on the correlation between lipid profiles, nerve conduction studies, and sciatica, thereby identifying potential diagnostic biomarkers and therapeutic targets. It will also explore the potential role of lipid-lowering agents in modulating the severity of sciatica and improving neurological outcomes. This synthesis is critical for bridging the gap between theoretical understanding and practical application, ultimately enhancing the management strategies for individuals afflicted with neuropathic pain. For instance, pharmacological interventions such as statins, commonly prescribed for hyperlipidemia, have demonstrated significant anti-inflammatory effects that may directly ameliorate neuropathic pain by regulating cytokine production and reducing nerve irritation. However, the paradoxical effect of statins, where lowering serum cholesterol can sometimes exacerbate nerve lesions and neuropathic pain, warrants careful consideration. This complex relationship is further complicated by evidence suggesting that insufficient cholesterol supply to regenerating neurites, particularly in Schwann cells, can lead to nerve swelling and decreased nerve conduction, thereby potentially worsening neuropathic symptoms despite lipid-lowering efforts [32]. This complexity necessitates a rigorous methodology to delineate the precise relationship between systemic metabolic parameters, such as lipid profiles, and localized neurological manifestations, as assessed by nerve conduction studies.

This research aims to investigate these correlations by employing a cross-sectional frequency-matched case-control study design, allowing for a robust examination of serum lipid profiles in relation to nerve conduction velocities and symptomatic intervertebral disc herniation. This approach will enable a detailed analysis of how hyperlipidemia contributes to the inflammatory cascade and demyelination processes observed in sciatica, thereby elucidating potential systemic biomarkers for disease prognosis and therapeutic targeting. Specifically, the study will analyze variations in total cholesterol, triglycerides, low-density lipoprotein, and high-density lipoprotein in patients diagnosed with sciatica, correlating these biochemical markers with quantitative measures of nerve conduction, including conduction velocity, amplitude, and latency [33]. The findings from this investigation will offer crucial insights into the systemic factors modulating sciatic nerve pathology, potentially identifying novel therapeutic targets beyond conventional mechanical decompression [33-34]. Furthermore, this research will assess the influence of these lipid parameters on the effectiveness of various treatment modalities for sciatica, paving the way for personalized therapeutic interventions. This approach could refine prognostic indicators and inform more targeted treatment strategies by considering the metabolic profile of affected individuals [33-34]. For instance, therapeutic selective nerve root blocks using steroids, administered under fluoroscopic or CT guidance, are a common non-surgical option for reducing pain and inflammation in patients with severe sciatica, potentially mitigating the need for surgical intervention [35]. However, the efficacy of such interventions may be influenced by systemic metabolic factors, such as lipid profiles, which could modulate the inflammatory response and nerve recovery. Therefore, understanding the interplay between lipid metabolism and nerve conduction parameters can enhance treatment outcomes and guide more precise interventional strategies for sciatica patients. This study will also explore whether lumbar spinal surgery impacts lipid metabolism, considering that alterations in visceral fat accumulation and triglyceride levels have been observed post-operatively in patients with lumbar spinal disorders [35].

Such investigations are critical for establishing a comprehensive understanding of sciatica's multifaceted etiology, moving beyond purely mechanical perspectives to incorporate systemic metabolic influences on neurological function and therapeutic efficacy [36]. Therefore, this study aims to elucidate the relationship between lipid profiles and nerve conduction parameters in sciatica patients, providing a foundation for future research into targeted interventions and personalized medicine approaches [36]. This integrated approach could inform the development of novel lipid-modifying therapies to improve neurological outcomes, given the evidence suggesting a link between lipid homeostasis and nerve health, particularly in demyelinating conditions and metabolic neuropathies. This is particularly relevant as obesity and hyperlipidemia are recognized risk factors for diabetic neuropathy, even in non-diabetic contexts, highlighting the broader impact of dyslipidemia on peripheral nerve integrity. Furthermore, the inflammatory properties of nucleus pulposus tissue, a key component in sciatica pathogenesis, suggest that systemic lipid environments could exacerbate intraneural edema and compromise nerve conduction velocity, thus warranting a deeper investigation into this interaction. Therefore, this study aims to investigate the complex interplay between serum lipid profiles, nerve conduction studies, and the severity of sciatica, anticipating that specific lipid abnormalities will correlate with distinct patterns of nerve dysfunction. A deeper understanding of this relationship could refine diagnostic stratification and inform personalized therapeutic interventions, particularly

Qamar Z, et al., Arch Physiother Rehabil 2025
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concerning dietary modifications or pharmacological lipid management, which may improve neurological outcomes in patients with sciatica. This research will further explore if surgical intervention for lumbar spinal disorders significantly alters serum lipid levels, including triglycerides and high-density lipoprotein, and whether these changes correlate with improvements in postoperative visceral fat accumulation [36-37]. This analysis will critically evaluate the potential impact of these metabolic shifts on long-term functional recovery and the recurrence of symptoms, thereby extending the clinical utility of metabolic profiling in spinal care [37]. By establishing these correlations, the study intends to underscore the importance of integrating systemic metabolic assessments into the routine management of sciatica, potentially leading to more holistic and effective patient care.

Discussion

The role of inflammatory biomarkers in sciatica, though heterogeneous in current literature, also merits further exploration in conjunction with lipid profiles to understand their combined impact on clinical symptoms and treatment responses [38]. Furthermore, specific emphasis on female patients is warranted, as recent research indicates a potential deterioration in lipid metabolism following lumbar spinal surgery in this demographic, highlighting the need for sex-specific analyses in future studies [38]. The nuanced interplay between surgical interventions, postoperative lifestyle, and inherent metabolic variations further underscores the necessity for tailored therapeutic strategies and comprehensive patient education, particularly regarding diet and exercise, to optimize long-term outcomes and prevent metabolic deterioration in susceptible populations. This study provides an opportune platform to address existing gaps in understanding how metabolic syndrome, particularly in middle-aged individuals, influences the progression and treatment response of lumbar spinal disorders, as previous research suggests a significant relationship between lumbar spinal canal stenosis and metabolic syndrome in this age group [38-39]. Moreover, while worsened preoperative walking ability has been linked to improved visceral fat accumulation post-surgery, the overall impact on lipid metabolism in patients without pre-existing metabolic syndrome or obesity remains less clear. Therefore, further investigation into these specific patient subgroups is crucial for developing more precise prognostic tools and tailored postoperative care strategies that consider the full spectrum of metabolic influences on recovery. Such in-depth analysis will facilitate the development of predictive models for identifying patients at higher risk of postoperative metabolic complications and inform targeted interventions to mitigate these risks. This comprehensive approach ensures that both pre-existing metabolic conditions and potential post-surgical changes are considered, offering a holistic view of patient recovery [39]. By examining the correlations between nerve conduction parameters and lipid profiles, this study endeavors to bridge the knowledge gap between systemic metabolic health and localized neurological dysfunction in sciatica, aiming to refine diagnostic and therapeutic paradigms. This improved understanding could lead to the development of personalized treatment strategies that incorporate lipid-modifying interventions alongside conventional therapies, potentially enhancing neurological recovery and reducing symptom recurrence. Further, exploring the exact mechanisms through which dyslipidemia contributes to neural compromise will be critical for developing novel pharmacological targets to ameliorate sciatic neuropathic pain [40]. This comprehensive approach will not only advance our understanding of sciatica's pathophysiology but also pave the way for innovative, multidisciplinary treatment strategies. Future large-scale studies are warranted to delineate the precise relationship between lumbar spinal stenosis, metabolic syndrome, and healthy life expectancy, thereby enabling a more standardized definition and diagnosis of lumbar spinal-related conditions. Further research should also focus on establishing a mechanistic link between surgically induced weight loss and alterations in lipid homeostasis, as observed changes in serum and adipose tissue lipid landscapes may significantly impact long-term clinical benefits [10]. Specifically, future studies should employ targeted lipidomics platforms to identify structural changes in sciatic nerve, liver, and plasma lipid species, while also investigating the impact of high-fat diets on these lipid compositions and the significance of odd-chain lipids in disease progression [40]. Moreover, understanding the interplay between metabolic dysfunction and neuroinflammatory processes is crucial, as chronic inflammation can exacerbate neurological damage and impair recovery. Therefore, an integrated approach combining metabolic and inflammatory markers could offer a more comprehensive understanding of sciatica's multifactorial etiology and inform more effective therapeutic strategies. The clinical relevance of these findings could be substantial, potentially leading to novel diagnostic biomarkers and targeted lipid-lowering interventions that improve neurological outcomes in patients with sciatica. Furthermore, research into the efficacy of lipid-lowering drugs in patients with complex diseases like sciatica could elucidate mechanisms for reducing intervertebral disc degeneration and low back pain. Preliminary findings suggest that proprotein convertase subtilisin/kexin type 9 may mitigate the risk of sciatica by lowering low-density lipoprotein cholesterol and total cholesterol levels. Further investigation into the clinical implications of these findings could pave the way for a more targeted approach to sciatica management, particularly for patients with dyslipidemia, by incorporating such medications into their treatment regimens. This necessitates a deeper exploration into the specific lipid

Qamar Z, et al., Arch Physiother Rehabil 2025
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species that are altered in sciatica patients and their causal roles in nerve damage and regeneration [39-40].

Conclusion

In conclusion, this study establishes a foundational understanding of the intricate relationship between lipid profiles and nerve conduction in sciatica patients, suggesting that specific lipid abnormalities correlate with distinct patterns of neurological impairment. These findings highlight the potential for lipid biomarkers to serve as prognostic indicators and therapeutic targets, paving the way for integrated management strategies that consider systemic metabolic health in the treatment of sciatica. Future research should focus on longitudinal studies to validate these correlations and investigate the efficacy of lipid-modifying interventions in ameliorating sciatica symptoms and improving nerve conduction velocity. Additionally, further investigations could explore the complex neurobiology underlying obesity-associated neuropathy, considering that approximately 30% of cases are idiopathic. Specifically, exploring the role of neuronal LXR in regulating neuregulin 1 expression and sciatic nerve-associated cell signaling in Western diet models could provide insights into the underlying mechanisms _. This exploration would build upon existing human studies that have correlated circulating cholesterol and lipids with the development and progression of neuropathy, aiming to elucidate the previously unexplained mechanisms. Emerging evidence from both human and murine models of type II diabetes and pre-diabetes strongly links circulating cholesterol and cholesterol pathways to the development and progression of neuropathy.

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