

CARPAL TUNNEL SYNDROME AND ANATOMICAL VARIATIONS: A LITERATURE REVIEW

SÍNDROME DO TÚNEL DO CARPO E VARIAÇÕES ANATÔMICAS: UMA REVISÃO DA LITERATURA

SÍNDROME DEL TÚNEL CARPIANO Y VARIACIONES ANATÓMICAS: UNA REVISIÓN DE LA LITERATURA

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ABSTRACT

Carpal Tunnel Syndrome (CTS), the most common compressive neuropathy of the upper limbs, results from compression of the median nerve, manifesting with pain and paresthesia. Its etiology is multifactorial, with a higher prevalence in women and the elderly. Intrinsic anatomical variations, such as a bifid median nerve (BNM), a persistent median artery (PMA), and anomalous muscles, can predispose to or aggravate CTS. BNM refers to a division of the median nerve, often associated with the PMA, an embryonic vessel whose persistence demonstrates microevolutionary growth in the population. The PMA is considered a compressive risk factor, although its protective role in nerve perfusion is debated. Anomalous muscles, such as the hypertrophied transversus carpi, also contribute to compression. The coexistence of multiple variations is common. Classic symptoms of CTS may present with atypical manifestations in the presence of anomalies. Diagnosis is clinical, complemented by examinations. Electroneuromyography (ENMG) is the gold standard. Ultrasound (USG) and Magnetic Resonance Imaging (MRI) are crucial for identifying anatomical variations, optimizing preoperative planning. Treatment ranges from conservative to surgical. Surgical decompression, preferably via open surgery in cases with anomalies, allows direct visualization, protects structures, and minimizes iatrogenic injuries. Preoperative

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identification of variations is therefore vital for surgical safety and efficacy. In conclusion, recognizing anatomical variations is essential in the management of CTS. Accurate diagnosis via imaging allows for personalized treatment planning, optimizing outcomes and patient safety.

Keywords: Carpal Tunnel Syndrome. Median Nerve. Persistent Median Artery. Anatomical Variation. Median Neuropathy. Compression Syndrome.

RESUMO

A Síndrome do Túnel do Carpo (STC), neuropatia compressiva mais comum dos membros superiores, resulta da compressão do nervo mediano, manifestando-se com dor e parestesia. Sua etiologia é multifatorial, com maior prevalência em mulheres e idosos. Variações anatômicas intrínsecas, como o nervo mediano bífido (NMB), a artéria mediana persistente (AMP) e músculos anômalos, podem predispor ou agravar a STC. O NMB refere-se à divisão do nervo mediano, frequentemente associado à AMP, um vaso embrionário cuja persistência demonstra aumento microevolucionário na população. A AMP é considerada fator de risco compressivo, embora seu papel protetor na perfusão nervosa seja debatido. Músculos anômalos, como o transverso do carpo hipertrofiado, também contribuem para a compressão. A coexistência de múltiplas variações é comum. Sintomas clássicos da STC podem apresentar manifestações atípicas em presença de anomalias. O diagnóstico é clínico, complementado por exames. A Eletroneuromiografia (ENMG) é o padrão-ouro. A Ultrassonografia (USG) e a Ressonância Magnética (RM) são cruciais para identificar variações anatômicas, otimizando o planejamento pré-operatório. O tratamento varia entre conservador e cirúrgico. A descompressão cirúrgica, preferencialmente por via aberta em casos com anomalias, permite visualização direta, proteção das estruturas e minimização de lesões iatrogênicas. A identificação pré-operatória das variações é, portanto, vital para a segurança e eficácia cirúrgica. Em conclusão, o reconhecimento das variações anatômicas é fundamental no manejo da STC. O diagnóstico preciso via exames de imagem permite um planejamento terapêutico personalizado, otimizando resultados e segurança do paciente.

Palavras-chave: Síndrome do Túnel do Carpo. Nervo Mediano. Artéria Mediana Persistente. Variação Anatômica. Neuropatia Mediana. Síndrome de Compressão.

RESUMEN

El síndrome del túnel carpiano (STC), la neuropatía compresiva más común de las extremidades superiores, se produce por la compresión del nervio mediano, que se manifiesta con dolor y parestesias. Su etiología es multifactorial, con mayor prevalencia en mujeres y ancianos. Variaciones anatómicas intrínsecas, como un nervio mediano bífido (NMB), una arteria mediana persistente (AMP) y músculos anómalos, pueden predisponer o agravar el STC. El NMB se refiere a una división del nervio mediano, a menudo asociada con la AMP, un vaso embrionario cuya persistencia demuestra un crecimiento microevolutivo en la población. La AMP se considera un factor de riesgo compresivo, aunque su papel protector en la perfusión nerviosa es controvertido. Músculos anómalos, como el transverso del carpo hipertrofiado, también contribuyen a la compresión. La coexistencia de múltiples variaciones es frecuente. Los síntomas clásicos del STC pueden presentarse con manifestaciones atípicas en presencia de anomalías. El diagnóstico es clínico y se complementa con exploraciones complementarias. La electroneuromiografía (ENMG) es el método de referencia. La ecografía (USG) y la resonancia magnética (RM) son cruciales para identificar variaciones anatómicas y optimizar la planificación preoperatoria. El

tratamiento varía de conservador a quirúrgico. La descompresión quirúrgica, preferiblemente mediante cirugía abierta en casos con anomalías, permite la visualización directa, protege las estructuras y minimiza las lesiones iatrogénicas. Por lo tanto, la identificación preoperatoria de variaciones es vital para la seguridad y la eficacia quirúrgicas. En conclusión, reconocer las variaciones anatómicas es esencial en el manejo del STC. Un diagnóstico preciso mediante imagen permite una planificación personalizada del tratamiento, optimizando los resultados y la seguridad del paciente.

Palabras clave: Síndrome del Túnel Carpiano. Nervio Mediano. Arteria Mediana Persistente. Variación Anatómica. Neuropatía Mediana. Síndrome de Compresión.

1 INTRODUCTION

Carpal tunnel syndrome (CTS) is the most common compressive neuropathy of the upper limbs, resulting from compression of the median nerve when passing through the carpal tunnel. This structure is formed by the carpal bones and the transverse ligament, and houses, in addition to the median nerve, nine flexor tendons (Yildizgoren et al., 2024). This nerve compression causes segmental or nodal demyelination of the nerve bundles and, in chronic cases, can progress to axonal degeneration, manifesting as paresthesia, pain, and numbness (Kouyoumdjian, 1999). The etiology of CTS is multifactorial, ranging from repetitive use and systemic conditions to inflammation and trauma. It affects a significant portion of the adult population, being more prevalent in women and the elderly (Gruber et al., 2025).

In addition to these factors, other alterations can predispose or aggravate CTS. This includes anatomical variations intrinsic to carpal tunnel, such as the bifidus median nerve (BMN), the persistent median artery (AMP), and anomalous muscles. Although extrinsic variations are less frequently associated with compression (Ragoowansi et al., 2002), the presence of these intrinsic anomalies can reduce the available space or alter local biomechanics, making the median nerve more vulnerable to compression (Yildizgoren et al., 2024; Solewski et al., 2021). Recognition of these variations is crucial for accurate diagnosis, proper preoperative planning, and prevention of iatrogenic complications during surgical release of the carpal tunnel (Pimentel et al., 2022; Alexander et al., 2020).

2 ANATOMICAL VARIATIONS OF CARPAL TUNNEL: TYPES, PREVALENCE AND CLINICAL IMPACT

The carpal tunnel is an osteofibrous compartment confined to the wrist, through which the median nerve and the flexor tendons of the fingers transit. Anatomical variations in these structures, although sometimes asymptomatic, may predispose to CTS.

The bifid median nerve (NMB) occurs when the median nerve, usually a single structure, splits into two bundles before or during its passage through the carpal tunnel (Yildizgoren et al., 2024). This bifidity, an anomaly of neural development, often coexists with a persistent median artery that interposes itself between nerve bundles (Quin et al., 2023; Aribindi et al., 2024). The prevalence of NMB varies widely but is about 50% higher in patients with CTS compared to healthy individuals, suggesting it as an anatomical risk factor (Asghar et al., 2022). However, its direct relationship as an independent risk factor is debated, as

many cases of NMB are asymptomatic or the prevalence does not differ significantly in control groups (Chen et al., 2017; Gruber et al., 2025). Variations in the anatomy of the median nerve, such as its early division, are observed in up to 50% of dissected hands (Agarwal et al., 2014; Kuhn et al., 2024).

The persistent median artery (AMP) is an embryonic vessel that normally regresses during development, but can persist with significant caliber in the adult (Rodríguez-Niedenführ et al., 1999; Singla et al., 2012). MPA can be classified as palmar type (it reaches the palm and contributes to the superficial palmar arch) or antebrachial (shorter, ends before the wrist) (Rodríguez-Niedenführ et al., 1999; Aragão et al., 2017). The overall prevalence of MPA varies considerably (0.6% to 44.2%), and studies indicate a remarkable microevolutionary increase in its frequency over time, with its prevalence evolving from approximately 10% in 1846 to 30% in 1997 (Lucas et al., 2020; Henneberg & George, 1992).

Traditionally, a significant caliber MPA is considered a risk factor for CTS, as it can directly compress the median nerve or serve as a fulcrum point (Yildizgoren et al., 2024; Alexander et al., 2020). Nayak et al. (2010) state that MPA may be involved in CTS, Pronator Syndrome, and Anterior Interosseous Nerve Syndrome. Complications such as thrombosis or MPA aneurysm can cause acute CTS or worsen symptoms (Abdouni et al., 2020; Quin et al., 2023). In up to 41% of reported cases, the artery can cross the median nerve (Natsis et al., 2009; Nayak et al., 2010). Interestingly, some studies show a lower prevalence of MPA in surgical patients with CTS compared to cadaveric studies, raising the hypothesis of a possible protective effect through better blood perfusion to the median nerve (Solewski et al., 2021; Chen et al., 2017; Gruber et al., 2025). In situations of vascular trauma, AMP can be vital to preserve hand perfusion (Calderon et al., 2021). In this sense, Rodríguez-Niedenführ et al. (1999) propose that MPA can play two main functional roles: to act as a satellite artery, supplying nutrients specifically to the median nerve — which favors its integrity and resistance to obstructive pathologies — or to function as a vessel destined to nourish adjacent non-nerve structures.

Anomalous muscles in or adjacent to the carpal tunnel, such as variations of the lumbricales muscles, can also contribute to median nerve compression (Quin et al., 2023). Among them, the transverse carpal muscle (TCM) stands out, which runs superficially and parallel to the transverse carpal ligament (Ragoowansi et al., 2002). When hypertrophied by repetitive strain of the wrist, MTC can lead to compression of the median nerve. In a study with patients who underwent carpal tunnel release surgery, MTC was found in 58% of

patients, showing a high prevalence of this structure compared to other variations such as MPA and NMB (Pimentel et al., 2022).

The coexistence of multiple variations, such as NMB and MPA, is common (Chen et al., 2017; Aribindi et al., 2024). Although the association between NMB and MPA is often pointed out as the most common (Pimentel et al., 2022), the coexistence of other anatomical variations is also described. These include TCM associated with the occurrence of an anomalous muscle belly of the superficial flexor tendon of the fingers, and the variable presence of the recurrent subligamentous (RRS) and transligamentous (RRT) branches of the median nerve. Although these branches do not always increase compression in the carpal tunnel, their presence can hinder surgical release in case of CTS. Other vascular variations, such as the brachioradialis artery (ABR) or the superficial ulnar artery (AUS), are equally important in the surgical context due to the risk of iatrogenic injuries (Lee et al., 2024; Herstam et al., 2022).

3 SYMPTOMS AND DIAGNOSIS

Classic symptoms of CTS include paresthesia and numbness in the fingers innervated by the median nerve, pain (with possible radiating to the forearm), and weakness in the hand. In advanced stages, muscle atrophy may occur. The presence of anatomical variations can lead to atypical manifestations, such as painless atrophy or paresthesia (Quin et al., 2023), and acute events such as MPA thrombosis can cause severe pain and paresthesia (Abdouni et al., 2020).

Diagnosis is primarily clinical, based on the patient's history and physical examination (including provocation tests such as Phalen and Tinel). Complementary tests are essential to confirm the diagnosis and, crucially, identify anatomical variations:

Electroneuromyography (ENMG) is the test that offers the greatest accuracy (gold standard) for the electrophysiological diagnosis of CTS, allowing the evaluation of nerve conduction (Pimentel et al., 2022).

High-resolution ultrasonography (USG) is an essential noninvasive tool for visualization of the median nerve (allowing identification of increased cross-sectional area) and for detecting NMB and MPA. This examination is crucial in preoperative planning to avoid iatrogenic injuries (Yildizgoren et al., 2024; Alexander et al., 2020). Its efficacy in detecting MPA in children has also been confirmed (Carry et al., 2019).

Magnetic Resonance Imaging (MRI) of the Wrist offers superior anatomical detail, being effective in detecting NMB, AMP, and anomalous muscles, complementing USG in complex cases (Quin et al., 2023).

CT angiography is used in vascular trauma or in cases of MPA thrombosis to evaluate in detail the circulation and the role of MPA in hand perfusion (Calderon et al., 2021; Abdouni et al., 2020).

4 TREATMENT

The treatment of CTS can be conservative or surgical, and the presence of anatomical variations influences the efficacy and choice of approach.

Conservative treatment, which includes the use of wrist splints, activity modifications, and medication, is indicated for mild to moderate cases (Yildizgoren et al., 2024). However, in cases of CTS secondary to significant structural anomalies, such as anatomical variations, satisfactory results with conservative treatment are less likely (Yildizgoren et al., 2024).

Surgical decompression is the definitive treatment when the conservative fails, or in cases of severe nerve compression. Classic open surgery is often preferable in cases with anatomical variations, as direct visualization allows the surgeon to identify and protect the NMB, AMP, and anomalous muscles, minimizing iatrogenic injuries (Pimentel et al., 2022; Alexander et al., 2020). This advantage, however, requires extra attention: MPA, for example, is usually located deep in the flexor retinaculum, but anatomical variations, such as its presence just below the palmar fascia, significantly increase the risk of iatrogenic injury (Butt et al., 2017). MPA should be preserved whenever possible, especially if it is crucial for hand circulation (Almodumeegh et al., 2021; Abdouni et al., 2020). Endoscopic surgery, although less invasive, offers a limited field of vision, increasing the risk of injury in the presence of variations (Natsis et al., 2009). Preoperative identification of variations is therefore essential for choosing the safest surgical technique (Yildizgoren et al., 2024).

5 CONCLUSION

CTS is a complex condition, and recognition of anatomical variations, such as NMB, AMP, and the presence of anomalous muscles, is critical for its effective management. Its embryological origin and prevalence, including the remarkable microevolutionary trend of increasing MPA in the population, influence the etiology and treatment of CTS.

Although the impact of some variations as risk factors is still debated, accurate diagnosis using tools such as ultrasound and MRI is crucial. These tools allow for safer and more personalized surgical planning, optimizing results. The integration of this knowledge into clinical practice significantly improves the diagnostic process and therapeutic management of CTS.

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