CASE REPORT

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PERSISTENT MEDIAN ARTERY AS A CAUSE OF CARPAL TUNNEL SYNDROME? A CASE REPORT HIGHLIGHTING THE INDISPENSABLE ROLE OF ULTRASONOGRAPHY IN DIAGNOSIS

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Abstract. *Carpal tunnel syndrome (CTS) stands as the most common entrapment neuropathy. The PMA, a transient embryonic vessel, is found in approximately 3-7% of the population, yet it is extremely rare to present as a possible symptomatic space-occupying lesion or a source of pulsatile compression within the carpal tunnel. Herein, we report a case of a 66-year-old female with CTS together with large PMA, surgically treated by simple open carpal tunnel release. Following meticulous surgical decompression and preservation of the artery in situ, the patient achieved complete symptomatic relief. Post-operative high-resolution sonography was utilized to confirm the vascular anomaly and document the successful decompression. This case underscores a critical diagnostic dilemma in orthopedic surgery: performing a blind carpal tunnel release in the elderly without prior imaging carries an inherent danger, as failure to detect such a variant preoperatively risks iatrogenic vascular injury.*

Key words: *persistent median artery, carpal tunnel syndrome, structural cause, undiagnosed risk, artery preservation, medical ultrasound*

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INTRODUCTION

Carpal Tunnel Syndrome (CTS) is the most common entrapment neuropathy, usually caused by a volume-pressure mismatch in the tight carpal tunnel [1]. Inflammatory changes and repetitive strain cause most cases, but structural anomalies account for a few [2, 3]. Missing these anomalies can cause serious risk during surgery [4]. The persistent median artery (PMA) is a remnant vessel from development that crosses the carpal tunnel, lying superficial to the median nerve (MN). PMA appears in up to 7% of people [5], but rarely causes CTS by chronic pulsatile compression [6], acute thrombosis [7], or in combination with nerve variations like a bifid MN [8, 9, 10].

This case report describes treating a 66-year-old patient in whom a large PMA was unexpectedly found during open carpal tunnel release. The large diameter of the PMA may have contributed to CTS. Despite an excellent

surgical outcome, this case underscores the significant surgical risk when high-resolution imaging is not used.

CASE REPORT

A 66-year-old right-hand dominant female presented to our clinic with an 18-month history of progressive right-hand symptoms. She reported severe nocturnal paresthesias that woke her 3-4 times nightly, pain radiating from the wrist to the forearm, and numbness corresponding to the MN distribution. Symptoms were transiently relieved by the Flick sign. Conservative management, including night splinting and NSAIDs, had failed to provide lasting relief. Physical examination elicited a positive Tinel's sign at the wrist and a positive Phalen's test (symptoms reproduced within 30 seconds). Mild hypotrophy of the thenar musculature was noted. The patient's subjective severity was quantified using the Bulgarian version of the Boston Carpal Tunnel Questionnaire (BCTQ) [11] prior to surgery, which revealed a Symptom Severity Scale (SSS) score of 3.8/5.0 and a Functional Status Scale (FSS) score of 3.1/5.0, confirming severe symptoms and moderate functional limitations. Electrodiagnostic studies confirmed a moderate-to-severe MN neuropathy at the wrist, marked by prolonged distal sensory and motor latencies. Due to the severity of her condition and the failure of conservative treatment, surgical decompression was indicated. An open carpal tunnel release was performed under regional anesthesia. Upon incision of the transverse carpal ligament, a patent, non-thrombosed, large-caliber PMA was unexpectedly exposed, lying radial to the MN (Figure 1). The MN itself appeared moderately swollen and displayed a distinct hourglass-like indentation at the site of compression. The PMA was meticulously preserved in situ, requiring careful release of all constraining fibrous attachments. Full decompression of the MN from the transverse carpal ligament and from any potential pulsatile compression was successfully confirmed. The patient reported an immediate, complete resolution of her nocturnal symptoms. At the six-week follow-up, she remained free of residual paresthesias. Physical examination showed a negative Phalen's sign and a resolving Tinel's sign. Postoperative BCTQ scores documented a clinical improvement- SSS of 4.6/5.0 and FSS of 4.2/5.0. High-resolution ultrasonography was performed postoperatively as a confirmatory measure to document the rare anatomical variant and ensure vascular patency. Sonography at the level of the pisiform demonstrated a thickened MN and clearly visualized the patent PMA (Figure 2a,b), confirming its position radial to the nerve. Color Doppler confirmed robust patency and flow within the PMA (Figure 2b,c). The contralateral hand was operated on due to CTS before the year; the sonography revealed no PMA (Figure 1c).

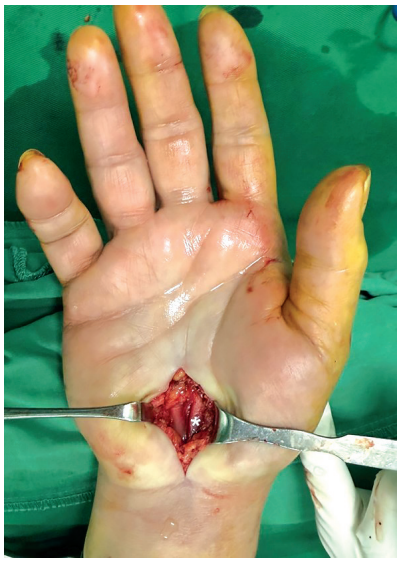

Fig. 1. Intraoperative imaging of PMA (asterisk)

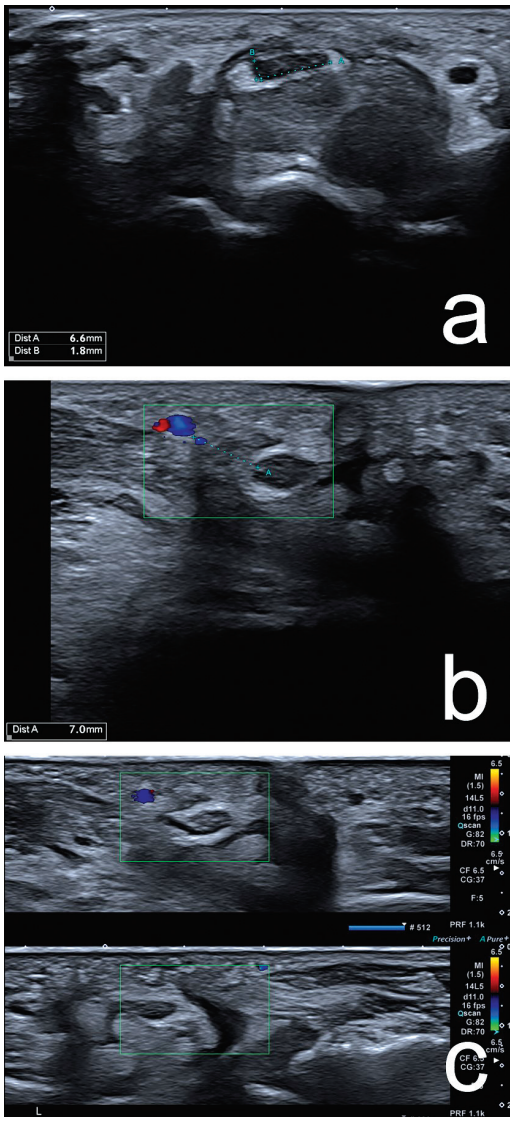

Fig. 2. Ultrasonography presented a thickened MN (a) and clearly visualized the PMA (b); color doppler confirmed robust patency and flow within the PMA on the left side and its absence of the contralateral left hand (c)

DISCUSSION

PMA as a predisposing structural factor

The accidental discovery and successful management of an unexpected patent PMA during this otherwise routine procedure offer several key lessons. The most urgent takeaway is the high risk introduced by performing a blind release when a highly vulnerable vascular anomaly could be the true causative lesion.

While current literature tends to focus on acute symptomatic PMA caused by thrombosis, typically in younger, highly active patients [19, 7], the presentation in our 66-year-old patient points strongly to the artery acting as a predisposing structural factor. Its chronic pulsatile compression became symptomatic only when compounded by age-related factors, specifically the reduced elasticity of the nerve and the non-compliant transverse carpal ligament, which increased the MN‘s susceptibility to chronic compression (Table 1).

Anatomical variants function as predisposing factors by occupying space and diminishing the functional volume of the carpal tunnel. As articulated by Georgiev et al. [12, 13], the simple presence of a variant structure – such as an accessory muscle or vessel – does not guarantee entrapment; rather, it drastically increases the nerve‘s vulnerability to compression when combined with secondary factors like edema, inflammation, or the reduced tissue compliance commonly observed in older patients [12, 13]. Given the complex etiology of CTS, a comprehensive examination for such underlying structural factors is essential [14, 15, 25].

The PMA, simply by occupying fixed space and pulsating, provided that critical structural element. This chronic pulsatile mechanism has been well-documented in earlier non-thrombotic PMA case series [6].

The spectrum of anatomical variants is broad and extends beyond just vascular structures. Muscular anomalies, including the reversed palmaris longus (RPL) or a variant abductor digiti minimi, can physically encroach upon the critical space or induce friction, contributing to MN or ulnar nerve compression [16, 17]. Georgiev and Jelev [18] described a rare coexistence of a variant abductor digiti minimi and an RPL, demonstrating how multiple anomalies can predispose a patient to dual nerve entrapment. The clinical risk posed by these soft tissue variants parallels that of the PMA: preoperative misidentification can lead to an unaddressed root cause or inadvertent surgical injury. This unified view of variant etiology – be it vascular or muscular – strongly mandates a comprehensive diagnostic protocol [14, 25].

The excellent clinical outcome, achieved solely through surgical release and meticulous preservation of the patent PMA in situ, is noteworthy. The successful result confirms that releasing the transverse carpal ligament was the definitive therapeutic maneuver. This outcome suggests that simple decompression of the confining tunnel is sufficient to treat chronic pulsatile compression, negating the need for complex transposition or excision when the artery is patent and non-aneurysmal. This conservative surgical approach is aligned with case reports where simple decompression was enough, even in the presence of this anomaly [4, 19]. Placing this case within the framework of anatomical variants, including the RPL [16, 17] and accessory slips, highlights that the orthopedic surgeon must maintain a broad perspective on structural etiology [14, 25, 18]. Any space-occupying or friction-inducing variant must be identified preoperatively for safe and definitive treatment. In Table 1, we briefly present different anatomical variations that are enrolled as predisposing factors for CTS.

Table 1. Anatomical variations as predisposing factors for CTS

Vascular	PMA [6, 7, 21]	Chronic pulsatile compression (patent artery); acute mass effect (thrombosis or aneurysm); high iatrogenic risk (laceration during blind surgery).
Neural	Bifid or trifid MN, Motor branch variations [8, 10]	Increases total neural tissue volume in fixed space (volume-pressure mismatch); can lead to internal mechanical constraint.
Tendinous/ Muscular	RPL, accessory lumbrical muscle, variant abductor digiti minimi slips, palmaris profundus [14, 16, 18]	Mass effect due to occupying space; dynamic friction or localized pressure during wrist/finger movement.

The indispensable role of ultrasonography

The presence of a patent PMA poses a severe surgical challenge. Performing a standard or simple open surgical release without prior knowledge of this vessel places it at high risk of iatrogenic transection, which could lead to massive bleeding or distal hand ischemia [4, 20].

Our case forcefully illustrates the inherent risk created by the omission of preoperative ultrasonography in the diagnostic pathway. While electrodiagnostic studies confirmed the existence and severity of the neuropathy, they failed to establish the underlying structural cause, which is paramount for surgical planning.

The ability of high-resolution ultrasonography is unique: it not only confirms nerve swelling but also directly visualizes the PMA and confirms its patency [21, 23]. Its utility extends to revealing subtle functional changes and precisely localizing the structural conflict [14]. Preoperative ultrasound would have provided the surgeon with the critical knowledge needed for:

- Planning a modified, safer incision [20].
- Formulating a predefined plan for preservation rather than a reactive, potentially dangerous intraoperative decision.
- Avoiding iatrogenic injury to this vessel, which could otherwise lead to hand ischemia.

Beyond safety, preoperative ultrasound offers prognostic value. Specific sonographic findings, such as the cross-sectional area and nerve mobility, correlate with the anticipated therapeutic response [23, 24]. In our case, identifying a patent PMA before surgery would have enabled the safest execution of the release and preservation technique. The continued use of ultrasound post-operatively confirmed the successful decompression and the vascular status of the preserved artery, validating the surgical outcome.

The current medical literature, including recent Bulgarian contributions, strongly supports a methodical approach to CTS diagnosis and surgical anatomy [25]. Comprehensive reviews underscore the essential need for clinicians to be fully versed in the diverse structural and neural variants that complicate the carpal tunnel, emphasizing that this knowledge is foundational to safe operative management [25]. Furthermore, a strong diagnostic index should be maintained for structural causes, especially in cases presenting a history suggestive of compression or non-response to conservative therapy [15].

CONCLUSION

Large PMA is a rare and hazardous anatomical variant that should be proactively excluded in patients with CTS. Due to the significant diameter of PMA, it could be speculated as a possible factor for CTS. This case, despite being successfully managed through release and preservation without transposition, serves as a powerful cautionary tale: the failure to perform preoperative high-resolution ultrasonography in CTS exposes the patient to unnecessary risk of major vascular complications during routine carpal tunnel release. Preoperative imaging remains the only non-invasive tool that guarantees the safe and planned management of this critical vascular anomaly.

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