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Original Article

Anterior interosseous nerve: anatomical study and clinical implications☆

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ABSTRACT

Objective: The goal of this study was to describe anatomical variations and clinical implications of anterior interosseous nerve. In complete anterior interosseous nerve palsy, the patient is unable to flex the distal phalanx of the thumb and index finger; in incomplete anterior interosseous nerve palsy, there is less axonal damage, and either the thumb or the index finger are affected.

Methods: This study was based on the dissection of 50 limbs of 25 cadavers, 22 were male and three, female. Age ranged from 28 to 77 years, 14 were white and 11 were non-white; 18 were prepared by intra-arterial injection of a solution of 10% glycerol and formaldehyde, and seven were freshly dissected cadavers.

Results: The anterior interosseous nerve arose from the median nerve, an average of 5.2 cm distal to the intercondylar line. In 29 limbs, it originated from the nerve fascicles of the posterior region of the median nerve and in 21 limbs, of the posterolateral fascicles. In 41 limbs, the anterior interosseous nerve positioned between the humeral and ulnar head of the pronator teres muscle. In two limbs, anterior interosseous nerve duplication was observed. In all members, it was observed that the anterior interosseous nerve arose from the median nerve proximal to the arch of the flexor digitorum superficialis muscle. In 24 limbs, the branches of the anterior interosseous nerve occurred proximal to the arch and in 26, distal to it.

Conclusion: The fibrous arches formed by the humeral and ulnar heads of the pronator teres muscle, the fibrous arch of the flexor digitorum superficialis muscle, and the Gantzer muscle (when hypertrophied and positioned anterior to the anterior interosseous nerve), can compress the nerve against deep structures, altering its normal course, by narrowing its space, causing alterations longus and flexor digitorum profundus muscles.

☆ Study conducted at Faculdade de Ciências Médicas e da Saúde, Pontifícia Universidade Católica de São Paulo (PUC-SP), Sorocaba, SP, Brazil.

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Nervo interósseo anterior: estudo anatômico e implicações clínicas

Palavras-chave:
Músculo esquelético/inervação
Nervo mediano
Síndrome compressiva
Pronação

RESUMO

Objetivo: Analisar as relações anatômicas e as variações do nervo interósseo anterior e suas implicações clínicas. A paralisia completa do nervo interósseo anterior resulta na incapacidade de fletir as falanges distal do polegar e indicador; na incompleta, ocorre menor dano axonal e apenas o polegar ou o indicador são afetados.

Método: Este estudo baseou-se na dissecção de 50 membros de 25 cadáveres, 22 eram do sexo masculino e três do feminino. A idade variou entre 28 e 77 anos, 14 da etnia branca e 11 não branca; 18 foram preparados por injeção intra-arterial de uma solução de glicerina e formol a 10% e sete foram dissecados a fresco.

Resultados: O nervo interósseo anterior originou-se do nervo mediano em média de 5,2 cm distal à linha intercondilar. Em 29 membros, originou-se dos fascículos nervosos da região posterior do nervo mediano e em 21 membros, dos fascículos posterolaterais. Em 41 membros, o nervo interósseo anterior posicionava-se entre as cabeças umeral e ulnar do músculo pronador redondo. Em dois membros, observou-se a duplicação do nervo interósseo anterior. Em todos os membros, registramos que o nervo interósseo anterior se desprendia do nervo mediano proximalmente à arcada do músculo flexor superficial dos dedos. Em 24 antebraços a ramificação do nervo interósseo anterior ocorreu proximalmente à arcada do músculo flexor superficial dos dedos em 26, distalmente.

Conclusão: As bandas fibrosas formadas pelas cabeças umeral e ulnar do músculo pronador redondo, a arcada fibrosa do músculo flexor superficial dos dedos e o músculo de Gantzer, quando hipertrofiado e posicionado anteriormente ao nervo interósseo anterior, podem comprimir o nervo contra estruturas profundas, alterar seu curso normal, por estreitar o espaço de sua passagem, causar alterações no músculo flexor longo do polegar e no flexor profundo dos dedos da mão.

Introduction

The anterior interosseous nerve (AIN) emerges on the posterior surface of the median nerve in different locations. At its origin, it is initially positioned parallel to the median nerve; more distally, it lies in the interval between the flexor pollicis longus (FPL) laterally, and the flexor digitorum profundus (FDP), medially, sending branches to these two muscles. It has a constant branch to the flexor indicis profundus and partially supplies the flexor digitorum profundus of the middle finger. The flexor digitorum profundus of the other fingers is supplied by the ulnar nerve. The AIN, after branching to the FDP and FPL, follows along the anterior interosseous artery, resting on the anterior face of the interosseous membrane and distally innervating the pronator quadratus (PQ) muscle. Its thinner terminal branch passes through the dorsal aspect of the PQ muscle, sending sensory branches to the carpal joints.[1] However, there is considerable variation in the proportion in which the median and ulnar nerves supply the flexor digitorum profundus.[1–3]

As the AIN is deeply located, it is protected by several structures, which make lesions to it rare; however, while these structures protect the AIN, they can be causes of its compression. The AIN can be compressed by the Struthers ligament; bicipital aponeurosis; fibrous arches between the humeral and ulnar heads of the pronator teres muscle (PT); the fibrous arch formed by the origins of the flexor digitorum superficialis (FDS) muscle; presence of anomalous muscles such as the Gantzer muscle; vascular changes, such as thrombosis or vessel hypertrophy that cross the nerve; tumor formations; cysts; hematomas; abscesses; iatrogenesis in fracture reduction or drugs injected in the forearm; and trauma, such as supracondylar fracture of the humerus and the proximal third of the forearm.[3,4]

AIN compressive syndrome is a rare compressive neuropathy of the upper limb. It was first described by Parsonage and Turner[5] in 1948, and later by Kiloh and Nevin[2] in 1952. It is characterized by the inability to flex the distal interphalangeal joints of the thumb and index finger, causing an inability to make a pulp pinch, hyperextension of the distal interphalangeal joint, and flexion of the proximal interphalangeal joint; in the thumb, there is flexion of the metacarpophalangeal joint and hyperextension of the interphalangeal joint, which results in a contact area of the thumb pulp with the indicator much more proximal than normal.[6] In incomplete AIN, less axonal damage is observed and only the flexion of the distal phalanx of the thumb or index finger is compromised. PQ muscle impairment can be demonstrated by resisted active pronation of the forearm with a fully flexed elbow to neutralize PT muscle action. No sensory deficits are observed in the clinical evaluation of the hand and forearm.[3,4]

It is difficult to determine the etiology of AIN syndrome precisely because there are no signs or clinical tests that can

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