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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.

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Nervo interósseo anterior: estudo anatômico e implicações clínicas

R E S U M O

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

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

indicate whether it is a compressive neuropathy or a brachial plexus neuritis. Kiloh and Nevin[2] have proposed that it is caused by AIN neuritis; in contrast, Fearn and Goodfellow[7] have suggested that it is a compressive neuropathy, and both hypotheses remain widely accepted. AIN syndrome has been increasingly understood as a neuritis and often resolves spontaneously after prolonged observation.[4,8,9] In contrast, those who considered it to be a nerve compression advocate treatment of the condition by surgical exploration and decompression of the nerve.[10]

This study was aimed at analyzing the anatomical relationship and the variations of the AIN, and its clinical implications, such as compressive neuropathy.

Material and methods

This study was based on the dissection of 50 limbs from 25 cadavers, 22 males and three females. Age ranged from 28 to 77 years; 14 of the cadavers were white and 11 were non-white. Of the total, 18 were prepared with an intra-arterial injection of glycerin and 10% formaldehyde, while seven specimens were freshly dissected cadavers. Cadavers whose forearms were deformed by traumas, malformations, and scars were excluded. No cases of muscular atrophy in the forearms were observed. Each forearm was dissected with the elbow in extension, wrist in neutral position, and forearm in supination. The skin, subcutaneous muscle, and fascia of the flexor surface of the distal third of the arm, forearm, and wrist were removed, thus exposing all the musculature. The median nerve was identified in the arm and dissected in a proximal to distal direction. The bicipital aponeurosis was sectioned. The pronator's superficial head was distally disinserted and retracted. The tendons of the flexor carpi radialis and palmaris longus muscles were sectioned in their distal third, after identification of their nerve branches. The AIN and its nerve branches to the FPL, FDP, and PQ muscles were dissected after the longitudinal division of the FDS muscle and its fibrous arch. The vascular structures were not preserved to facilitate nerve dissection. The origin of each muscle branch of the median nerve was measured from the intercondylar line. The ratios of AIN with the humeral and ulnar heads of the PT muscle and with the arcade of the FDS muscle were measured, as well as the branches of the AIN. These dissections also aimed at identifying the anatomical sites that may be responsible for the compression of the median and anterior interosseous nerves, such as the Struthers ligament, bicipital aponeurosis, and the site between the humeral and ulnar heads of PT muscle and the fibrous arch of the FDS muscle. The presence of the Gantzer muscle and of the Martin Gruber anastomosis were recorded. In certain phases of the dissection, a 2.5× magnifying glass was used. The anatomical variations were registered and photographed. This study was approved by the Ethics Committee under opinion No. 1,611,295.

Results

In the present study, the AIN detached from the median nerve at a distance of 1.5 to 7.5 cm, at a mean of 5.2 cm distal to the intercondylar line. In 29 limbs, it originated from the nerve
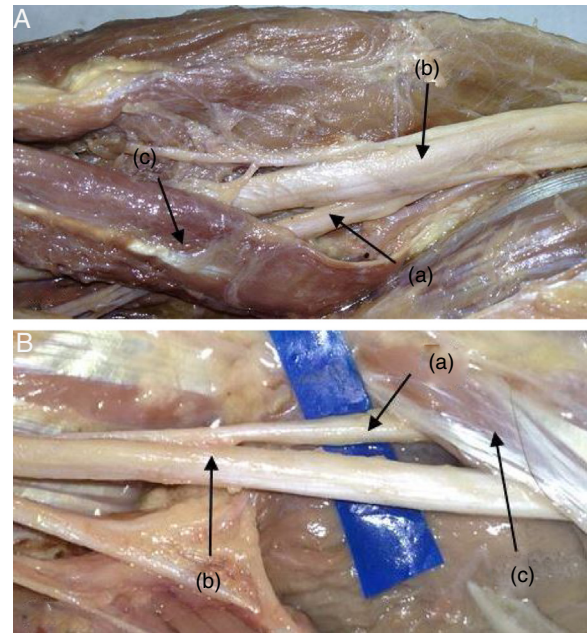

Fig. 1 – (A) In 29 limbs, the AIN (a) originated from the nerve fascicles of the posterior region of the median nerve (b). PT muscle (c); (B) in 21 limbs, the AIN fascicles (a) originated from the posteromedial fascicles the median nerve (b). FDS muscle arch (c).
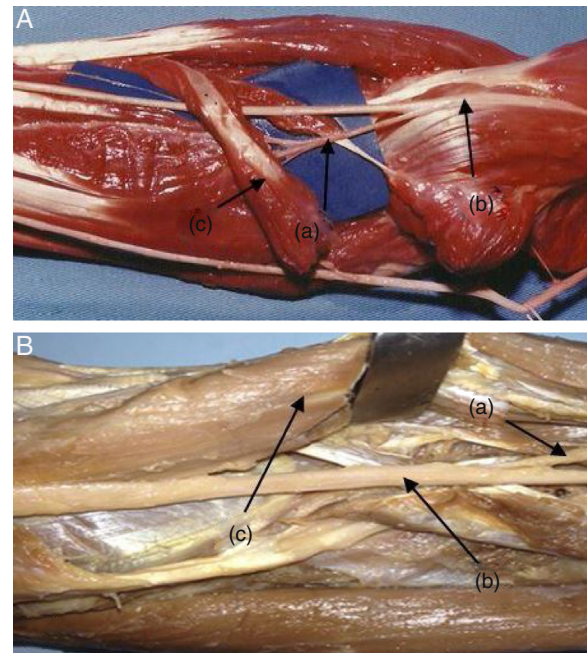

Fig. 2 – (A) AIN (a) branched off the median nerve (b) proximally to the PT muscle (c) in 12 limbs; (B) AIN (a) branched off the median nerve (b) distally to the PT muscle (c) in six limbs.

fascicles of the posterior aspect of the median nerve (Fig. 1A), and in 21 limbs, from the posterolateral fascicles (Fig. 1B). In 12 limbs, the AIN detached from the median nerve proximally to the PT muscle (Fig. 2A), and in six, distally to it (Fig. 2B). In
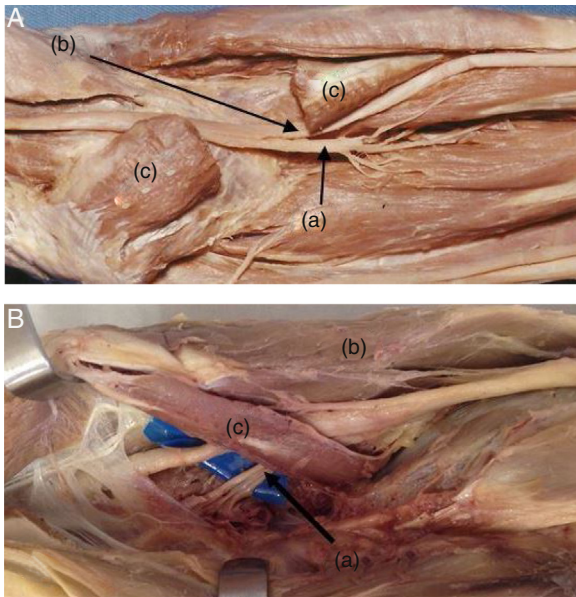
Fig. 3 – (A) In 32 limbs, the AIN (a) branched off the median nerve (b) under the muscle mass of the PT muscle (c); (B) in the vast majority of cases, the AIN (a) was positioned between the humeral (b) and ulnar (c) heads of the PT muscle.
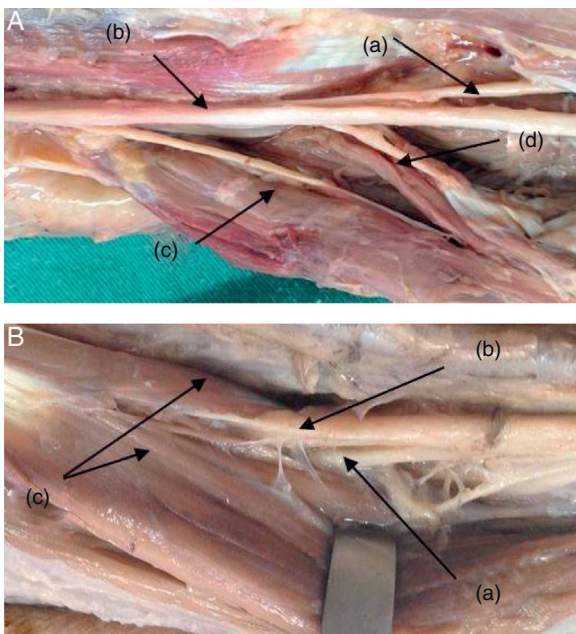

Fig. 4 – (A) In three limbs, the AIN (a) and median nerve (b) were positioned posteriorly to the humeral (c) and ulnar (d) heads of the PT muscle, which were disinserted and retracted laterally; (B) AIN (a) and median nerve (b) cross the humeral head (c) of the PT muscle.

32 limbs, it was detached from the median nerve under the PT muscle mass (Fig. 3A). In most cases (41 limbs), the AIN was positioned between the humeral and ulnar heads of the PT muscle (Fig. 3B). In two limbs, the AIN was positioned posteriorly to the two heads of the PT muscle (Fig. 4A). In the seven
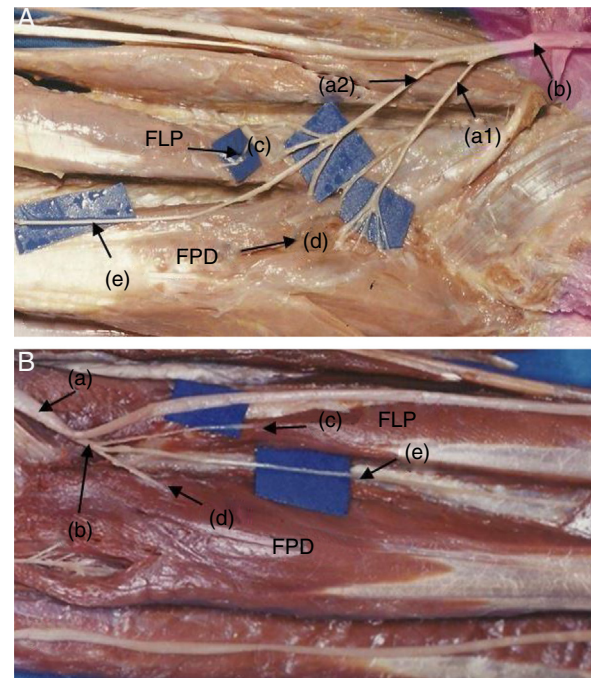

Fig. 5 – (A) Duplication of the AIN, the proximal branch (a1), and the distal branch (a2). Median nerve (b) FPL (c), FDP (d). Branch to the PQ (e); (B) median nerve (a). AIN (b). A branch to the FPL (c) and one to the FDP (d). Branch to the PQ (e).

limbs in which the ulnar head was not present, it was observed that the AIN was positioned posteriorly to the humeral head in five limbs (Fig. 3A) and passed through it in two (Fig. 4B). In one limb, it was observed that the AIN was doubled. The proximal branch detached from the median nerve 4 cm distally to the epicondylar line, supplying the FDP; in turn, the distal branch emerged 1.0 cm below it, supplying part of the FDP and FPL, and was directed toward the PQ muscle and the carpal joint (Fig. 5A). In another limb, the branches to the FDP and FPL originated separately from the median nerve, with the branching to the FDS muscle between them. In all the dissected limbs, it was observed that the AIN branches off the median nerve proximal to the arch formed between the insertions of the flexor superficialis muscle (Fig. 1B). In 24 forearms, the AIN ramification occurred proximally to the arch (Fig. 3B), and in 26, distally to it (Fig. 1B). The fibrous arch was identified in 32 forearms (Fig. 1). In three forearms, the arch was considered to be irregular, as there was a discontinuity between the fibers that formed the arch. The study also assessed the number of ramifications from the branches to the FPL and FDP muscles that penetrated at different points of the muscles. The presence of one branch to each of the muscles was recorded in six limbs (Fig. 5B); of two branches to the FDP and one to the FPL, in 14 limbs (Fig. 6); of two branches to each muscle, in 14 limbs (Fig. 3A); of three branches to the FDP and one to the FPL, in seven limbs (Fig. 6B); of three branches to the FDP and two to the FDS, in five limbs (Fig. 7A); of three to the FDP and three to the FPL, in three limbs (Fig. 7B); and another six branches to FDP and two to FPL, (Fig. 5A). The longest branch was always to the FPL (Figs. 5A and 6B). The AIN left the median nerve proximally to the median nerve
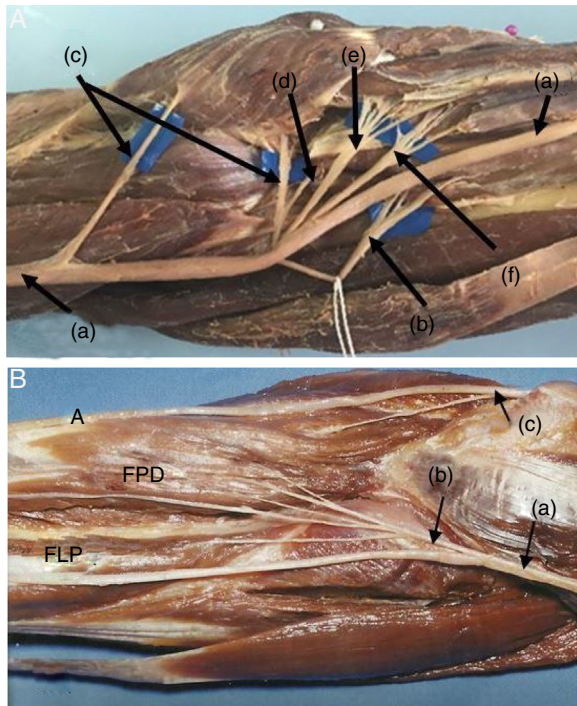
Fig. 6 – (A) Median nerve (a), AIN (b), two branches to the FDP and one to the FPL. Two superfluous branches of the median nerve to the PT muscle (c), branch to the palmaris longus (d), branch to the flexor carpi radialis (e), FDS (f). They can be neurotized to the AIN (d); (B) median nerve (a). AIN (b). Ulnar nerve (c). Three branches to the FDP, and one to the FPL.
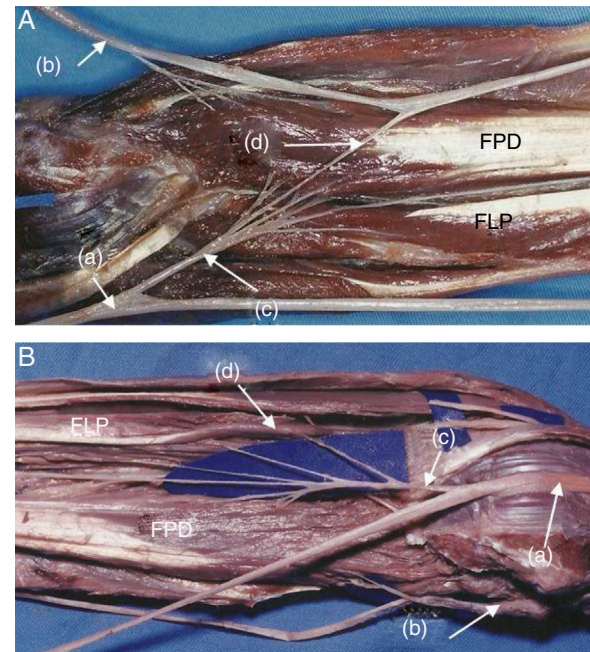

Fig. 7 – (A) Median nerve (a). Ulnar nerve (b). AIN (c). Martin Gruber's anastomosis (d). Three branches of the AIN to the FDP and two branches to the FPL; (B) median nerve (a). Ulnar nerve (b), AIN (c) Three branches of the AIN to the FDP (c) and three branches to the FPL, one directed to the Gantzer muscle (d).

branch to the FDS muscle in 41 limbs (Fig. 8A); in four, at the same level as the branch to the FDS; in three, distally to it; in one, it originated between the AIN branches to the FDP and FPL muscles. In two limbs, it originated from a common trunk with the median branches to the FDS and flexor carpi radialis muscles. In four limbs, the FDS muscle received a branch from the AIN, as well as the innervation from the median nerve (Fig. 8B). In one limb, the presentation was absolutely atypical; the AIN branched off the median nerve, proximally to the intercondylar line and proximally to the innervation of all the muscles of the forearm, including the PT muscle (Fig. 9A). Four limbs presented an expansion of the AIN that provided ramification to the flexor profundus of the fourth finger (Fig. 5B). The Gantzer muscle was observed in 34 forearms, and in nine cases this muscle was positioned anteriorly to the AIN (Fig. 9B). In all the forearms, the Gantzer muscle received exclusive innervation from the AIN. Martin Gruber's anastomosis was identified in 13 forearms, and in eight the nerve communication occurred between the ulnar nerve and the AIN (Fig. 7A).

Discussion

There is some controversy as to where the AIN leaves the median nerve. Sunderland[1] reported that the AIN left the median nerve between 2.3 and 8 cm distally to the medial
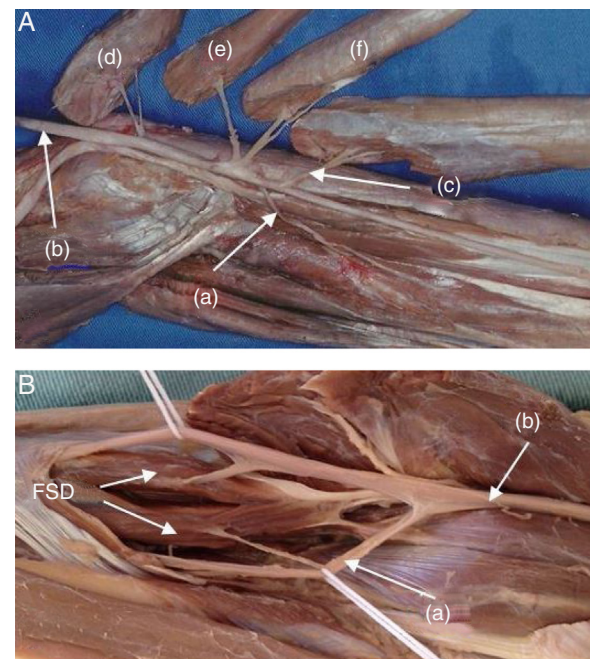

Fig. 8 – (A) The AIN (a) branched off the median nerve (b) proximally to the branch to the FDS (c) into 41 limbs. PT muscle (d). Palmaris longus muscle (e). Flexor carpi radialis muscle (f); (B) in four limbs, the FDS muscle received a branch from the AIN (a) in addition to the innervation of the median nerve (b).
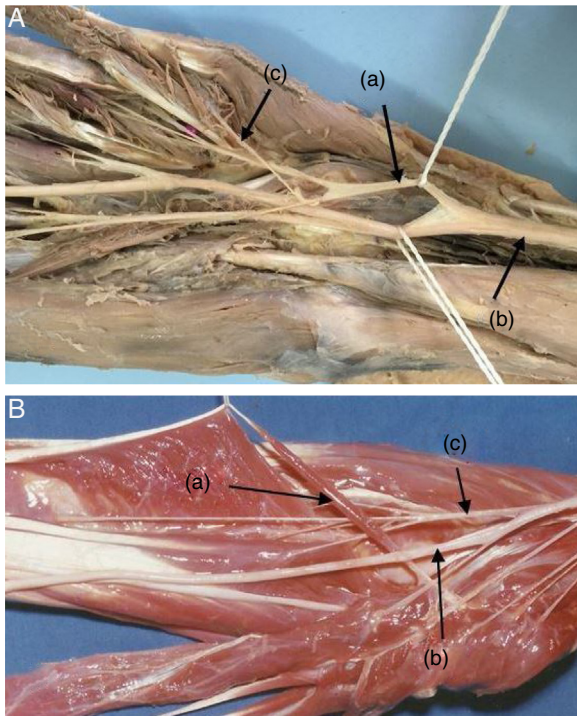
Fig. 9 – (A) Atypical case, AIN (a) branched off the median nerve (b), proximally to the intercondylar line and proximally to the innervation of all muscles of the forearm, including the PT muscle (c); (B) Gantzer muscle (a) is positioned anteriorly to the AIN (b) and posteriorly to the median nerve (c).

humeral epicondyle. Tubbs et al.[11] observed that the AIN originated from the median nerve at a mean of 5.4 cm distally to the joint line. According to Freiberg,[12] the AIN left the median nerve 8 cm distally to the medial epicondyle. Vincelet et al.[13] dissected 35 cadaveric limbs and observed that the AIN originated on average 45 mm below the intercondylar line. In the present study, the AIN left the median nerve at a distance of 1.5–7.5 cm, at a mean of 5.2 cm distally to the intercondylar line.

Sunderland[1] and Tubbs et al.[11] reported that the fascicles that formed the AIN were posteriorly or posterolaterally located in the median nerve and that a group of nerve fascicles separated from the median nerve had already formed approximately 2.5 cm before the emerging site. Dellon and Mackinnon[9] observed that the AIN originated from the posterior aspect of the median nerve in 12 of 31 limbs and from the posterolateral aspect, in 19. In the present study, the nerve originated in the posterior fascicles in 29, and in the posterolateral fascicles in 21. The present authors had the same impression as Dellon and Mackinnon[9] that the AIN is more susceptible to compression when it originates from the lateral aspect, and that it is more protected from fibrous arches when it is located posteriorly. Botte[14] reported that the AIN originates 5–8 cm distal to the medial epicondyle, just below the arch of the FDS muscle; the present results are not in agreement with this statement, since the AIN was not observed to branch off from the medial nerve distally to the FDS muscle

arch in any of the studied limbs. Tubbs et al.[11] stated that AIN can be compressed by the fibrous arch between the insertions of the FDS muscle. In the present study, in 32 of 50 forearms, the fibrous arch was supported on the median nerve and the AIN; this could be the cause of nerve compression. Park et al.,[10] in a study with 11 patients with spontaneous AIN syndrome treated by surgical exploration, observed that the most common structure responsible for nerve compression was a fibrous arch of the FDS muscle. Dellon and Mackinnon[8] identified that, in 16 of 31 limbs, the AIN was related to fibrous arches. In three, the fibrous arches were formed only by the arch of the FDS muscle; in two, by the fibrous arches of the ulnar head of the PT muscle; in three, by the humeral head of the PT muscle; and in eight, by the combination of fibrous arches formed by the arch of the FDS muscle and by the humeral and ulnar heads of the PT muscle. Guo and Wang[15] reported that the AIN branched distally to the arch of the FDS muscle in 74% of the forearms, but those authors did not mention the frequency with which the AIN left the median nerve distally to the arcade of the FDS muscle. In their study, the AIN originated from the ulnar side of the median nerve in only 8% of the cases; those authors recommend that the surgical decompression of the arch should be done on the ulnar side of the median nerve, thus preserving the branches of the AIN. In contrast, Tubbs et al.[11] reported that the AIN originated proximally to the arch of the FDS muscle in all the 60 limbs assessed. The present findings confirm those by Tubbs et al.,[11] as the AIN originated proximally to the arch in all forearms, often very close to the arch but not distally to it. The AIN ramification was proximal to the arch in 26 limbs and distal to it in 24.

There are controversies in the literature regarding the relationships between AIN and PT muscle; some report that the AIN originates distally from the humeral and ulnar heads of the PT muscle, while others have described it as originating between the two heads of that muscle. Tubbs et al.[11] reported that they always observed the AIN positioned posteriorly to the humeral head of the PT muscle; if the ulnar head is present, the AIN lies between them. Chidgey and Szabo[16] reported that the AIN may be positioned posteriorly to the two heads of the PT muscle. Johnson et al.[17] dissected 40 preserved cadavers; in 90% of the dissections, the AIN branched off the median nerve distally to the PT muscle; in only 10% of the dissections, did the AIN branch off the median nerve under the PT muscle, and no separation between the AIN and the median nerve proximal to the PT muscle was observed. In 41 limbs assessed in the present study, the humeral and ulnar heads of the PR muscle were well partitioned as two distinct structures. In 39 limbs, the AIN was positioned between the humeral and ulnar head of the PT muscle; in two limbs, it was positioned posteriorly to the two heads of the PT muscle. In seven limbs, the ulnar head was absent; in five of these, the AIN was positioned posteriorly to the humeral head, and in two it passed through it. The AIN originated proximally to the PT muscle in 12 limbs, distally to it in six limbs, and under it in 32 limbs.

Regarding the anatomical variations of the typical AIN innervation, Chidgey and Szabo[16] reported that the extension of the innervation to the flexor digitorum profundus of the middle finger was observed in approximately 50% of the cases.

Kiloh and Nevin[2] consider that there is a considerable variation in the proportion that the median and ulnar nerves supply the flexor digitorum profundus of the fingers. These authors described two clinical cases of AIN involvement; in the first, there was paralysis of the FPL and FDP, and in the second case, only the FPL was paralyzed. Ulrich et al.[3] reported that seven of their 14 patients presented isolated total paralysis of the FPL function; in five, the FPL and the deep flexor of the indicator were paralyzed, and in two patients, only the index flexor was impaired. None of their patients had total paralysis of the flexor digitorum profundus of the middle finger, but in four cases there was some loss of flexion force of the finger. Two patients presented a decrease in pronation force. Botte[14] described that in all of the medial hand, the AIN also supplies the flexors of the fourth and fifth fingers. In the present study, in five limbs it was possible that the AIN could supply the third and fourth fingers, but due to their syncytial nature, it is difficult to make this affirmation based only on the dissections. Sunderland[1] identified a branch of the AIN to the FDS muscle in 30% of cases. In the present study, the FDS was innervated by the AIN and by the median nerve, in four limbs. Out of the 13 limbs where Martin-Gruber's anastomosis was identified, in eight a communication was observed between the AIN and the ulnar nerve; in these cases, it is possible that motor nerve fibers from the AIN innervate the intrinsic muscles of the hand usually innervated by the ulnar nerve.[6,18]

Dellon and Mackinnon[9] reported that a hypertrophied Gantzer muscle may compress the AIN. They report that, in the most distal part of its course, the nerve can cross posteriorly to the tendon of the Gantzer muscle and thus compress the branch of the AIN to the PQ muscle, causing weakness at forearm pronation. Those authors also reported that a hypertrophied Gantzer muscle, even passing anteriorly to the AIN, may compress the nerve between the Gantzer muscle and the musculoaponeurotic structures of the PT muscle and FDS. In the present study, the Gantzer muscle was identified in 34 limbs; the authors speculate that a hypertrophied Gantzer muscle can cause AIN syndrome, but this was an infrequent finding, as this hypertrophy was only observed in three limbs. Tabit et al.[19] reported the clinical case of a patient with incomplete AIN and paralysis only of the FPL, which was interoperatively proven to be caused by the Gantzer muscle. The present findings indicated that in nine of the limbs, the AIN crossed only the branches to the FPL or FDP; in these cases, it could only cause incomplete AIN syndrome, which can be misdiagnosed as a tendon injury.[20] Digital pressure along the course of the musculotendinous component produces a slight flexion of the interphalangeal joint and confirms the integrity of the tendon.

Conclusion

The fibrous arches formed by the humeral and ulnar heads of the PT muscle, the fibrous arch of the FDS muscle, and the Gantzer muscle when hypertrophied and positioned anterior to the AIN can compress the nerve against deep structures, changing its normal course by narrowing the space of its passage, and cause alterations in the FPL and FDP muscles.

Conflicts of interest

The authors declare no conflicts of interest.

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