Accessory Abductor Digiti Minimi- A Case Report

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Abstract: The accessory ADM is the most common anomaly among all the accessory hypothenar muscles, with a prevalence of 24%, and present bilaterally in 50% of the cases. During routine dissection of undergraduates we have come across the presence of this accessory muscle in the wrist when the carpal tunnel is opened. This presents with Abductor Digiti Minimi (ADM) muscle bilaterally, involving ulnar artery (UA) and ulnar nerve (UN) distribution.

Key words:- Hypothenar, Accessory, Abductor digiti minimi, ulnar artery, ulnar nerve.

I. Introduction

With respect to the possible Ontogenic interpretation of the anamoly, it is first to be noted that original undifferentiated mesenchyme into 2 layers, superficial and deep. 3 primordia are developed radial, medial and ulnar. Medial – flexor digitorum superficialis, radial- abductor policis brevis, ulnar- ADM. These arise from superficial layer. The rest develop from deep layer at a later stage. FDS then extends from its present position to fore arm and reaches its adult position. With this ontogenic origin of muscles it is considered that these accessory fascicles are due to defect in migration above and not in super numerary origin of antebrachial muscles. Although muscular, vascular and nervous variations of the hypothenar eminence are rare (differing from anomalous muscles in the wrist), they have been reported [1-10]. According to Sheppard, three cases of accessory ADM were first reported by Wood in 1868. Most authors call such additional ADM head an "accessory" component which can be unilaterally or bilaterally presented, producing compression in Guyon's canal. Harvie et al., found an accessory ADM in 41% of 116 volunteers' asymptomatic ultrasound examinations, greater prevalence being found in males and bilateral presentation in 50% of cases for both genders. The flexor digiti minimi brevis muscle can also be present in this area; Madhavi et al., have stated that it relates to the common phylogeny of these muscles from the same muscle mass. Murata [7] found an accessory ADM in 35 hands, having one (17%), two (80%) and three fascicles (3%). Furthermore, Wulle [8] presented eleven cases of accessory ADM, but having "longus" presentation.

II. Case Report

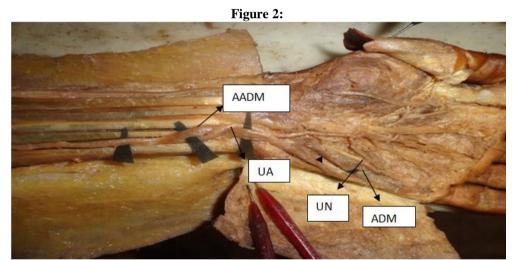
This case is reported in Department of Anatomy, Andhra Medical College, Visakhapatnam; during routine dissections. The specimen was fixed in 10% formaldehyde solution. Dissections were performed by the authors, involving the antebrachial and hand dorsal and ventral area. The hypothenar area was carefully dissected following vascular and nervous structures neighbouring the ADM's accessory head. These findings were recorded and photographed (digital camera) according to UN side and collateral origin, UA vascular distribution and muscular variation. The Right hand (Figure 1.) presented an accessory ADM head originating in antebrachial fascia. According to Murata et al., [7] classification resembles "variation 2", however without additional palmaris longus tendon origin. It passed obliquely through Guyon's canal, enclosing the UN and vessels.



Rt hand: AADM : Accessory abductor digiti minimi, PL: Palmaris Longus, UA: Ulnar artery, ADM: Abductor digiti minimi. AADM originating from Antebrachial fascia.

The accessory head had a late union in its distal trajectory to form a single pennate with the ADM originating in pisiform bone. It was localized over the flexor digiti minimi brevis muscle and lateral to Opponens Digiti Minimi muscle. Length was 10cms, 2mm and 2.43 mm thick compared to the ADM. The head was 5cms, 4mm wide and 7.09 mm thick. This right accessory ADM head had 34.3% of the thickness of a usual ADM head. UA is seen crossing it at the level of Guyon's canal, and UA was covered by this ADM accessory head. Sensorial UN branches had both superficial and deep branches regarding muscular variation and were presented in relation to them. Sensory branch coming from the ulnar nerve's dorsal cutaneous branch and a deep sensory branch coming from the ulnar trunk. They were connected after coursing the accessory ADM head. A third sensorial trajectory course was observed toward the fourth finger after passing Guyon's canal as the "fourth common digital nerve".

The left hand (Figure2) also presented an accessory ADM head originating in the ante brachial fascia. It also passed obliquely through Guyon's canal, enclosing the UN. This accessory head also had a late union in its distal trajectory to form a single pennate with the ADM originating in pisiform bone. The origin was found to be at 2.5 cms from the flexor retinaculum. The Ulnar Artery was seen first passing above it in the tunnel and later became superficial and formed the superficial arch without joining with any of the branches of radial artery as it is usual. The left hand muscle length was 10cms, 1.5mm wide and 1 mm thick compared to the usual ipsilateral. ADM head which was 5cms, and of same thickness as on other side. This accessory ADM head had 45.8% of the thickness of a usual ADM head. The UA adopted a superficial course above the accessory ADM head while the UN and all its sensorial branches were covered by this variation. These accessory ADM heads received deep UN branch motor innervations on both sides. Right -hand hypothenar zone. ADM: abductor digiti minimi muscle. AH: accessory head of abductor digiti minimi muscle. UA: ulnar artery. FR: flexor retinaculum. AF: antebrachial fascia..



Left Hand: AADM: Accessory Abductor Digiti Minimi, ADM: Abductor Digiti Minimi; UA: Ulnar Artery, UN: Ulnar Nerve. Here too originates from AnteBracial Fascia.

This class of muscular variation entrapping neuro-vascular bundles may have sensorial and/or muscular implications in a range of compression neuropathies [5,6]. Entrapment neuropathies can produce heterotopic projected pain, symptomatic consequences arising from mechanical nerve injury passing through a narrow anatomical space or under a muscular structure which may potentially compress the neuro-vascular package. We think that muscle size, tightness and course may be important factors in considering whether such variations are able to produce compression. This scenario can cause nerve oedema and ischemia by muscle structure friction over the peryneuroma which can cause neuritis and raise endoneural pressure. In addition to traumatic neuropathic pain, symptoms in distal nerve distribution (dysesthesia, paresthesia and anaesthesia), paresis, hyporeflex, hypotonicity and atrophy, such as inferior motor neuron lesion, may be equally feasible. The deep UN motor branch may be compressed by this ADM accessory head and compromise palmar and dorsal interosseous muscles, hypothenar eminence muscles, lumbricals III and IV and hallux adductor, thereby producing a clinical "claw hand" appearance or resembling Guyon's canal syndrome.

Having dealt with this variation's possible sensorial and motor effects, vascular effects must also be considered. UA compression (left hand) may produce peripheral vascular disease from two possible events depending on the type of compression (i.e. constant or intermittent muscular compression). Muscular hyperfunction can reduce these arterial branches' distal irrigation, causing hypoesthesia or hyperesthesia in the hand. Likewise, muscular spasm can produce anaerobic metabolism and consequent nerve irritation by anoxic

acidified setting with neurogenic inflammation and peripheral hyperalgesia . Permanent loss of vascular supply during muscular spasm can lead to vascular claudication and pain.

We believe (from a functional perspective) that precipitating factors (gender, occupation, side dominance, traumatic history, anatomical characteristics) may develop neurovascular compression and symptoms in an anomalous muscle; we thus present this anatomical case. The morphological characteristics led to all the above explained symptomatic possibilities; however, lacking clinical judgment, further statements lose their functional value and become merely speculative.

III. Conclusion

In the above case both the AADM of RT and Lt hands originated from ante brachial fascia. But in the previous reports, the accessory ADM muscles originated either from the deep antebrachial fascia or from aponeurosis or Palmaris longus. (Wood, 1868; Roberts, 1972; Tubiana, 1981. Muscular variations compromising neurovascular bundles may produce clinical symptoms such as dysaesthesic pain, sensory loss, wakefulness and paresis, highlighting these anatomical discrepancies' importance when they are presented. However, the existence of an accessory ADM is usually asymptomatic and only rarely results in nerve compression. It does appear that muscle size, tightness and course may be an important factor in considering whether such variation is able to produce UN compression at Guyon's canal or UA compression resulting in a poor relationship between blood-flow and artery diameter. A larger sample of cadaver specimens is needed. Also, further clinical studies are required to confirm the existence of these variations' clinical effects

Referances

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