

Hypothenar Hammer Syndrome. Case Report

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ABSTRACT

Hypothenar hammer syndrome is a thrombosis of the ulnar artery in Guyon's canal induced by repeated trauma. It is a rare disorder that can be diagnosed with an extensive medical history and physical examination, and confirmed by vascular studies. Management options include medical treatment and reconstructive surgery. The aim of this article is to provide a brief overview of this disorder and to discuss the case of a 45-year-old patient who developed paresthesias and signs of hypoperfusion of the fourth and fifth fingers following multiple injuries to the hypothenar eminence. The Allen test revealed the absence of vascularization in the ulnar artery, and thrombosis was verified by Doppler ultrasound and angiotomography. The thrombosed portion was excised, and Guyon's canal was cleared. The evolution was satisfactory; no signs of ischemia were found, and paresthesias improved. Follow-up was performed for 1 year.

Keywords: Hammer; hypothenar; thrombosis; aneurysm; artery; ulnar.

Level of Evidence: IV

Síndrome del martillo hipotenar. Presentación de un caso

RESUMEN

El síndrome del martillo hipotenar es la trombosis de la arteria cubital en el canal de Guyon causada por traumatismos repetitivos. Se trata de un cuadro infrecuente que se diagnostica mediante la detallada valoración de los antecedentes y el examen físico, y se confirma con estudios vasculares. El manejo incluye desde tratamiento médico hasta cirugía reconstructiva. El objetivo de este artículo es brindar una breve reseña de esta enfermedad y presentar el caso de un paciente de 45 años, con parestesias y signos de hipoperfusión del cuarto y quinto dedos luego de múltiples traumatismos en la eminencia hipotenar. En la prueba de Allen, se detectó ausencia de vascularización de la arteria cubital, y la trombosis se confirmó mediante ecografía Doppler y angiotomografía. Se resecó el fragmento trombosado y se liberó el canal de Guyon. La evolución fue satisfactoria, no se observaron signos de isquemia y las parestesias mejoraron. Se realizó un seguimiento por 1 año.

Palabras clave: Martillo; hipotenar; trombosis; aneurisma; arteria, cubital.

Nivel de Evidencia: IV

INTRODUCTION

Hypothenar hammer syndrome is thrombosis of the ulnar artery associated with repetitive trauma to the hypothenar eminence.

Guttani and Von Rosen described the first case in 1934 as a post-traumatic thrombosis of the ulnar artery at the distal level discovered after an operation in an industrial worker, but it was not until 1970 that Conn et al. termed the lesion *hypothenar hammer syndrome* (HHS).¹⁻⁴ It is commonly seen in people who use their palms as hammers, continuously pounding or compressing the ulnar artery against the hook of the hamate in Guyon's canal, where the artery is most vulnerable.

Typically, HHS affects men with an average age of 40 years,² on the dominant hand in 53-93% of cases,⁵ and in occupational settings where the worker uses the hypothenar portion of the hand as a tool for hammering, pushing, or squeezing hard objects. People at greatest risk for this disease include metal workers, auto mechanics, lathe

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operators, machinists, miners, sawmill workers, butchers, bakers, bricklayers and carpenters. Cases have also been described in athletes who practice field hockey and athletics. Occasionally, a single episode of significant trauma may be the cause of HHS.^{1-3,6}

Guyon's canal is formed between the hamate and pisiform bones with only a thin roof above.⁷ Thus, there is an approximately 2-cm section of the ulnar artery that is very vulnerable to acute and chronic trauma.⁸ Frequent blunt trauma to the hypothenar eminence compresses the unprotected ulnar artery against the hook of the hamate and triggers a vasospasm of the artery. Continued trauma causes damage to the tunica intima, which favors platelet aggregation and thrombus formation. Distal embolization of the digital arteries exacerbates ischemia,^{1,7} a phenomenon described in up to 50% of patients.⁹ Less commonly, repetitive blunt trauma results in ulnar artery aneurysm formation. The superficial palmar branch of the ulnar artery provides the main blood supply for most of the fingers and, in 31% of patients, the superficial arch arises entirely from the ulnar artery. In 16-22% of patients, the superficial arch is incomplete.²

It is not uncommon for the initial injury to appear trivial and, consequently, to be ignored,² as it is a rare medical condition that affects less than 1% of the general population.¹⁰ In one study, 7% of 330 factory workers had HHS.⁶

Diagnosis is primarily clinical, and therefore requires a high index of suspicion. Patients usually present with hypothenar pain, cold and pale fingers (3rd, 4th, and 5th), color changes, trophic lesions due to digital ischemia (findings in the fingertips, such as splinter hemorrhages, ulcerations, and gangrene), paresthesias in the ulnar nerve territory, and, on rare occasions, a palpable mass in the hypothenar eminence due to the formation of an aneurysm over this site followed by a thrombus.^{1,3,6,11} The absence of the triphasic color change found in classic Raynaud's phenomenon, which does not affect the thumb, provides a diagnostic clue.¹¹ Hypothenar hypersensitivity and an abnormal Allen test (slow or complete absence of filling of the hand when the radial artery is occluded) help to confirm the diagnosis.^{2,3} Of course, rheumatoid arthritis, Buerger's disease, thoracic outlet syndrome, Raynaud's phenomenon, lupus erythematosus, and scleroderma are all disorders that can cause many of these symptoms and should be considered as well.⁴

Angiography is considered the gold standard for diagnosis, often showing a characteristic "corkscrew" appearance of the affected portion of the artery as it courses along the hook of the hamate.^{2,11} While angiography can show detailed arterial anatomy and is superior to other diagnostic studies in patients with a smaller ulnar artery or an occlusion in the most distal part of the fingers, it is an invasive procedure that may not be available in all medical facilities. Doppler ultrasound, magnetic resonance angiography and CT angiography are also useful tests to confirm the diagnosis.¹

Treatment may vary according to the intensity and speed of onset of symptoms.¹ In mild cases, it involves lifestyle changes (quitting smoking, wearing gloves while working), medications such as calcium channel blockers (nifedipine, diltiazem), antiplatelet agents or anticoagulants, and pentoxifylline to decrease blood viscosity. In more severe cases or when conservative treatment fails, surgery may be required, which consists of arterial ligation (assuming an intact radial/palmar arch), resection of the thrombosed arterial segment or aneurysm with end-to-end anastomosis, and resection and vascular reconstruction with a vein or artery graft or thrombolysis.^{1-3,11}

The purpose of this article is to provide a brief overview of this disease and to report a clinical case of acute-onset HHS caused by ulnar artery thrombosis, which was treated with excision of the affected segment.

CLINICAL CASE

A 45-year-old right-handed man with no significant medical history, a mechanic by trade, presented to the Emergency Department with numbness in the 4th and 5th fingers of his left hand after a day of work in which he impacted with the heel of his hand on multiple occasions while working, four days prior to the consultation. He did not report smoking or alcohol consumption, nor a family history of hyperviscosity syndrome, nor previous sensitivity disorders.^{3,9}

He had no deformities or swelling of the fingers, hand or wrist. Passive and active range of motion was complete. He presented with severe pain (9 out of 10 on palpation) in the hypothenar eminence and a small pulsatile hypothenar mass was observed.⁵ Paresthesias were detected at the palmar level of the 5th and 4th fingers, and sensation in the dorsum of the hand was preserved. He had no signs of median nerve compression in the carpal tunnel, negative Tinel's sign in the cubital tunnel, and positive in Guyon's canal. Finger separation against resistance (interosseous

muscles) was not limited, and Froment's sign was absent.¹ Raynaud's phenomenon was observed in the 4th and 5th fingers (without erythematous phase)² with distal cyanosis, no symptoms in the thumb and index finger, and pallor in the middle finger (Figure 1). All three ulnar fingers were cold. Allen's test was positive in the left wrist, without vascularization of the ulnar artery.¹²



Figure 1. Image of the first day of acute symptomatology. Signs of ischemia in the 4th and 5th fingers.

Complementary studies

Radiographs and CT scans were negative (no hamate fracture).³ Additional blood samples were taken to rule out rheumatic and collagen diseases or vasculitis. Likewise, the evaluation by the Rheumatology physicians did not detect any disease.

An electromyography of the upper limb showed decreased conduction velocity of the left ulnar nerve in Guyon's canal, with increased distal latency velocity, suggesting ulnar neuropathy at the left wrist.¹⁰

A bilateral arterial Doppler scan of the upper limbs performed during the initial days of hospitalization revealed no atheromatous calcifications of the ulnar artery at the level of Guyon's canal, but did reveal tunica intima thickening and distal monophasic flow (Figure 2). CT angiography was requested and confirmed the diagnosis (Figure 3), as well as the presence of the superficial palmar arch (Figure 4).

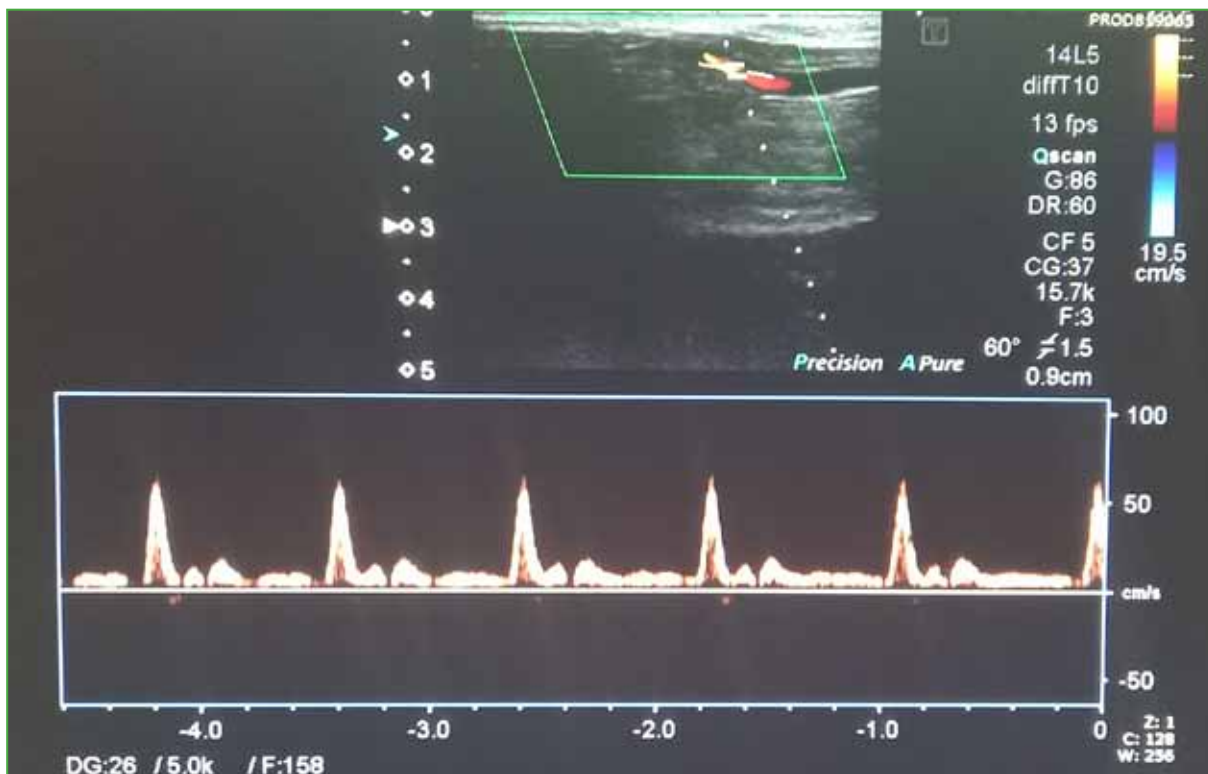


Figure 2. Doppler ultrasound of the ulnar and radial arteries. Decreased flow is observed in the ulnar artery, which is monophasic in the distal region of the wrist.



Figure 3. CT angiography of the left hand. Common palmar digital arteries and princeps pollicis artery (red arrows). The deep palmar arch is intact (green arrow).

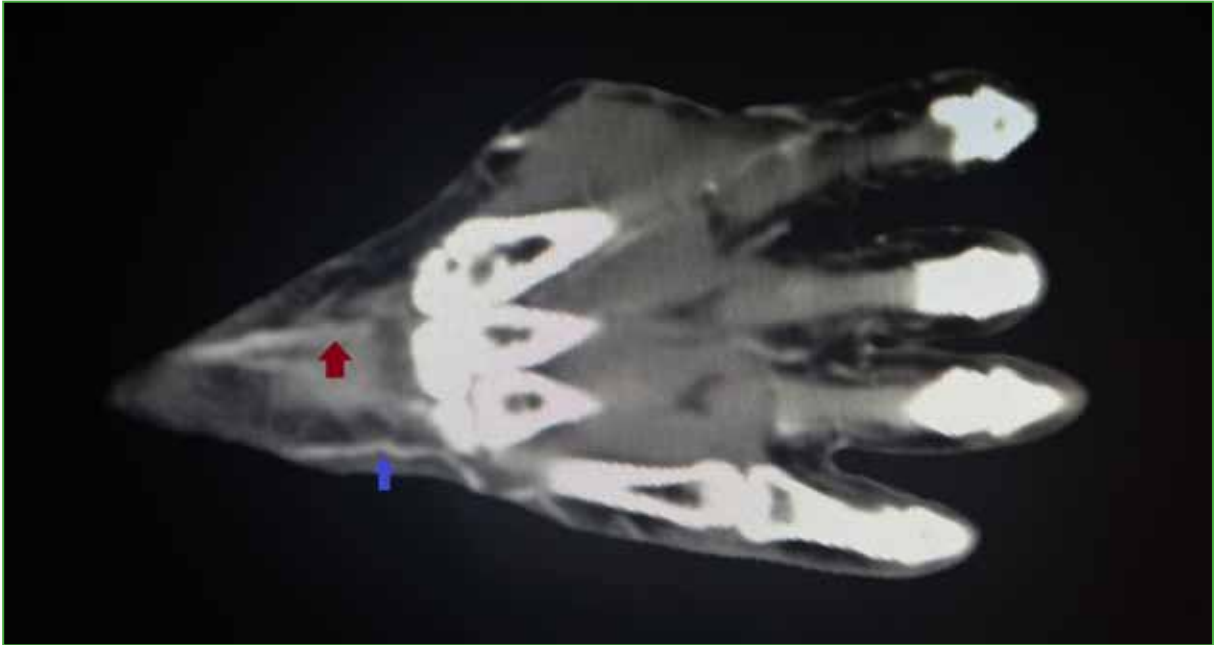


Figure 4. CT angiography of the left hand. Ulnar artery with stenosis and flow that stops in Guyon's canal (blue arrow). Radial artery with good diameter and flow (burgundy arrow).

Medical treatment

The patient was hospitalized for further studies, control, and pharmaceutical treatment. He was given cilostazol 100 mg/day, nimodipine 30 mg, every 6 h; enoxaparin 80 mg, subcutaneously, every 12 h; aspirin 100 mg/day, pregabalin 75 mg/day, ketorolac 60 mg/day and tramadol 100 mg/day.

He was monitored for one week: signs and symptoms improved, pain decreased to 2 out of 10, finger perfusion improved, with reduced capillary refill time. The patient was discharged with outpatient management (Figure 5).



Figure 5. Images taken at the time of discharge. Improved coloration and capillary filling.

At the time of discharge, Allen's test was positive for the ulnar artery in the left wrist, Tinel's test was positive in Guyon's canal and he had paresthesias in the volar region of the ulnar border of the ring finger and in the pinkie finger; for this reason, surgical treatment was decided.

Surgical resolution

The surgery was performed as an outpatient procedure under axillary brachial plexus block, with the patient in dorsal decubitus and the arm on a surgical table. A distal antebrachial and palmar approach was made to the ulnar artery, the artery was identified proximally in the healthy area, and the carpal and Guyon's canals were opened. In the ulnar artery, a tortuous tract of approximately 2 cm was identified, containing a thrombus and an aneurysm of about 1 cm (Figure 6). The ulnar nerve was divulsed (both the motor branch and the sensory branch of the ulnar nerve). The thrombosed fragment was resected.² The ischemia tourniquet was removed and a good capillary refill was verified.¹ The skin was closed and an adequate local temperature of the fingers was verified, with capillary refill time <2 seconds.



Figure 6. Ulnar artery dissection with thrombosis and aneurysm.

The paresthesias of the 4th and 5th fingers disappeared immediately after surgery. In the postoperative period, the patient had a good evolution, with no signs of infection and a clear improvement of pain on palpation of the hypothenar eminence. The sutures were removed after 15 days. The patient was prescribed 20 sessions of physical therapy. Three months after surgery, the patient had no symptoms and resumed his work activities. Follow-up lasted a year (Figure 7).



Figure 7. Images taken one year after the intervention. Full recovery is observed.

One year after surgery, the patient had a capillary refill time of less than 2 seconds and normal mobility and sensitivity. He had no pain or trophic skin lesions. He manifested hyperalgesia discomfort with exposure to cold.

DISCUSSION

HHS is an infrequent cause of digital ischemia, accounting for <2% of the more than 1300 cases presenting to a vascular surgery center with hand-related symptoms.¹ The true incidence is not exact since chronic patients may have flow compensation from the radial artery, resulting in the formation of collateral circulation. For this reason, the speed of onset of signs and symptoms is fundamental to define the severity of the condition.¹¹

Given the low incidence of the condition and the possibility of a subclinical presentation with few symptoms due to the aforementioned flow compensation by the radial artery, reaching an early diagnosis is difficult. Despite these difficulties, once diagnosed, different treatment algorithms are available in published research studies.^{1,2}

Based on our experience, we suggest the following treatment possibilities:

1. *Asymptomatic or mildly symptomatic patients with no paresthesias in the ulnar region* (with a palpable mass and hypothenar eminence or sporadic pain, with occlusion or subocclusion of the ulnar artery, but without signs of ischemia). Medical treatment.^{2,11,12}

2. *Asymptomatic or mildly symptomatic patients with paresthesias in the ulnar region* (with a palpable mass and hypothenar eminence or sporadic pain, with occlusion or subocclusion of the ulnar artery, but without signs of ischemia). Surgical treatment with Guyon's canal release and resection and ligation of the thrombosed artery fragment.^{2,11,12}

3. *Symptomatic patients with symptoms of digital ischemia*. Initiate medical-pharmacological treatment. If there is clinical improvement, proceed with points 1 or 2 of the algorithm. If there is no improvement, proceed with vascular reconstruction surgery or end-to-end anastomosis.^{2,3,11}

CONCLUSIONS

HHS is often misdiagnosed or diagnosed late, partly because of compensation by the radial artery, but also because it is an uncommon condition. A meticulous clinical history, including an assessment of occupational or sports injuries, a thorough physical examination, and a high index of suspicion are required to make the diagnosis.

In asymptomatic cases or those with mild symptoms, observation, pharmacotherapy and risk factor management are indicated. When the evolution is more torpid, the signs and symptoms are of rapid onset, and medical treatment provides little relief, as in our patient, surgery resolves the nerve compression that produces pain and paresthesia. The treatment of choice if there is vascular ischemia is end-to-end anastomosis or vascular reconstruction with vein grafting.

With this case, we illustrated the relevance of including HHS in the differential diagnosis of patients who present in a clinical setting similar to our professional practice.

Conflict of interest: The authors declare no conflicts of interest.

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