

# Current Options for Treatment of Hypothenar Hammer Syndrome



Helen G. Hui-Chou, MD, Michael A. McClinton, MD\*

## KEYWORDS

- Hypothenar hammer syndrome • Ulnar artery thrombosis • Ulnar artery aneurysm
- Arterial aneurysm • Vein/artery grafting

## KEY POINTS

- Arteriography with contrast remains the gold standard for diagnosis and evaluation.
- Predisposing factors include tobacco smoking, manual labor (using hand as a hammer on a daily basis, daily pressure to palm of hand, daily exposure to vibrating tools), dominant hand, and male gender.
- Medical management includes smoking cessation, calcium channel blockers (nifedipine), and prostaglandins.
- Indications for operative treatment include failure of conservative management and critical digital ischemia.
- Surgical management includes resection of abnormal segment with ligation of vessel or reconstruction with venous or arterial grafts.
- Long term evaluation of graft and vessel patency remains inconsistent with patient symptoms.

## INTRODUCTION

Thrombosis of the ulnar artery (UA) secondary to blunt trauma of the hand was first described in 1934 by von Rosen in a 23-year-old factory worker who was successfully treated with excision of the thrombosed artery.<sup>1</sup> The UA at the wrist is the most common site of arterial aneurysms of the upper extremity because it is superficial and lies over the hook of the hamate. Hypothenar hammer syndrome (HHS) is a rare vascular disorder resulting from injury to the UA at the level of Guyon canal. Conn and colleagues<sup>2</sup> coined the term HHS in 1970 because it is classically seen in workers who repeatedly use the hypothenar eminence as a substitute for a hammer.

The incidence of HHS has been reported from 1.6% to 14% in manual laborers, craftsmen, and

habitual hand hammerers owing to repetitive blunt trauma.<sup>3–6</sup> Although most commonly associated with hand-intensive laborers such as mechanics, construction workers, and auto body repair workers, the condition has been diagnosed in athletes. These athletes were engaged in recreational activities including golf, badminton, squash, mountain biking, weightlifting, martial arts, baseball, basketball, football, hockey, and tennis.<sup>5,7–11</sup> Patients can present with a history of either a single or multiple repetitive traumas to the hand at the hypothenar eminence.

## RELEVANT ANATOMY AND PATHOPHYSIOLOGY

The UA passes beneath the volar carpal ligament in Guyon canal covered only by the palmaris brevis

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The Curtis National Hand Center, MedStar Union Memorial Hospital, 3333 North Calvert Street, #200, Baltimore, MD 21218, USA

\* Corresponding author. Care of Anne Mattson, The Curtis National Hand Center, Medstar Union Memorial Hospital, 3333 North Calvert Street, #200, Baltimore, MD 21218.

E-mail address: [anne.mattson@medstar.net](mailto:anne.mattson@medstar.net)

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muscle, subcutaneous tissue, and skin. Beneath this unprotected 2-cm segment of UA lies the hook of the hamate bone against which the artery can be pounded by external forces. In addition, the dorsal branch of the UA tethers the main UA, preventing it from being displaced off the hook of hamate during traumatic episodes. Repetitive trauma and occasionally single acute episodes can damage the medial wall of the artery, leading to UA thrombosis, occlusion, distal embolization, and aneurysmal degeneration and expansion of the wall.

UA aneurysms can be true aneurysms with all 3 layers of the arterial wall expanded producing a fusiform or sometimes corkscrew configuration. False or pseudoaneurysms result from penetrating trauma to the UA. In this case, the intima is breached and bleeding occurs external to the artery, forming a hematoma. Because this hematoma is connected to the UA, it may recannulize and form an eccentric fibrous outpouching of the artery without the 3 layers of the arterial wall. Pseudoaneurysms have a lower propensity for distal embolization, but they can expand and rupture, causing external bleeding.

Arterial injury may cause thrombosis and occlusion distal to the segment overlying the hamate bone extending into the superficial palmar arch and digital arteries. Damage to arterial wall media and elastic lamina results in an influx of platelets and macrophages to the site. Inflammatory mediators secreted by these cells stimulate

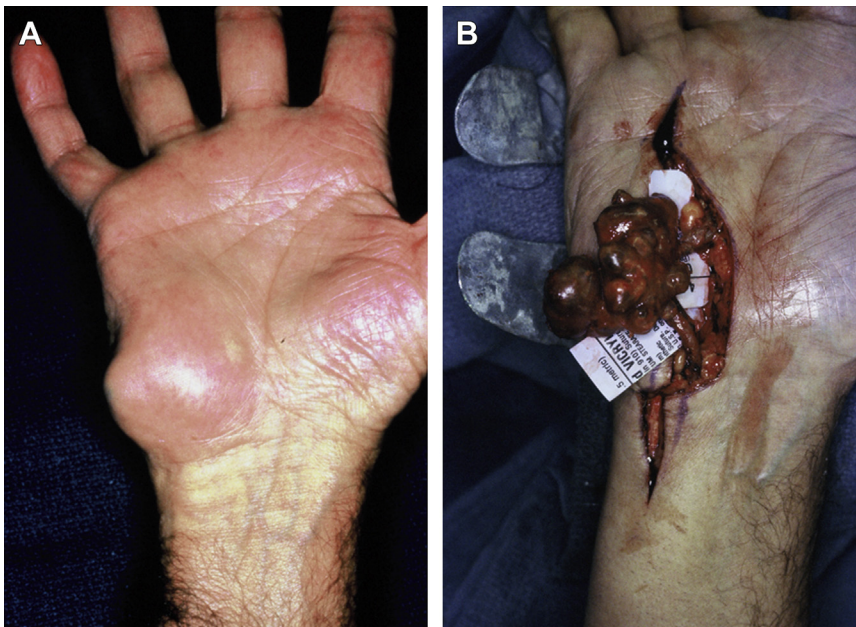
hyperplasia, fibrosis, and thrombosis. Additionally, a mechanism of preexisting palmar UA fibrodysplasia has been proposed; despite the UA's vulnerable anatomic position, HHS has a relatively low incidence in the general population.<sup>3,12</sup> Thrombosis, segmental occlusion, and microemboli to the digital arteries may result in vascular insufficiency of the hand.

## CLINICAL PRESENTATION AND EXAMINATION

Patients are predominantly male and present with symptoms of unilateral hand and digit ischemia, affecting the dominant hand most commonly (53%–93%).<sup>5,13–16</sup> Some cases of bilateral involvement have been reported.<sup>17</sup> Symptoms may be acute or chronic with recurrent episodes of pain, numbness, tingling, cold intolerance, and weakness in the ulnar nerve distribution. Symptoms vary depending on severity of occlusion and vascular insufficiency (Fig. 1).

On physical examination, the hand and ulnar digits, usually the long, ring, and small fingers, may have pallor, cyanosis, splinter hemorrhages, ulcerations, and/or wounds. Signs in the thumb have never been described.<sup>15,18</sup> A pulsatile mass may be found at the level of the wrist, with, at times, an absent pulse distal to the mass. **Table 1** lists signs and symptoms of HHS.

Several validated questionnaires have been designed in attempts to quantify the various symptoms and disabilities caused by hand ischemia.



**Fig. 1.** Hypotenar pulsatile mass (A), which revealed an ulnar artery aneurysm (B).

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