Jones Tendon Transfer

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KEYWORDS

• Jones tendon transfer • Cavus foot • Claw hallux • Hallux malleus

KEY POINTS

- Jones tendon transfer or Jones tenosuspension is a tendon transfer performed to remove the deforming force for hallux malleus deformity. It is most often performed with interphalangeal joint (IPJ) fusion of the hallux.
- Indications include hallux malleus, in conjunction with cavus foot, caused most commonly by progressive peripheral neuropathy.
- The goal of this tendon transfer is not to elevate the first metatarsal. The intact peroneus longus (PL) tendon, which has maintained the plantar flexed position of the first metatarsal, prevents significant elevation alone by this tendon transfer.
- The complications associated with this procedure are minimal and related either to rupture of the tendon or stress fracture to the first metatarsal as a result of the hole made for the tendon transfer.

INTRODUCTION

Sir Robert Jones first described the Jones tendon transfer, or Jones tenosuspension, in 1916.[1] An orthopedic surgeon in the British military, Jones was also credited with the first description of a transverse fracture at the metaphyseal/diaphyseal junction at the base of the fifth metatarsal, the Jones fracture. In his 1916 article, Jones described a surgical procedure for the treatment of clawfoot with a coexisting clawed hallux.[2]

Jones described 5 degrees of deformity for the treatment of the clawfoot. The first degree develops in childhood. It is not associated with any significant deformity, and patients have more subjective complaints, such as clumsiness, often tripping over their feet with activity. A patient's physical examination can reveal a tight heel cord, but no structural deformity is observed. Stretching is the only treatment necessary to resolve this problem. The second degree of clawfoot is described as follows: flexible in nature with a contracture of the plantar fascia, Achilles tendon, and contracture of the hallux. This type of clawfoot deformity required transfer of the extensor hallucis longus (EHL)

Disclosure: No funding or conflict of interest.
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Clin Podiatr Med Surg 33 (2016) 55–62
http://dx.doi.org/10.1016/j.cpm.2015.06.004 **podiatric.theclinics.com**
0891-8422/16/$ – see front matter

tendon. The Jones tendon transfer was described as a transfer of the EHL from its insertion, through the neck of the first metatarsal and tied on itself. Jones combined this tendon transfer with an incision of the plantar fascia.[2] Over the years, the indications have remained similar to Jones' original description in the literature. The IPJ fusion of the hallux was added to the description with the tendon transfer in recent literature.

The third degree of the clawfoot described by Jones was a rigid contracture of the lesser digits at the metatarsophalangeal joint (MPJ) level as well as the hallux. Tendon transfer was not performed in the later stages due to the fixed deformity of the lesser toes. The forth and fifth degrees were described as fixed in nature and had the same contracture as noted by the third degree but with an equinovarus deformity. In the most severe cases, fifth degree, Jones recommended a distal amputation removing all the toes as well as metatarsal heads.[2]

INDICATION/ETIOLOGY

The Jones tendon transfer is performed for the treatment of the clawed hallux or hallux malleus deformity. Typically, this tendon transfer is performed in combination with a hallux IPJ fusion. A clawed hallux is defined as extension of the first MPJ and flexion of the IPJ. The deformity is attributed to muscular imbalances that include the intrinsic musculature, the *flexor hallucis longus* (FHL) tendon, the EHL tendon, and the PL tendon.[3] The cause of this imbalance has yet to be quantitatively described. Several studies have implicated that the 3 extrinsic muscles acting on the hallux are thought to be the main deforming forces, but the intrinsic muscles may have a more significant role than previously thought.[1,4] Olson and colleagues,[4] in 2003, looked at the 3 extrinsic muscles acting on the hallux in a cadaveric study. The investigators concluded the main attribute that led to plantar flexion of the first metatarsal that leads to increased pressure under the first metatarsal head is due overpowering of the PL tendon. Overpowering of the EHL resulted in the excessive extension deformity of the MPJ and the pull of the FHL led to the largest deformity of the IPJ, which caused increase pressure under the hallux. Giannini and colleagues,[1] in 1985, concluded the etiology of the hallux claw toe was a cause of muscular imbalances but the FHL and EHL are not the main factors and might contribute and aggravate the deformity.[1]

The closed kinetic chain function of the intrinsic muscles is to stabilize the hallux to the ground at the MPJ. Weakness or overpowering of the intrinsic muscles by the extrinsic muscles is the cause of hallux malleus deformity, resulting in the contracture at the MPJ and flexion deformity at the IPJ.

There are 2 specific categories that result in the muscular imbalance causing claw toe deformity. The etiology can be either neurologic or idiopathic. Idiopathic refers to an unspecified cause of the deformity. The neurologic group can be further divided into either a result from an upper motor neuron disease (spastic) or lower motor neuron disease (flaccid). Charcot-Marie-Tooth (CMT), poliomyelitis, Roussy-Lévy syndrome, and Friedreich ataxia are the most common lower motor neuron diseases causing disorders of the foot and lower extremities. CMT is the best-studied and most common disorder causing a cavus deformity. Approximately 90% of patients with CMT present with a cavus foot deformity.[5] A study by Palma and colleagues,[6] in 21 subjects treated with a Jones procedure for clawed hallux, reported that 6 had poliomyelitis, 3 had cerebral palsy, 3 had spina bifida, 2 had unknown neurologic disease, and 7 were idiopathic cavus feet. A study by Kadel and colleagues,[7] however, reported a large dominance in CMT compared with other patient populations when seeing a clawed hallux. The mechanism that leads to the hallux claw toe is recruitment of the EHL and extensor digitorum longus as accessory dorsiflexors, to balance the weakness of the tibialis anterior.[5]