DOI: https://doi.org/10.30795/jfootankle.2021.v15.1577

Special Article

Concept, etiology, and pathomechanics of hallux rigidus

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Abstract

There is a continuous anatomical, functional and pathomechanical roadmap from functional hallux limitus to hallux rigidus. Although many etiologies for hallux rigidus have been studied it is very probable that it has a primary origin with less-than-ideal movement when we are born. Upon a restricted range of motion, symptoms may arise depending on the amount of work and how compensatory mechanisms work around the first metatarsophalangeal joint. Changes occurring at the joint that allow the transition from a sliding movement mechanism (physiological) to a rolling mechanism (pathological) may trigger anatomical and functional changes resulting in pain and dysfunction. Any surgical technique that is able to restore the sliding mechanism to the first metatarsophalangeal joint will have a positive impact on pain and function in a patient with a symptomatic functional hallux limitus/rigidus.

Level of Evidence V; Therapeutic Study; Expert opinion.

Keywords: Hallux limitus/etiology; Hallux rigidus/etiology; Biomechanics.

Introduction

Hallux rigidus (HR) is the second most frequent disease of the great toe, after hallux valgus(1). Lower dorsiflexion mobility of the first toe under weight-bearing conditions is initially known as functional hallux limitus (FHL). In some patients, the evolution of FHL leads to the development of HR(2). Both FHL and HR may be more or less symptomatic in our patients. The pathogenic mechanisms of disease progression remain unknown, as well as the reason why an asymptomatic foot eventually develops symptoms. This study presents a pathogenic mechanism that may explain these issues.

Definition and concepts

The term "hallux rigidus" originates from the Latin words *"hallux"* and *"rigeo"*, i.e., rigid great toe. In the field of Orthopedic Surgery, this term is used to describe a condition that affects the metatarsophalangeal joint (MTPJ) of the great toe and is characterized by a progressive loss of dorsiflexion and the formation of periarticular osteophytes that eventually cause stiffness, generally painful, which may even become incapacitating.

In 1887, Davies-Colley described the existence of a plantar flexion position of the proximal phalanx in relation to the first metatarsal head and coined the term "hallux flexus"(3). Some months later, Cotterill(4) described the same disease, but named it as "hallux rigidus". During the years following the initial definition, other names were also used to refer to HR: "dorsal bunion," "hallux dolorosus," and "hallux malleus"(1).

Each person is born with a given mobility in their great toe joint. Mobility examined with the patient seated (open kinetic chain) may be very different from mobility while walking (close kinetic chain). When walking, around 60° of dorsiflexion in the first MTPJ is required for feet to take off during the third rocker of gait without generating loading transfers nor changes that may be mechanically detrimental to neighboring structures. Great toes that are unable to perform this ideally necessary dorsiflexion are known as *"functional hallux limitus"*. This condition is not pathological as long as it does not cause pain and limitation. When the mechanics of the structures that land and stabilize the first metatarsal head over the sesamoid flexor complex of the first toe fails, there is a pathological change in the way how the first MTPJ works, which will produce a greater contact between the dorsum of

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Conflicts of Interest: none. **Source of funding:** none. **Date received:** September 11, 2021. **Date accepted:** October, 04, 2021. **Online:** December 20, 2021.

How to cite this article: Monteagudo de la Rosa M, Viladot-Pericé R. Concept, etiology, and pathomechanics of hallux rigidus. J Foot Ankle. 2021;15(3):193-7.

the metatarsal head and the dorsum of the proximal phalanx of the hallux. After this moment, and always determined by patient's intrinsic (internal compensation mechanisms) and extrinsic circumstances (physical activity, footwear), there will be progression to the development of HR (Figure 1). This condition is the second leading cause of great toe problems, after hallux valgus[(4)]. Gould at al.[(2)] found an incidence of 1 in every 45 individuals older than 40 years.

Etiology

We classically make a distinction between primary etiology (birth or constitutional and mechanical ones) and secondary etiology (posttraumatic ones, systemic diseases, osteochondritis dissecans, iatrogenic ones after hallux valgus surgery). In this section of our study, we will focus on primary etiology and its relationship with intrinsic, constitutional, and mechanical factors that could favor the development of HR. Several factors have been proposed to explain its development, but none of them could be proven with a significant level of evidence.

Sometimes, an anatomical disposition and its opposite have been correlated with the etiology of HR. For instance, an excessively long first MTPJ (index plus) has been related to increased pressure on the first MTPJ that could have a harmful effect on this joint[(5,6)]. Similarly, an excessively long first toe (Egyptian foot) in relation to the smaller toes have also been proposed by the same mechanism[(6-8)]. However, other authors have suggested that a short first metatarsus (index minus) could be the origin of HR[(9)]. The existence of hallux valgus interphalangeal or intraphalangeal has also been associated with the development of HR, but this finding seems to be an adaptive consequence rather than a cause[(10)].

Retraction of plantar soft tissues, with consequent limitation of first toe dorsal mobility, was one of the most debated etiological theories in the past[(11)]. Retraction of intrinsic muscles affects hallux dorsiflexion[(12)]. Excessive traction of plantar fascia in cadavers reduces hallux dorsiflexion, which increases by nearly 10° when plantar fascia is sectioned close to the hallux[(13)]. Progressive flexor hallucis longus fibrosis at its myotendinous junction leads to a significant limitation in hallux dorsal mobility and increases compressive forces on the first MTPJ[(14)]. Distal plantar fascia release is able to improve first MTPJ dorsiflexion[(11,13,15)]. Theories centered on soft tissues are inconsistent and often refer to consequences rather than to causes of HR.

Figure 1. Osteology of hallux rigidus in the first ray of a cadaver with large osteophytosis. (Photograph Pau Golanó†/Patricia Ruiz, Universidad de Barcelona).

For a long time, one of the etiopathogenic theories on HR had been centered on the existence of primary first metatarsal elevation (*"metatarsus primus elevatus"*) in relation to smaller metatarsi[(16)]. It was tempting to think that primary first metatarsal elevation would considerably change the reverse windlass mechanism of plantar fascia and would significantly limit dorsal mobility of the first MTPJ, causing the well-known chain of pathogenic events culminating in joint destruction. In 1938, Lambrinudi[(17)] suggested that primary first metatarsal elevation is a causal factor of HR. However, several authors have shown that an elevation of 5 mm could be observed in up to two-thirds of normal feet and advised against first metatarsal plantar flexion osteotomy in the treatment of symptomatic HR[(16,18-20)]. Most first metatarsal elevations improve after any surgery, with positive effects on pain and first MTPJ motion; thus, this elevation is currently considered a possible consequence rather than a cause of HR[(21)].

The non-spherical morphology of first metatarsal head has been also associated with the development of HR. Studies by Coughlin and Shurnas and by Shumas reported up to 74% of flat, quadrangular, and chevron-shaped metatarsal heads in their patients with HR[(10,22)], and other authors found similar figures[(23)]. Again, it was not possible to demonstrate what is a cause and what is a consequence. Inconsistence in the bilateral involvement of HR prevented to draw reliable conclusions about the contralateral foot with regard to the morphology of the first metatarsal head.

Other possible mechanical causes reported, such as metatarsus adductus, pronated foot, hallux valgus, first cuneometatarsal joint hypermobility, effect of footwear, an anomalous nucleus of ossification within the metatarsal, could not be demonstrated, and several studies show lack of cause-effect relationship to produce HR[(24-27)]. Behind these mechanical causes, there is instability, which would be easily responsible for joint degeneration[(10)].

There is a family cluster in the presentation of cases, and Coughlin and Shurnas[(10)] suggested that almost two-thirds of patients had a family history among their close relatives. However, no genetic review could make us think of some type of concrete heritage.

Pathomechanics

Normal first MTPJ range of motion is around 110°, with a plantar flexion of 35° and dorsiflexion of 75°. Although there is great variability in the estimated values, nearly 60° of first MTPJ dorsiflexion are required for a normal third rocker of gait[28]. It is not clear yet why great toes with good or acceptable mobility eventually develop painful and limiting HR, but it is known that the etiological factors that have been historically proposed in the literature and mentioned in the previous section of this study do not justify this transition.

Maceira and Monteagudo[21] worked on a pathomechanical explanation to elucidate why hallux limitus eventually becomes symptomatic and progresses to HR. There would be an explanation to understand why, at a certain point of life, the great toe becomes symptomatic and does not improve. This explanation should also justify clinical and radiological findings and elucidate why apparently different surgical treatments, such as a plantar flexion osteotomy or an extensive cheilectomy, can both improve symptoms[21]. In a normal foot, the first MTPJ works with a sliding mechanism on the sesamoids while there is an advance of the mass center of the body during the transition from the second to the third rocker of gait. In this sliding mechanism, rotation centers remain constant and centered throughout the entire joint movement and are located in the anatomical center of the first metatarsal head[21]. In HR, rotation centers are located in an eccentric position, dorsal to the first metatarsal head, thus causing compression with greater friction between the first metatarsus and the dorsal region of the first phalanx. In this line of mechanical knowledge, transition from the sliding mechanism to the rolling mechanism would make FHL symptomatic. When maintained over time, the rolling mechanism would progressively tear the joint, causing HR (Figure 2).

The first MTPJ range of motion with which one is born may significantly determine the age of symptom onset. Patient's physical fitness also determines the action mechanisms that impair the proper functioning of the first MTPJ and determines the onset of pain. For the ideal functioning of the joint during sliding, the triceps and the subtalar joint should allow for the peroneus longus to promote plantarization of the first metatarsus, in order to stabilize the metatarsal head by "sinking it" into the sesamoid flexor apparatus. In this position, the head would be "trapped" in the plantar position, favoring passive dorsiflexion of the toe by the reverse winch mechanism. Conversely, when these mechanisms are not able to effectively achieve plantarization of the first metatarsal head (eg, due to foot aging), the head starts to work with a rolling mechanism, thus "jumming into" the dorsal proximal phalanx of the hallux and producing the chain of damages known as incipient HR (Figure 3). This anomalous dorsal compression of the head against the phalanx would lead to greater supination of the forefoot, with the emergence of secondary symptoms. The associated morphological changes are justifiable within this context. The Delpech's Law explains that, when the proximal phalanx is obliged to exert greater pronation efforts during the third rocker of gait and has an asymmetrical shape, it may cause hallux valgus inter/intraphalangeal, present in almost 100% of cases of HR. Two of the common, but very different, surgical techniques, such as extensive cheilectomy with Möberg-Akin osteotomy and proximal plantarization osteotomy of the first metatarsal, produce the same effect of restoring the sliding mechanism, obliging the metatarsal head to plantarize itself against the sesamoid flexor apparatus and favoring the passive dorsiflexion mechanism without the jamming of the metatarsal head into the dorsal aspect of the proximal phalanx (Figure 4). Any surgical technique for

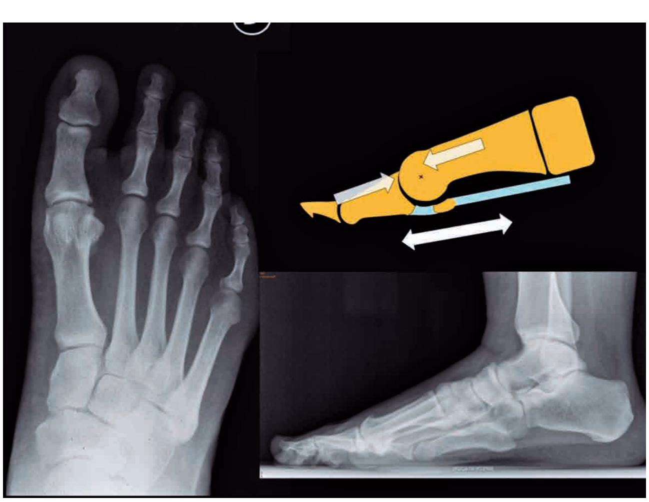

Figure 2. The rotating mechanism represented here favors the relative first metatarsal elevation and dorsal jumming against the phalanx in functional hallux limitus. Weight-bearing radiographs show radiological signs of incipient osteophytes, relative first metatarsal head elevation, and hallux valgus inter/intraphalangeal.

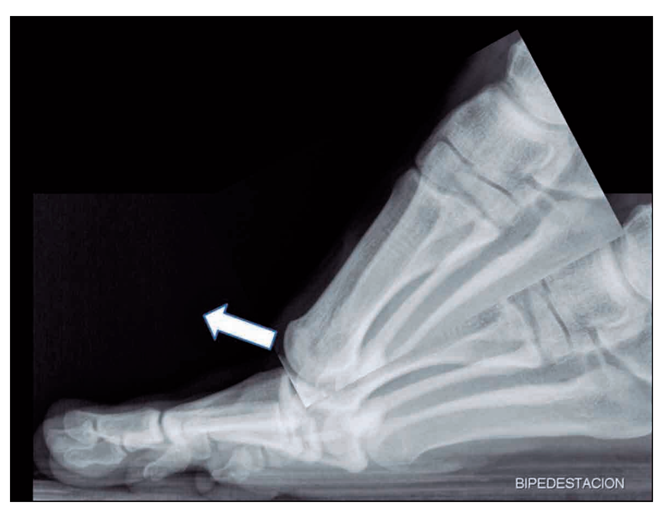

Figure 3. Radiological representation of progression of the third rocker of gate when the metatarsophalangeal joint of the first ray works with a pathological rolling mechanism. It is possible to predict harmful mechanical effects on the site of pressure and jumming between the first metatarsal head and the proximal phalanx of the hallux.

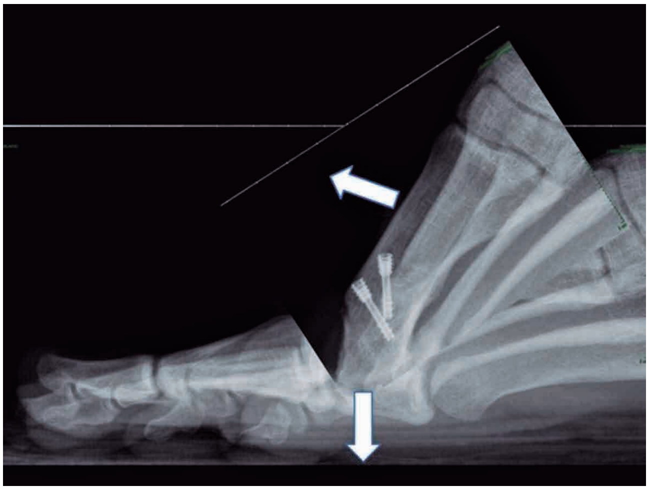

Figure 4. Same representation after first metatarsal Weil osteotomy with lowering of the metatarsal head and cheilectomy. The created "hinge" region restores the sliding mechanism that prevents the jumming of the first metatarsal head over the proximal phalanx of the hallux.

treating FHL that manages to put the sliding mechanism into operation and to prevent the rolling mechanism from working will achieve a good mechanical effect on the first MTPJ and to inhibit progression to HR.

Conclusion

Progression from FHL to HR often leads to the onset or worsening of patient's painful symptoms. Although many etiopathogenic factors have been described for the emergence and worsening of this condition, it is not clear which of them cause a patient to present with pain and to eventually require surgery. We do believe that the mechanical work of the MTPJ has an "expiration date" when the compensation mechanisms wear out and inexorably replaces the physiological sliding mechanism with the rotating mechanism, which is pathologic. Conservative and surgical treatments that manage to restore the sliding mechanism to the first MTPJ and to prevent the rolling mechanism will be successful in promoting pain relief and functional improvement.

Authors' contributions: Each author contributed individually and significantly to the development of this article: MMR *(https://orcid.org/0000-0002-0107-5675) Wrote the article, participated in the review process, bibliographic review, formatting of the article, interpreted the results of the study, performed the surgeries; RVP *Conceived and planned the activity that led to the study, participated in the review process, bibliographic review, interpreted the results of the study. *(https://orcid.org/0000-0002-8254-2916) All authors read and approved the final manuscript. *ORCID (Open Researcher and Contributor ID) iD.

References

1. Yee G, Lau J. Current concepts review: hallux rigidus. Foot Ankle Int. 2008;29(6):637-46.
2. Gould N, Schneider W, Ashikaga T. Epidemiological survey of foot problems in the continental United States: 1978-1979. Foot Ankle. 1980;1(1):8-10.
3. Davies-Colley N. Contraction of the metatarsophalangeal joint of the great toe (hallux lexus). Br Med J. 1887;1:728.
4. Cotterill JM. Stiffness of the great toe in adolescents. Br Med J. 1888;1(1378):1158-62.
5. Nilsonne H. Hallux rígidus and its treatment. Acta Orthop Scand. 1930;1:295-303.
6. Munuera PV, Domínguez G, Castillo JM. Radiographic study of the size of the first metatarso-digital segment in feet with incipient hallux limitus. J Am Podiatr Med Assoc. 2007;97(6):460-8.
7. Calvo A, Viladot R, Giné J, Alvarez F. The importance of the length of the first metatarsal and the proximal phalanx of hallux in the etiopathogeny of the hallux rigidus. Foot Ankle Surg. 2009; 15(2):69-74.
8. Calvo A. Case-control study for the evaluation of the association between morphological parameters of the foot and the presence of hallux rigidus [thesis]. Tarragona: Universitat Rovira i Virgili; 2005.
9. Ogilvie-Harris DJ, Carr MM, Fleming PJ. The foot in ballet dancers: the importance of second toe length. Foot Ankle Int. 1995; 16(3):144-7.
10. Coughlin MJ, Shurnas PS. Hallux rigidus: demographics, etiology, and radiographic assessment. Foot Ankle Int. 2003;24(10):731-43.
11. Asunción Marquez J. Martín Oliva X. Hallux rigidus: aetiology, diagnosis, classification and treatment. Rev Esp Cir Ortop Traumatol. 2010;54(5):321-8.
12. Durrant MN, Siepert KK. Role of soft tissue structures as an etiology of hallux limitus. J Am Podiatr Med Assoc. 1993;83(4):173-80.
13. Harton FM, Weiskopf SA, Goecker RM. Sectioning the plantar fascia. Effect on first metatarsophalangeal joint motion. J Am Podiatr Med Assoc. 2002;92(10):532-6.
14. Flavin R, Halpin T, O'Sullivan R, FitzPatrick D, Ivankovic A, Stephens MM. A finite-element analysis study of the metatarsophalangeal joint of the hallux rigidus. J Bone Joint Surg Br. 2008;90(10):1334-40.
15. Rochera R, Lluis L, Viladot R. The importance of plantar muscles in the Hallux-Rigidus. In: Actas del 2nd EFORT Congress, 1995. Munich, Alemania: EFORT. p. 74-5.
16. Meyer JO, Nishon LR, Weiss L, Docks G. Metatarsus primus elevatus and the etiology of hallux rigidus. J Foot Surg. 1987;26(3):237-41.
17. Lambrinudi C. Metatarsus Primus Elevatus. Proc R Soc Med. 1938;31(11):1273.

18. Horton GA, Park YW, Myerson MS. Role of metatarsus primus elevatus in the pathogenesis of hallux rigidus. Foot Ankle Int. 1999;20(12):777-80.
19. Caminear DS. Role of metatarsus primus elevatus in the pathogenesis of hallux rigidus. Foot Ankle Int. 2000;21(11):967.
20. Roukis TS. Metatarsus primus elevatus in hallux rigidus: fact or fiction? J Am Podiatr Med Assoc. 2005;95(3):221-8.
21. Maceira E, Monteagudo M. Functional hallux rigidus and the Achilles-calcaneus-plantar system. Foot Ankle Clin. 2014;19(4):669-99.
22. Shurnas PS. Hallux rigidus: etiology, biomechanics, and nonoperative treatment. Foot Ankle Clin. 2009;14(1):1-8.
23. Hunt KJ, Anderson RB. Biplanar proximal phalanx closing wedge osteotomy for hallux rigidus. Foot Ankle Int. 2012;33(12):1043-50.
24. McMaster MJ. The pathogenesis of hallux rigidus. J Bone Joint Surg Br. 1978;60(1):82-7.
25. Blázquez Viudas R. Hallux Limitus and relationship with the foot pronated as etiological factor. Rev Int Cienc Podol 2011;5(1):21-7.
26. Colò G, Fusini F, Zoccola K, Rava A, Samaila EM, Magnan B. May footwear be a predisposing factor for the development of hallux rigidus? A review of recent findings. Acta Biomed. 2021;92(S3):e2021010.
27. Saggini R, Colotto S, Innocenti M. [Presence of a nucleus of distal ossification of the first metatarsus and its correlation with the pathogenesis of juvenile hallux rigidus]. Arch Putti Chir Organi Mov. 1984;34:59-69.
28. Camasta CA. Hallux limitus and hallux rigidus. Clinical examination, radiographic findings, and natural history. Clin Podiatr Med Surg. 1996;13(3):423-48.