Abstract

Objective: This work performs a critical review of the different causes described to explain the etiopathogenesis of hallux valgus.

Methods: The authors divide the causal factors into two groups: extrinsic and intrinsic factors. In the first group, footwear and mechanical overload caused by different causes such as ballet, trauma, long walks, obesity, etc., should be considered. In the second group we include a series of factors: constitutional ones, such as heredity, sex and age; anatomical aspects, among which we must highlight the morphology and obliquity of the metatarsocuneiform joint; hypermobility of the first ray; metatarsus primus varus; muscle function; and atavism.

Results: Hallux valgus probably has a multifactorial etiology whose triggering factor is unknown at the moment.

Conclusion: If we know the etiopathogenesis of a deformity, in this case hallux valgus, we can perform a treatment as early and effective as possible.

Level of Evidence V; Therapeutic Studies; Expert Opinion.

Keywords: Hallux valgus/etiology; Intrinsic factor; Extrinsic factor.

Introduction

Hallux valgus (HV) is the most frequent deformity of the locomotor system, being present in more than 35% of people over 65 years old (1). It is defined as a three-dimensional deformity (2), in which the three spatial planes should be considered: transverse, sagittal, and coronal. In the transverse plane, the following variables are assessed: angles between the 1st and 2nd metatarsals, the metatarsophalangeal joint, PASA (Proximal Articular Set Angle), DASA (Distal Articular Set Angle), location of the sesamoid bones and of the CORA (Center of Rotation of Angulation), metatarsocuneiform joint obliquity, and presence of arthrosis in metatarsophalangeal and metatarsocuneiform joints. In the sagittal plane, elevation and descent of the first metatarsal head are observed. Finally, in the coronal plane, hallux pronation is found in more than 80% of the cases, as well as the status of sesamoid bones.

Our aim is to perform a critical analysis of the different causes to explain the etiopathogenesis of HV.

Pathogenesis

HV pathogenesis is a complex topic that evokes different opinions, which explains the emergence of many controversies.

In a lecture delivered at the Royal College of Surgeons of England in 1956, Lake (3) (Consulting Surgeon, Charing Cross Hospital) commented: “[…] yet there is still controversy about
the etiology of such a simple and common condition as hallux valgus, for, despite the large amount of anatomical, statistical and sociological study which has recently been carried out, the problem remains unsolved.” In their paper “The pathogenesis of Hallux Valgus”, Perera et al. (4) (University Hospital of Wales, Cardiff, United Kingdom) state: “A century of debate has failed to settle the importance of intrinsic versus extrinsic causes in the etiology of HV” (Figure 1 A and B).

**Extrinsic factors**

Inadequate footwear

Durlacher (5), a surgeon-chiropractist that served Queen Victoria, published one of the first treatises on diseases of the foot in 1845 (“A treatise on corns, bunions, the diseases of nails and the general management of the feet”), in which he states: “One of the most certain causes of a “bunion” is the wearing of shoes made too short, and with a narrow sole”.

It is noteworthy that, both in England and in France, health authorities were interested in controlling the characteristics of footwear. In France, Charles V (1338-1380) issued an edict establishing the length of footwear, and, in England, Queen Elizabeth I (1533-1603) restricted its width. However, as mentioned by Lake (3), the historian Viollet le Duc commented: “as always happens, fashion proved stronger than all the edicts of kings and councils”.

For many authors, improper footwear acts as a potential cause for the onset of HV (6,7). It is also important to mention cases of congenital HV that are isolated or part of a generalized disease, juvenile HV in adolescents who have never worn narrow shoes and, finally, HV in individuals who have never worn any type of footwear or who wear very different shoes from those of the western world (8,9). Studies conducted with primitive peoples found a low number of cases in Solomon islands and in Belgian Congo (10,11).

In our opinion, the most probable reason for these findings may be the fact that footwear is likely to favor the progression of deformity instead of being the initial cause of the structural anomaly.

**Ballet**

Within HV pathogenesis, one should assess the scenarios in which feet will maintain a constant posture and be subjected to constant overload. These situations may include classical dance, in which dancers often suffer from metatarsalgia and hallux deviations resulting from joint and muscle overload.

Seki et al. (12) conducted investigations to develop appropriate training methods to prevent the progression of HV. A study with female classical ballet dancers at the advanced college-level concluded that the degree of HV is correlated with basic techniques of classical ballet such as the first position.

In their studies, Pérez and Massó (13) state that classical positions, especially first positions, often leads to the onset of HV, due to the muscular effort demanded by these positions (Figure 2).

In a study conducted with 106 dancers, these authors concluded that HV and hammer toes are often observed in classical dance. In their series, there was a percentage of 67% of square feet, 26% of Egyptian feet, and 8% of Greek forefeet. The highest percentage of HV was found in Egyptian feet.

In the pointe position of ballet, a longer hallux tends to deviate towards the second toe, in order to equal the length of both toes and increase contact with the ground. On the other hand, muscle control over the position of the hallux by the abductor hallucis muscle is reduced in the “en dehors” position. Dancers with joint laxity show an increased number of cases of HV. Metatarsalgia and claw toes are more frequent in Greek feet, and less frequent in square feet.

There is not enough evidence to conclusively show that dance, specifically the pointe technique and physical preparation, increases the prevalence or the severity of HV. Most authors think that “permanent aggression” to the first metatarsocuneiform joint may be a factor to consider in the presentation of HV when this joint is not able to withstand the overload to which it is permanently subjected.

Figure 1. A. Extrinsic factors. B. Intrinsic factors.
Trauma

Low-energy trauma affecting the Lisfranc ligament may cause a Lisfranc fracture-dislocation that is radiologically manifested by diastasis between the first and the second cuneiform bones (subtle injury), sometimes accompanied by bone fragments resulting from avulsion of the Lisfranc ligament insertion on the second metatarsal base (fleck sign).

It is worth remembering that the Lisfranc ligament connects the first cuneiform bone to the second metatarsal base in an oblique, lateral, distal direction (Figure 3). The first metatarsocuneiform joint is located in front of the Lisfranc ligament, and its stability is achieved by the action of intrinsic and extrinsic muscles, especially peroneus longus, ligaments, and articular capsule. Therefore, this joint represents a "weak point", which Klaue(14) attributes to the fact that the foot is at a "young stage", from a phylogenetic point of view, and is subjected to mechanical overloads. This may cause metatarsocuneiform joint instability, favored by the unbalance of muscles inserted into the phalangeal base, therefore leading to the development of metatarsal varus.

Direct trauma would cause minor or major fracture-dislocation of the Lisfranc joint, whereas mechanical overload may favor the onset of HV. Post-traumatic HV following direct injury is little frequent. It may be observed among athletes experiencing a strong impact on the forefoot. Most studies were conducted with soccer players with internal lateral ligament injuries of the first metatarsophalangeal joint. Lui(15) also mentions other possible causes, such as sequel of Lisfranc joint trauma, first metatarsocuneiform trauma, first metatarsal fractures, and entrapment of the internal plantar nerve in distal tibial fractures.

Other possible factors

The presence of HV was also related with other possible causes that imply overload, such as: long walks, carrying excessive load, obesity, etc. However, there are no significant statistical studies confirming these supposed associations.

Intrinsic factors

Heredity and race

Genetic probably plays an important role in the development of the deformity. This is possibly due to a dominant autosomal inheritance pattern with incomplete dominance. Piqué-Vidal et al.(16) studied a group of 350 individuals across three generations, confirming such hypothesis.
There is an accepted maternally inherited predisposition to HV, especially in juvenile HV and that affecting young adults. In a meta-analysis with 5925 Caucasian individuals, Arbeeva et al. (17) identified a new locus (COL. 24A 1) on chromosome 1 that partly encodes collagen and would be related to the onset or to the higher frequency of HV.

The presence of HV is two-fold higher in the white than in the black population (18).

Sex

The clear predominance of women compared to men with HV, in a 10:1 ratio, probably has a genetic cause. Published investigations revealed that the hallux metatarsal head is more rounded and smaller in women than in men, which could favor the onset of HV (19), as well as ligament hyperlaxity and hypermobility, which are more frequent among females. Footwear may also exert an influence.

Age

In a meta-analysis published by Shere et al. (20), the estimated grouped prevalence of HV was 23% in women aged 18-65 years old, reaching 35.7% in elderly people aged over 65 years. It means that one out of three elderly women had HV. In another study, Roddy et al. (21) found that this deformity had an incidence of 26.4% in the population over 35 years old.

Anatomical aspects

Some authors state that flat foot favors the presence of HV (22), whereas others think there is no such relationship (23). According to our criterion, flat feet with flattening of the medial arch secondarily results in abducted, pronated forefoot, which is often associated with HV.

Núñez-Samper observed that adult-acquired flat foot due to posterior tibial dysfunction (stages III and IV) was associated with moderate or severe HV in most treated cases (Figure 4).

HV may present in any type of forefoot, regardless of metatarsal and digital formula. This is observed in daily practice. However, an Egyptian digital formula, together with an index minus metatarsal formula, would favor the onset of HV.

Mann and Coughlin (24) studied the shape of the metatarsal head, concluding that rounded heads would favor the onset of HV. At the level of the metatarsal cuneiform joint, we believe that joint surface obliquity has a significant role in HV with major deformity (Figure 5).

Hypermobility of the first ray

Lapidus (25) states that hypermobility of the first ray has a major role in the development of deformity in HV. In investigations with cadaveric foot specimens with HV, Coughlin et al. (26) observed that joint mobility decreased when first ray realignment was performed with crescentic osteotomy.

When manually exploring hypermobility in the sagittal plane, it is important that the knee remain in the neutral position during examination, since dorsiflexion tensions the plantar fascia and reduces the range of motion in the sagittal plane. Conversely, plantar flexion relaxes the fascia and increases mobility. It is recommended that the knee flexed during examination.

For many years, the concept of hypermobility of the first metatarsal cuneiform joint was centered on the sagittal plane; however, in our opinion, transverse and coronal (pronation) planes should also be assessed, since they contribute for the development of HV.

Some factors should be considered when investigating first metatarsal hypermobility. Moderate ligament laxity is common in patients with HV. Conversely, hypermobility of the first ray is a factor that favors relapses (27). Morphology and inclination of the first metatarsal-cuneiform joint is closely related to hypermobility of the first metatarsal.

In conclusion, hypermobility of the first ray is related to several factors leading to the persistence of mobility of the first metatarsal cuneiform joint, as occurring in primates.
Metatarsus primus varus

Some studies give the same meaning to the expressions HV and metatarsus primus varus. HV is defined, incorrectly in our opinion, as a deformity involving a first metatarsal varus deviation with an intermetatarsal angle greater than 9° and a hallux valgus deviation with a metatarsophalangeal angle greater than 15°. Strictly speaking, metatarsus primus varus would correspond to medial first metatarsal deviation, whereas HV would be a lateral hallux deviation. However, the expression HV usually covers first metatarsal and hallux deviation.

Some authors, such as Kilmartin(29) and Klaue(14), believe that metatarsus primus varus is a specific foot morphotype, and compare it with other foot types, such as Egyptian, square, or Greek foot. In the case of metatarsus primus varus, it is characterized by a clear separation between the first metatarsal and the second and the varus hallux.

This arrangement is observed in the fetus up to the ninth week of intrauterine life and persists in a great number of individuals at birth. It is known as “fan-shaped foot”.

Truslow(30) was the first to relate HV to metatarsus primus varus and interpreted it as an anatomical variant.

Currently, it is accepted that metatarsus primus varus favors the onset of HV, especially its juvenile cases.

Muscle action

The five muscles inserted into the hallux have a great importance in the development of HV deformity, since they tend to displace the toe outwards. In 1887 Wyeth(31) wrote: “The action of the muscles inserted into the hallux should not be ignored in the etiology of hallux valgus”.

In 1978, Iida and Basmajian(32) published an electromyography study that compared the electromyography responses of adductor and abductor hallucis muscles and flexor hallucis brevis muscle in normal feet and those with HV. Feet with HV had a relatively weak medial flexion force, a strong lateral flexion force, weak adduction, and no abductive force at the level of the metatarsophalangeal joint. According to the authors, these changes in muscle balance around the joint may favor the presentation of HV.

Muscle imbalance in adductor and abductor muscles is evident in HV deformity. However, the study did not determine whether changes in muscle action are a cause or a consequence of HV.

As Lelievre(33) states in the book “Patología del Pie”: “The action of muscles tends to accentuate deformity”. In HV the extensor hallucis muscle form the cord of an arch, together with the flexor longus muscle. Both muscles are functionally converted to abductor hallucis muscles. The external portion of the flexor hallucis brevis acts the same way. There are no antagonists, since the adductor muscle is situated at the sole of the foot.

This anatomical and functional change at the level of the muscles leads the toe to become internally rotated or pronated. In turn, the cause of metatarsal pronation should be more proximal, as occurring with primates in which internal rotation or pronation is produced in the tarsometatarsal joint.

How musculotendinous aspects act on the first metatarsal-cuneiform joint?

Bohne et al.(34) state that peroneus longus plays an important role in maintaining stability both in the sagittal and in transverse planes of the first metatarsocuneiform joint, with a most notable action in the sagittal plane.

Gastrocnemius shortening is an important point to consider. It is found in 40% of the population, according to Kowalski et al.(35), and is a frequent cause favoring the onset of HV. In our opinion(36) this shortening has an atavic nature. When human gain started, our ancestors walked on tiptoes, in a valgus position, and the heel was distant from the ground. Achilles tendon retraction leads to joint overload at the end of the 2nd rocker. It is important the assess the retraction of the gemelli muscles through the Silfversköld test(37) and proceed with their enlargement in case of failure of rehabilitation treatment.

Atavism

We understand atavism as “the reappearance, in living beings, of regressive characters typical of their ancestors within the evolutionary line”.

The foot of our relatives in the phylogenetic scale had a three-dimension structure very similar to that of our hands, in which the first ray was separated from the others and was pronated.

At the knee level, mobility of primates is greater than that of humans. Due to its anatomical configuration and to the role played by intra-articular soft tissues(38), the knee behaves as a ball and socket joint, from a biomechanical point of view.

The subtalar joint of primates has a much greater mobility compared to that of humans, since the axis of motion of anterior y posterior joints does not limit rotation capacity at this level.

These characteristics of the subtalar joint and of the knee facilitate the passage from pronation to supination in the foot without losing joint stability.

Primates also show a greater mobility in the tarsometatarsal, since the embedment of the second metatarsal base within the mortise created by the three cuneiform bones, provide humans with greater foot stability, which is required for remaining in the standing position, but reduces mobility and thus prehensile capacity of foot. The metatarsocuneiform joint has evolved throughout history(39). Between the Triassic and Jurassic periods (215 Ma) the bone named os tarsale or 1st cuneiform bone appeared for the first time. A small divergence was observed between the 1st ray and the lateral rays. During the Eocene period (53 Ma), there is the emergence of modern-prosimian primates. They live an arboreal life, due to the prehensile capacity of their hands and feet. Plantar dermatoglyphics emerge; mo-
however, fingers and toes terminate in nails rather than in claws. The metatarsocuneiform joint acquires a saddle shape, reinforced in the insertion area of the peroneus longus.

In the Oligocene period (35 Ma), there is the emergence of anthropoid primates. A desertification of the landscape occurs, with the formation of steppes, savannas, and large open spaces on the earth’s surface.

Primates come down from the trees and lose part of prehensile capacity of foot.

In primates, both the extinct and the living ones, the three-dimension shape of the metatarsocuneiform joint is essential for the abduction-rotation movement and for prehensile capacity; moreover, morphologically speaking, this is a spherical joint. When humans become bipeds, this joint is nearly flat. It changes from a moveable joint to a buffering joint, which is crucial for the standing posture.

Many characteristics of primate forefoot, such as first metatarsal pronation, indispensable for prehensile capacity of the foot and arboreal displacement, hypermobility of the first ray, increased angle between first and second metatarsals, and obliquity of the metatarsocuneiform surface, are observed in patients with HV, usually at a severe stage (Figure 6). Therefore, we believe that atavism is a hypothesis to consider in HV pathogenesis.

The development of phalangeal may be influenced by the muscles attached to the hallux. However, first metatarsal pronation arises from the metatarsocuneiform joint, as occurring with apes, and first metatarsal pronation is present in most cases of HV.

It was observed that Neanderthal man have a short first metatarsal, with the characteristics observed in Morton’s ances-

try toe(39), which changes after homo sapiens sapiens, with the presentation of Greek, square, and Egyptian feet.

Current men present with slight variants in the metatarsocuneiform joint. Some of these variants have been related to metatarsus primus varus and HV, which favor an atavistic regression in first ray morphology.

In the book “Patología del antepié”, Viladot(40) studies changes of metatarsal in the pathological anatomy of forefoot in HV and says: “the metatarsal presents with shortening, varus deviation, and pronation, all of which characterizing atavism”.

Not all authors agree with atavistic pathogenesis. Klaue(54) believes that HV cannot be considered an atavism and is actually related with ligamentous failure at the level of the tarsometatarsal joint between the first and the second metatarsals, which may secondarily lead to mechanical overload.

Kilmartin and Wallace(41) asked: Why do not primates have HV? We believe that this is because they have never worn any type of footwear. However, there are some cases of congenital HV in the human species, which does not occur with primates. These authors(41) conclude that atavistic pathogenesis can be neither confirmed nor refused. This opinion is shared by Perera et al.(4).

However, most authors believe that atavism should be considered in the pathogenesis of HV. In a conference on “The problem of Hallux Valgus” held in 1956, which has already been mentioned, Lake(3) approaches the theme of atavism: “[...] divergence of the metatarsal with the associated rotation of the toe brings it into a position reminiscent of the prehensile digit of the anthropoid apes [...]”, and says as follows: “[...] in the apes rotation occurs at the tarsometatarsal and not the metatarsophalangeal joint”.

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Figure 6. A. When in the standing position, the feet of primates are flat; B. Increased mobility of the knee and of the subtalar joint facilitates the passage from pronation to supination; C. The characteristics of the tarsometatarsal joint, especially first metatarsocuneiform obliquity, are required for the first ray to perform a movement of pronation to grasp the branches of the trees (“grasping foot”) and thus to move through the trees (brachiation).
Discussion

Humankind has been presenting with HV for thousands of years, and science has been searching for its specific cause in order to achieve a solution for this condition. Currently, it is possible to say that HV is a three-dimension deformity with a multifactorial pathogenesis. Literature accepts that there is familial sex-linked predisposition to this condition. Constitutional, static, and dynamic factors may lead to the development of HV and be triggered by a “cascade” mechanism, but the initial cause leading to this deformity is still unknown.

First metatarsal pronation, metatarsocuneiform joint obliquity, and first ray hypermobility are key aspects in the development of HV. As previously mentioned, the forefoot of primates exhibits a variety of typical characteristics of HV; thus, we believe that atavism is present in many cases of moderate or severe HV.

Muscular function and changes are highly debatable, and it is often not possible to ascertain whether they are a cause or a consequence of HV. It is logical to think that the treatment of HV would be initiated earlier and be more effective if the etiology of this condition were known. However, it is worth remembering that we are dealing with the world of hypotheses. In our opinion, adopting dogmatic positions on the theme with current knowledge is a serious mistake.

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