

Postulating a pathophysiological association between hallux valgus and plantar venous thrombosis through cadaveric dissections – Geometric measurements pedobarographic and phlebology correlates and literature review

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SUMMARY

This original article postulates a pathophysiological association between two uncommon clinical entities in a cadaveric subject. A female cadaver from a willed body donor was procured for educational and research purposes.

During surgical dissection, the subject was observed to have hallux valgus (HV) and lateral plantar venous thrombosis (PVT) on the left foot. Clinical significance of HV was confirmed by geometric measurements of the great toe and first metatarsal.

Severity of lateral PVT was established by meticulous dissection along full course of the vein and all its grossly accessible tributaries. Dissections in other regions of the same cadaveric subject did not reveal evidence of venous thrombosis elsewhere.

Extensive search of the contemporary literature confirmed HV is most common in elderly females, though not very common in the general population. It also established that isolated PVT is very

rare, the medial type being rarer. No study mentioned both HV and PVT in the same subject or tried to establish a pathophysiological association between both entities.

Analyzing the pedobarographic and phlebology literature on HV, PVT, foot veins, gait, posture, and foot pressure points led the authors to postulate that flattening of medial arch and increased medial forefoot pressure from HV during life in elderly females could render Uhl-Gillot's postulated 'foot-pump' incompetent. Since the principal conduit of the foot-pump is the lateral plantar vein (LPV), ineffective foot-pump could lead to lateral PVT. Considering the paucity of literature on coexisting HV and PVT in living and cadaveric subjects, more studies are required to confirm our hypothesis of this pathophysiological association.

Keywords: Hallux valgus – Plantar venous thrombosis – Cadaveric subject – Foot pump – Pathophysiological association

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INTRODUCTION

A postmortem dissection study is presented wherein both hallux valgus (HV) and plantar venous thrombosis (PVT) coexisted on the same side in an elderly female cadaveric subject. HV *per se* is a well-documented entity, with a plethora of references over the last 90 years (Piggott, 1960; Inman, 1974; Wilson, 1980; Mann, 1993; Perera, 2011; Kohls, 2022). One of the earliest descriptions of HV was in 1928, when they described a conservative lateral ligament release surgery for ‘bunion’, a hoary procedure that carries the surgeon’s name till this day (McBride, 1928). Isolated PVT, on the other hand, has been much less frequently reported than HV, with the exact prevalence still being debatable. One study documented 78 cases of PVT from the 1990’s to the 2020’s (Rastel, 2021). Another study reported less than 20 cases of PVT before 2013 (Karam, 2013). Bernathova (2005) was among the first to demonstrate PVT using sonography and magnetic resonance imaging (MRI). All cases of PVT documented thereafter coincided with advances in radiological techniques (Czihal, 2015; Quinn, 2018; Rastel, 2021). No description of PVT in a cadaveric subject could be found in the literature. Neither did we find the coexistence of HV and PVT in the same individual after extensive search of the literature.

Pathophysiology of calf deep vein thrombosis (DVT) after HV surgery has been well established (Radl, 2003; Saragas, 2014; Kohls, 2022). However, no pathophysiological association between HV and PVT has ever been described or postulated. Therefore, the objective of this study was to postulate a pathophysiological association between HV and PVT in the same individual. To achieve that objective, this study focused on geometric measurements of the cadaveric subject’s foot, analysis of HV-associated gait, followed by correlation with pedobarographic and lower limb phlebology literature (Blomgren, 1991; Bryant, 1999; Menz, 2005; Uhl, 2010). Consistent with the study objective, Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines were considered redundant in this study (Page et al., 2021).

MATERIALS AND METHODS

A cadaveric subject wrapped in custom fixative was procured from Research for Life, Phoenix, AZ. after it was approved for release. The cadaver was a donated non-clinical human tissue, intended for medical research and education purpose only, with permission to be exported, with the dissected remains imported back to the lending institution in the United States for cremation under government regulations stipulated under the Pan American Health Organization (PAHO) resolution of October 1996 XVII Conference. A donor form giving permission from the executor of the deceased is on file at the lending institution. This form may not be released to anyone because of the United States HIPAA (Health Insurance Portability and Accountability) Law, which was enacted to give privacy in the medical field to all patient information.

As per legally disclosed information at the receiving institution, at the end of life the cadaveric subject was an 84-year-old Caucasian female, 5 feet tall, weighing 70 pounds. Serology tests on the cadaver for hepatitis B surface antigen, anti-hepatitis C virus antibody and anti-human immunodeficiency virus (HIV) 1 and 2 antibodies were non-reactive or negative. The cause of death of the subject was a stroke.

The cadaveric subject was placed in a prone position such that both feet were plantar flexed, heels were pointing up and soles were facing out. The position of the toes and the condition of the skin of the sole in both feet were noted. A curvilinear, inverted U-shaped incision was made on the left foot from lateral to medial, bridging across the calcaneal tuberosity. The skin of the sole was carefully dissected out from the underlying plantar aponeurosis (PA) and lifted as a flap based on heads of the metatarsal (MT) bones. The preliminary findings superficial to PA and plantar fasciae were noted. Next, the PA was incised at its proximal attachment to the calcaneus and carefully dissected off to expose the structures in first anatomical layer of sole.

The flexor digitorum brevis (FDB) was released from its proximal attachment to the calcaneus and reflected distally, based on its distal digital attach-

ments. Next, the abductor digiti minimi (ADM) was incised at its distal attachment and reflected laterally, thereby completely exposing the structures deep to the first anatomical layer of the sole. Finally, the lateral plantar vein (LPV), artery and nerve, and their branches and tributaries were meticulously dissected out to their maximum extent from their origin medial to the ankle to their grossly visible limits distally and deeply. The condition of these neurovascular structures as well as the orientation of the bones of the forefoot were noted. The dissected tissues were sprayed with proprietary Carolinas Perfect Solution® (www.carolina.com) to preserve the tissues, prevent desiccation and enable high-contrast image acquisition.

Using dual iPhone 13 camera, with 12-megapixel (MP), f/1.6, 26 mm (wide), 1.7 μ m, dual pixel phase-detection autofocus (PDAF) features, and sensor-shift optical image stabilization (OIS) 12-MP, f/2.4, 13 mm, 120° (ultrawide) capabilities, all findings were graphically documented in landscape mode in high resolution and contrast.

A high-resolution image of the dissected sole of left foot was scaled to its real size with Microsoft Photos (©2022 Microsoft). Black circles were drawn on the image to denote the location of medial and lateral sesamoid bones under the head

of first MT, which were visible and palpable. Next, one red line was drawn on the scaled image along the long axis of the proximal digit of the left great toe and extended proximally till the head of the first MT. A second red line was drawn along the long axis of the first MT on the same image (Fig. 1). Next, this image was superimposed on the image of an Online Protractor (OP) from an online app Ginifab® that was freely available on the Web (copyright © www.ginifab.com). The digital controls on the app were adjusted till the orientation and lines on the OP matched the lines drawn on the image. The angulation between the two red lines was noted on the OP (Fig. 2 / Graphic 1).

Extensive search of the literature published from the 1920's onwards was conducted. The keywords used in search query were, but not limited to: 'plantar vein', 'plantar venous thrombosis', 'plantar vein thrombosis pathophysiology', 'hallux valgus', 'plantar vein thrombosis AND hallux valgus', 'hallux valgus and gait', 'hallux valgus and foot pressure', 'hallux valgus and posture', 'hallux valgus AND deep vein thrombosis'. The search databases and repositories included Pubmed, Medline, Google Scholar, Public Library of Science (PLOS) and ScienceDirect, among others.

The search queries were structured to extract the contemporary pedobarographic and lower

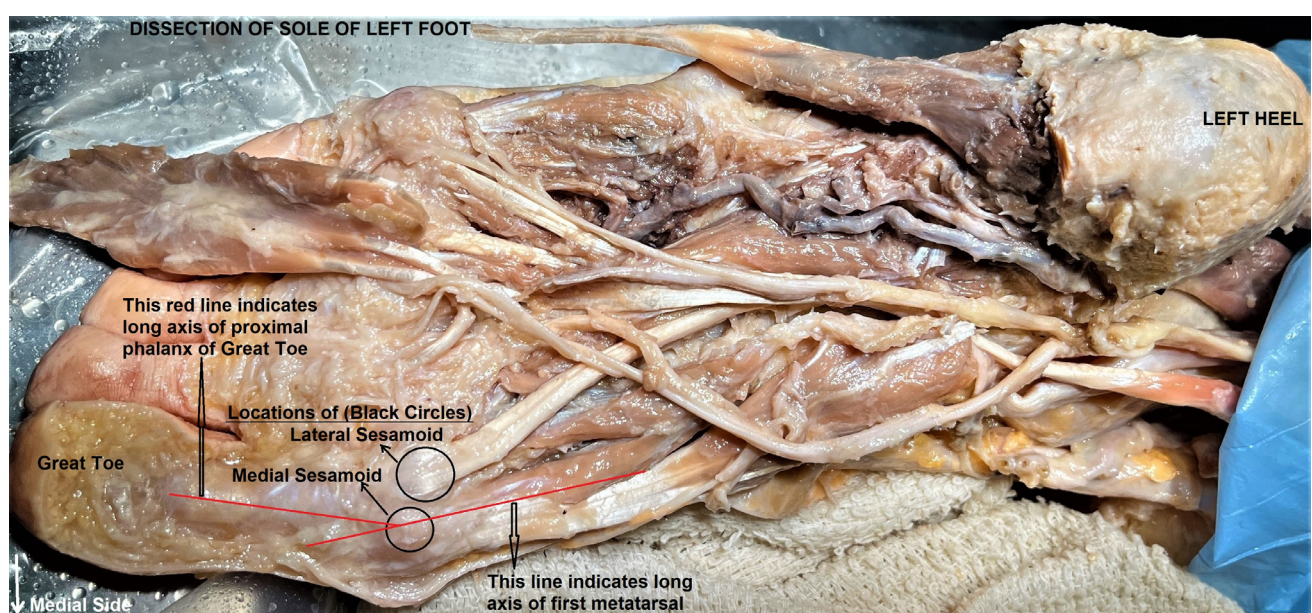


Fig. 1.- Image of dissected sole of left foot to demonstrate how HV angle was delimited on the image and elucidated in the subject's foot. Plantar aponeurosis has been removed. Black circles denote location of sesamoid bones, which are displaced laterally, under head of the medially deviated first MT. Distal red line indicates axis of proximal phalanx of great toe. Proximal red line denotes long axis of first MT. Acute angle between the red lines denotes the HV angle.

limb phlebology literature in relation to HV and PVT. This study was not intended to be a systematic review or meta-analysis of HV and PVT literature. Hence, Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines (Page et al., 2021) were considered redundant in this study.

RESULTS

The first level of dissection of the sole of the left foot revealed numerous punctate and linear bluish-black discolorations, extending from the medial side of the ankle, going under the PA, then progressing along the lateral aspect of the sole till the distal end of the fifth MT. These discolorations were observed piercing through the lateral plantar but not the medial plantar fascia. A few punc-

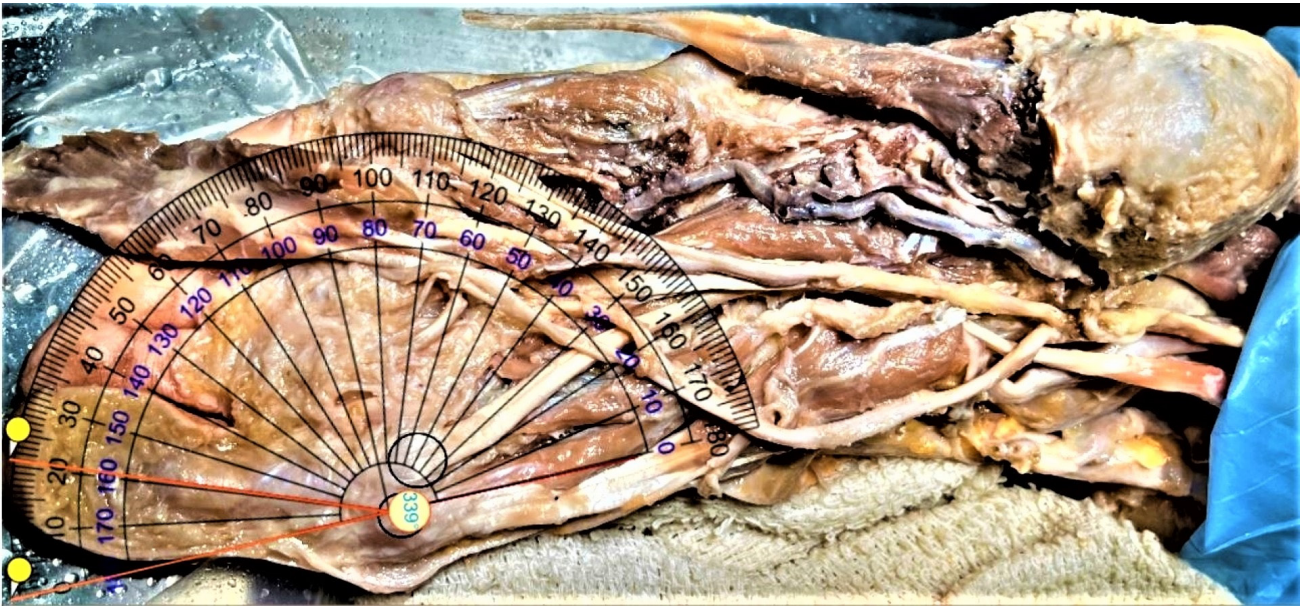
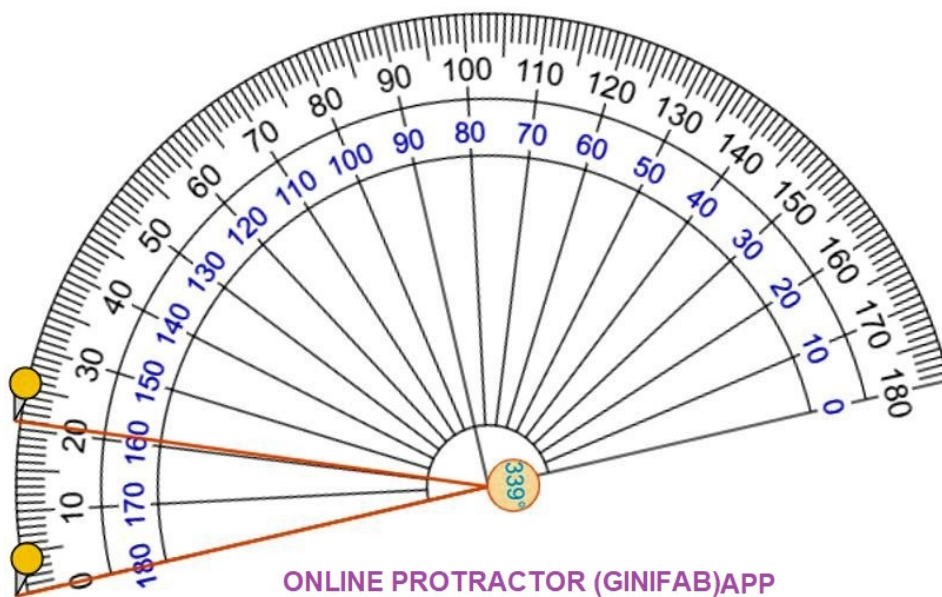


Fig. 2.- Image of dissected sole of foot to demonstrate how HV angle was recorded using the online app OP from Ginifab©. The image of OP is superimposed on image of Fig. 1. Coordinates of OP are adjusted to match the red lines drawn in Fig. 1. The acute angle between long axis of first MT and proximal phalanx of great toe was calculated as $21^\circ (= 360^\circ - 339^\circ)$. This is suggestive of clinically significant HV (Normal HV angle is $<15^\circ$).



Graphic 1. Isolated graphic of OP from Ginifab© showing angulation between long axis of first MT and proximal phalanx of great toe. 339° subtracted from 360° gives 21° , which is the angle between long axis of first MT and long axis of proximal phalanx of great toes. This is suggestive of clinically significant HV (Normal HV angle is $<15^\circ$).

tate spots were also observed piercing through the proximal part of the PA, but most were distributed along its lateral margin (Fig. 3). The sole of the right foot appeared clinically normal.

Deeper dissection under the PA, and especially under the first anatomical layer of the sole, revealed extensively thrombosed LPV along its entire extent, starting proximally superficial and medial to the ankle, and extending distally and laterally. The proximal part of the thrombosed LPV was in the surgical plane between FDB and qua-

dratus plantae (QP) muscles, which are typically characterized as the first and second anatomical layers of the sole respectively (Moore et al., 2021). The thrombotic processes even included the superficial, deep, and muscular tributaries of LPV. Thrombosis of muscular tributaries of LPV from FDB and proximal part of ADM muscles had rendered a bluish-black discoloration to these muscles as well. The left medial plantar vein was very small and otherwise normal. The veins in other areas of the body were normal (Fig. 4).

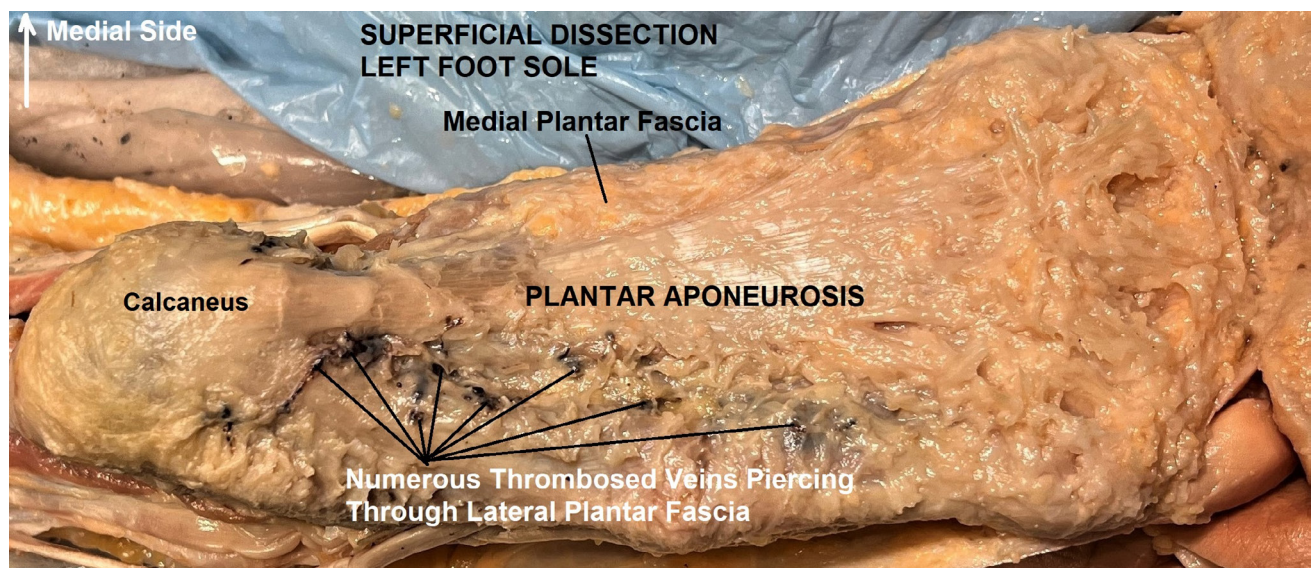


Fig. 3.- Image of dissected sole of left foot immediately after removal of the skin of sole. Thrombosed veins are visible extending from medial side of left ankle and progressing to lateral side of left foot. Most of the thrombosed venules are seen piercing through the lateral plantar fascia but not through medial plantar fascia. Some thrombosed veins are piercing through the proximal part of PA also.

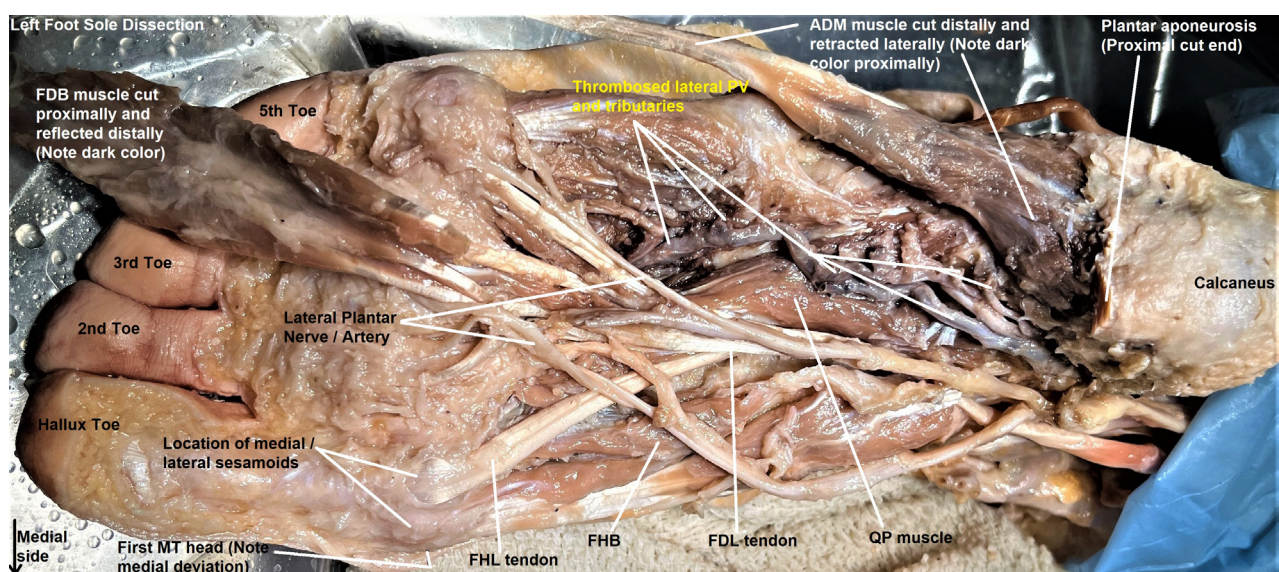


Fig. 4.- Image of dissected sole of left foot, showing structures in first and second anatomical layers of sole. PA has been excised off. FDB has been incised at its proximal attachment and reflected distally. ADM has been incised at its distal attachment and reflected laterally. Head of first MT is deviated medially. FHL tendon is over-riding the lateral sesamoid bone under first MT head. Entire LPV is dilated, tortuous and thrombosed. Thrombosis extends to its deep and muscular tributaries. FDB and ADM muscles are discolored by the thrombotic process.

The head of the left first MT was deviated medially, and the two sesamoid bones under the head of the same MT were displaced laterally. The left flexor hallucis longus (FHL) tendon was over-riding the lateral sesamoid bone instead of passing between the two sesamoids. Distinct angulation was noted between the long axis of the proximal phalanx of the left great toe and the long axis of the first left MT. Measurement with the online app OP on the digital image revealed an angulation of 21° between these two axes (Graphic 1).

DISCUSSION

Hallux valgus

Angulation of less than 15° between the proximal phalanx of the great toe and the first MT is considered normal (Hecht and Lin, 2014; McKean and Park, 2022). With an angulation of 21° in the left foot of our cadaveric subject, a diagnosis of HV is reasonable. This is substantiated by the observation of partial over-riding of the great toe on the second toe in the same subject. This was also visible on gross examination of the right foot in our subject, although the angle was not measured on the right foot. HV *per se* is a well-documented entity, with a plethora of references ranging from the 1920s till the twenty first century (McBride, 1928; Hardy et al., 1951; Haines et al., 1954; Piggott, 1960; Inman, 1974; Wilson, 1980; Mann et al., 1981; Hung et al., 1985; Mann et al., 1993; Coughlin, 1995; Einarsdottir et al., 1995; McDonald, 1996; Torkki et al., 2001; Pique-Vidal et al., 2007; Perera et al., 2011; Reina et al., 2013; Hecht et al., 2014; Loveday, 2020; Kohls, 2022; McKean, 2022). HV has been clinically documented to be more common in elderly females (McKean and Park, 2022). Clinically significant HV subjects also have first intermetatarsal (IM) angle of more than 9° (Hecht and Lin, 2014; McKean and Park, 2022), but the circumstances of this cadaver dissection precluded measurement of IM angle in our subject. HV is associated with progressive lateral shift of sesamoid bones under the head of the first MT, due to bowstringing traction effect by the flexor hallucis brevis (FHB) muscle. As a result, the course of the FHL tendon to the great toe also gets deviated laterally. HV is also associated with

partial flattening of the medial longitudinal arch of the foot (Moore et al., 2021). This may explain the disproportionately higher plantar pressures on the medial forefoot during gait and stance in HV patients (Plank, 1995; Yamamoto et al., 1996; Bryant et al., 1999).

Plantar venous thrombosis

The venous dissection findings in our cadaveric subject are consistent with the diagnosis of lateral PVT. Isolated PVT has been much less frequently reported than HV, with the exact prevalence still being a moot question. One study documented 78 cases of PVT from 1990 to 2022, giving an average prevalence of 2.6 cases per year (Rastel, 2021). However, another study reported less than 20 cases of PVT before 2013, thereby indicating most case reports began appearing during the last two decades (Karam et al., 2013). All clinical cases of PVT documented in the recent years coincided with advances in foot venous compression imaging techniques ranging from duplex ultrasound (DUS) to MRI (Karam et al., 2013; Czihal et al., 2015; Quinn, 2018; Swellengrebel, 2019; Edwards, 2021; Rastel, 2021, Sheikh, 2022). The most remarkable aspect of PVT is its rarity, and the resultant paucity of its reporting in the contemporary literature (Karam et al., 2013; Rastel, 2021).

Roughly two-thirds of cases of PVT are females (Czihal et al., 2015). Since the medial plantar vein was normal, our subject can be considered a case of isolated lateral PVT. Although a few reports have described medial PVT, lateral PVT has a preponderantly higher frequency, ranging from 30% to 96% in different studies, often extending into plantar arch and metatarsal veins, and a quarter of them extending into deep calf veins (Czihal et al., 2015; Vansevenant and Vanhoenacker, 2015; Quinn, 2018; Edwards, 2021; Sheikh et al., 2022). Prior dissections in this cadaver subject had not revealed any evidence of DVT in the calf.

About half of all cases of PVT are idiopathic, although a few may have occult malignancy, as part of paraneoplastic syndrome, or other hypercoagulable states, commensurate with Virchow's triad (Karam et al., 2013; Czihal et al., 2015; Vansevenant and Vanhoenacker, 2015). Few reports

of PVT caused by physical or mechanical strain or local trauma to the foot or use of orthoses have been mentioned in the literature (Czihal et al., 2015; Vansevenant and Vanhoenacker, 2015; Swellengrebel et al., 2019; Rastel, 2021).

Considering calf DVT in general, factors such as age, gender, footwear, genetic predisposition or mutations have all been postulated to play significant roles in their pathogenesis. Numerous studies have established other risk factors of DVT, namely postoperative recumbency, foot surgery, smoking, cardiac conditions, paralytic stroke, family history, and drug usage, among others (Radl et al., 2003; Peterson et al., 2011; Saragas et al., 2014; Masaragian et al., 2019; Loveday, 2020; Kohls, 2022).

Although our cadaveric subject was known to have died from a stroke, it is difficult to speculate the role of any of these factors in the pathophysiology of isolated lateral PVT in our subject. In the absence of any other sites of venous thrombosis like calf DVT, or obvious etiology of isolated lateral PVT, and not knowing the subject's medical history during her lifetime, analysis of pedobarographic and phlebology literature provided vital clues towards a possible pathophysiological association between HV and lateral PVT in our subject.

Foot phlebology analysis

The foot is drained by a medial and lateral venous network respectively, the latter being larger. The two networks are connected by the first IM perforating vein (Uhl and Gillot, 2010; Vansevenant and Vanhoenacker, 2015). LPV, being the larger of the two plantar veins in the sole of the foot, is the main conduit of venous blood flow from the sole. It drains venous blood from the toes and deep MT veins into posterior tibial Venae Comitantes (VC). LPV follows a course between the FDB and QP muscles, which are fleshy muscles of the first and second anatomical layers of the sole of the foot respectively (Uhl and Gillot, 2010; Moore et al., 2021). According to Uhl and Gillot's 'foot-pump' hypothesis (2010) in their prize-winning seminal phlebology work, these muscles are the principal actuators of the foot-pump, among other intrinsic foot muscles. During various phases of normal walking and standing, venous blood

drains sequentially from toes through deep MT veins into the functional 'receptacle' of the LPV, through the latter's physiological distal 'suction pole'. Then the blood is transmitted to the posterior tibial VC through the proximal 'ejection pole' of LPV. This is achieved by the 'milking action' of the intrinsic foot muscles, including the FDB and QP, between which the LPV passes, assisted by normal lateral plantar pressure of longitudinal arch of foot during various phases of gait and stance. The components of the foot-pump are designed to function synchronously and synergistically in normal foot configuration with normal arch supports. In most cases the medial plantar vein is small and does not play a significant role in the foot-pump hemodynamics (Uhl and Gillot, 2010).

Pedobarographic analysis

Pedobarographic studies, which assess foot pressure patterns and distribution during stance and gait, have amply demonstrated abnormal stress patterns on metatarsal heads during various phases of gait in patients with HV. Only one study mentioned increased lateral forefoot pressures during stance and gait in HV patients (Blomgren et al., 1991). Most other studies have recorded increased medial foot pressure patterns, namely significantly higher mean, and peak medial plantar pressures under first three MT heads, especially under first MT head, and increased medial foot contact time in patients with HV. The higher peak pressures under first MT head correlated with higher HV angle and IM angle (Plank, 1995; Yamamoto et al., 1996; Bryant et al., 1999). The slight flattening of the medial longitudinal arch of the foot in patients with HV may explain the higher medial plantar pressures and increased medial foot contact times recorded in these studies (Moore et al, 2021).

Pathophysiological association of HV and PVT

Correlating these phlebology and pedobarographic findings leads us to a possible pathophysiological association between HV and lateral PVT in our subject. Increased medial forefoot plantar pressures are counter-productive to effective functioning of the anatomical and physiological foot-pump (Uhl and Gillot, 2010). Our female ca-

daveric subject with HV would have had partially flattened medial longitudinal arch during her lifetime (Moore et al, 2021). She would have experienced increased medial plantar pressures and increased medial foot contact times during stance and gait (Plank, 1995; Yamamoto et al., 1996; Bryant et al., 1999). It could have led to ineffective functioning of the intrinsic foot muscles, thereby impeding venous return from the toes and deep MT veins into the LPV. Furthermore, inadequate 'milking action' of FDB and QP muscles, between which LPV passes, could have led to failure of functional components of LPV, namely the 'suction pole', 'reservoir', and 'ejection pole' of the foot-pump conduit. Resultant stasis in the LPV could lead to lateral PVT (Uhl and Gillot, 2010). The initiation of lateral PVT may have been from the first perforating IM vein, which connects the medial and lateral plantar venous networks (Vansevenant and Vanhoenacker, 2015; Rastel, 2021). Eventually, lateral PVT may be the precursor of calf DVT in susceptible subjects (Uhl and Gillot, 2010) (Fig. 5).

Gait studies in elderly people with HV have demonstrated temporospatial gait impairment,

instability, and risks of falling, especially when walking on uneven surfaces (Menz and Lord, 2005). Physical or mechanical stress on the foot may potentially precipitate PVT in elderly subjects (Vansevenant and Vanhoenacker, 2015; Rastel, 2021). Therefore, it can be postulated that postural factors may have engendered lateral PVT in our cadaveric subject in a secondary way. Encumbered with HV and advancing age, our subject may have been experiencing abnormal mechanical stresses on her foot during her lifetime, leading to lateral PVT.

CONCLUSION

This study proposes a possible pathophysiological association between HV and PVT in an elderly female cadaveric subject. Given the unique coexistence of HV and lateral PVT in this subject, substantiated by a review of the relevant literature, it is our postulation that clinically significant HV in elderly females may alter posture and gait, leading to incompetence of the anatomical and physiological foot-pump postulated by Uhl and Gillot (2010). Since LPV is the main venous conduit of the foot-pump, and intrinsic foot muscles,

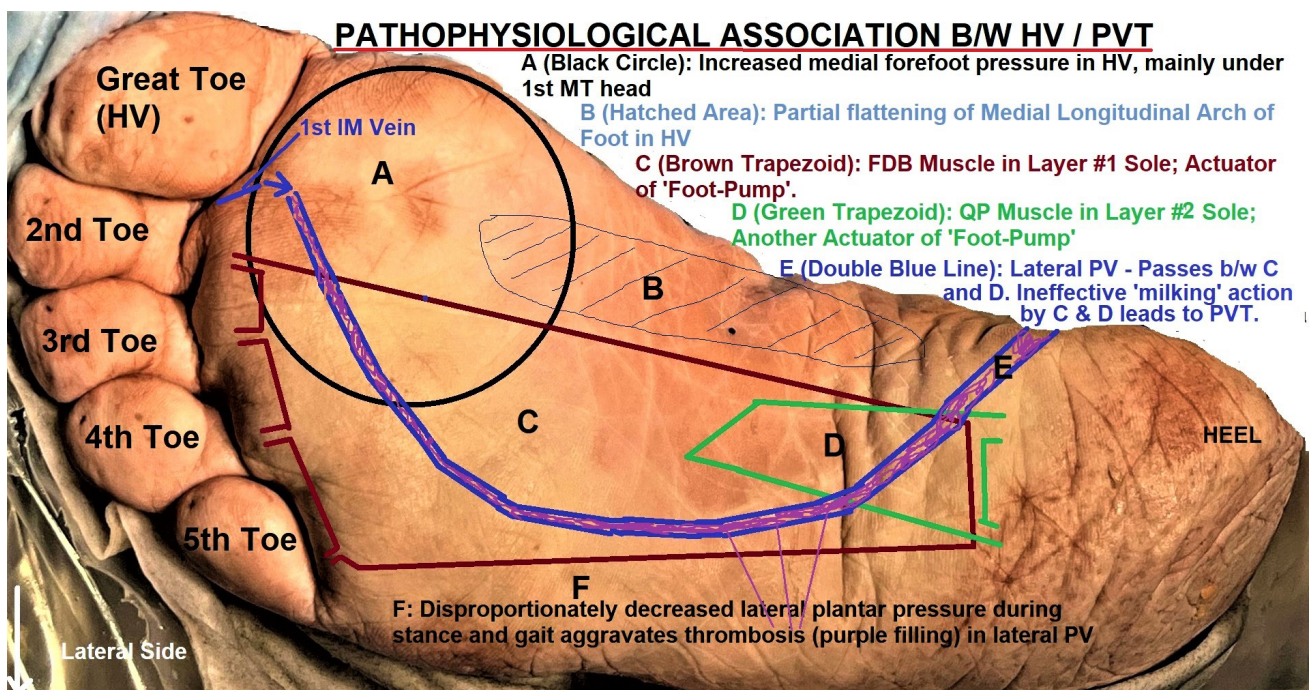


Fig. 5.- Diagrammatic overlay on image of sole of foot to illustrate the 'foot-pump' hypothesis of Uhl and Gillot and the possible pathophysiology association of lateral PVT and HV in this subject. The 'foot-pump' needs properly functioning intrinsic foot muscles, especially FDB and QP in a normally configured and arched foot to propel venous blood effectively through the lateral PV into the posterior tibial VC. HV disrupts the medial longitudinal arch of foot, leads to disproportionate increased medial forefoot pressures, and renders the 'foot-pump' ineffective. This paves the way for PVT and possibly DVT.

especially FDB and QP, are the actuators of the foot- pump, ineffective functioning of the latter can lead to altered venous hemodynamics in foot, leading to lateral PVT.

While pathophysiology of calf DVT and its sequelae have been documented and described *ad infinitum*, the literature is rather sparse in the context of foot PVT and its etiopathogenesis. It is almost nonexistent regarding specific pathophysiological association between HV and PVT, notably because of the relative rarity of both individually, and especially in combination, in the general population. Therefore, more pedobarographic and phlebology studies are required in living subjects with coexisting HV and PVT, for our pathophysiological association postulation to be confirmed.

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REFERENCES

- BLOMGREN M, TURAN I, AGADIR M (1991) Gait analysis in hallux valgus. *J Foot Surg*, 30(1): 70-71.
- BRYANT A, TINLEY P, SINGER K (1999) Plantar pressure distribution in normal, hallux valgus and hallux limitus feet. *Foot*, 9(3): 115-119.
- COUGHLIN MJ, MANN RA (1995) Juvenile hallux valgus: etiology and treatment. *Foot Ankle Int*, 16: 682-697.
- CZIHAL M, RÖLING J, RADEMACHER A, SCHRÖTTLE A, KUHLENCORDT P, HOFFMANN U (2015) Clinical characteristics and course of plantar vein thrombosis: a series of 22 cases. *Phlebology*, 30(10): 714-718.
- EDWARDS SR (2021) Plantar vein thrombosis masquerading as plantar fasciitis: A case report. *Phlebology*, 36(2): 160-162.
- EINARSDOTTIR H, TROELL S, WYKMAN A (1995) Hallux valgus in ballet dancers: a myth? *Foot Ankle Int*, 16: 92-94.
- HAINES RW, MC DA (1954) The anatomy of hallux valgus. *J Bone Joint Surg Am*, 36: 272-293.
- HARDY RH, CLAPHAM JC (1951) Observations on hallux valgus; based on a controlled series. *J Bone Joint Surg Br*; 33: 376-391.
- HECHT PJ, LIN TJ (2014) Hallux Valgus. *Med Clin North Am*, 98(2): 227-232.

- HUNG LK, HO YF, LEUNG PC (1985) Survey of foot deformities among 166 geriatric inpatients. *Foot Ankle*, 5: 156-164.
- INMAN VT (1974) Hallux valgus: a review of etiologic factors. *Orthop Clin North Am*, 5: 59-66.
- KARAM L, TABELT G, NAKAD J, GERARD JL (2013) Spontaneous plantar vein thrombosis: state of the art. *Phlebology*, 28(8): 432-437.
- KOHL S editor (2022) Foot & Ankle Surgery – Hallux Valgus Correction. Foot Forward Clinic – One Orthopaedics, UK.
- LOVEDAY DT editor (2020) Bunions (Hallux Valgus). Norfolk and Norwich University Hospitals NHS Foundation Trust, UK.
- MANN RA, COUGHLIN MJ (1981) Hallux valgus—etiology, anatomy, treatment and surgical considerations. *Clin Orthop Relat Res*, 157: 31-41.
- MANN R, COUGHLIN M (1993) Adult hallux valgus, surgery of the foot and ankle. 1. CV Mosby, St Louis, MO, USA, pp 204-216.
- MASARAGIAN H, PERIN F, CORIA H, MIZDRAJI L, AMERISO N, REGA L (2019) A prophylaxis algorithm for deep vein thrombosis in ankle and foot surgery. *Rev Asoc Argent Ortop Traumatol*, 84(3): 252-259.
- MCBRIDE ED (1928) A conservative operation for bunions. *J Bone Joint Surg*, 10: 14.
- MCDONALD MG, STEVENS DB (1996) Modified Mitchell bunionectomy for management of adolescent hallux valgus. *Clin Orthop Relat Res*, 332: 163-169.
- MCKEAN J, PARK J, editors (2022) Hallux Valgus – Foot and Ankle. Ortho Bullets. Santa Barbara, CA, USA.
- MENZ HB, LORD SR (2005) Gait instability in older people with hallux valgus. *Foot Ankle Intl*, 26(6): 483-489.
- MOORE KL, DALLEY AF, AGUR AMR, editors (2021) Clinically Oriented Anatomy. Lippincott Williams Wilkins, Philadelphia, USA.
- PAGE MJ, MCKENZIE JE, BOSSUYT PM, BOUTRON I, HOFFMANN TC, MULROW CD, SHAMSEER L, TETZLAFF JM, AKL EA, BRENNAN SE, CHOU R, GLANVILLE J, GRIMSHAW JM, HROBJARTSSON A, LALU MM, LI T, LODER EW, MAYO-WILSON E, MCDONALD S, MCGUINNESS LA, STEWART LA, THOMAS J, TRICCO AC, WELCH VA, WHITING P, MOHER D (2021) The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. *BMJ*, 372: n71.
- PERERA AM, MASON L, STEPHENS MM (2011) The pathogenesis of hallux valgus. *J Bone Joint Surg Am*, 93: 1650-1661.
- PETERSON KS, MENDICINO RW, CATANZARITI AR, SALTRICK KR (2011) Deep vein thrombosis after bunionectomy: a case report of two genetic mutations. *J Foot Ankle Surg*, 50(6): 733-735.
- PIGGOTTH (1960) The natural history of hallux valgus in adolescence and early adult life. *J Bone Joint Surg Br*; 42: 749-760.
- PIQUE-VIDAL C, SOLE MT, ANTICH J (2007) Hallux valgus inheritance: pedigree research in 350 patients with bunion deformity. *J Foot Ankle Surg*, 46: 149-154.
- PLANK MJ (1995) The pattern of forefoot pressure distribution in hallux valgus. *Foot*, 5(1): 8-14.
- QUINN SF editor (2018) Plantar Vein Thrombosis. MRI Web Clinic, Radsourc Brentwood TN, USA.
- RADL R, KASTNER N, AIGNER C, PORTUGALLER H, SCHREYER H, WINDHAGER R (2003) Venous thrombosis after hallux valgus surgery. *J Bone Joint Surg Am*, 85(7): 1204-1208.
- RASTEL D (2021) Four new cases of isolated foot vein thrombosis: Is the first metatarsal interspace perforator responsible? *J Médecine Vasculaire*, 46(3): 114-122.
- REINA M, LAFUENTE G, MUNUERA PV (2013) Effect of custom-made foot orthoses in female hallux valgus after one-year follow up. *Prosthet Orthot Int*, 37: 113-119.
- SARAGAS NP, FERRAO PNF, SARAGAS E, JACOBSON BF (2014) Venous thromboembolic disease in hallux surgery. *SA Orthop J*, 13(3): 28-31.

SHEIKHRN, NGUYEN C, CUMMINS M (2022) Plantar vein thrombosis in the medial branch of the posterior tibial vein: a case report. *J Am Podiatr Med Assoc*, 112(2): 20-158.

SWELLENGREBEL HJC, BACKUS T, ZIJTA FM, VAN DER ZWAAL P (2019) Plantar vein thrombosis provoked by mechanical strain to the foot: a rare cause of plantar heel pain. *BMJ Case Rep*, 12(11): e230054.

TORKKI M, MALMIVAARA A, SEITSALO S (2001) Surgery vs orthosis vs watchful waiting for hallux valgus: a randomized controlled trial. *JAMA*, 285: 2474-2480.

UHL J-F, GILLOT C (2010) The plantar venous pump: Anatomy and physiological hypotheses. *Phlebology*, 17(3): 151-158.

VANSEVENANT M, VANHOENACKER FM (2015) Plantar vein thrombosis: an unusual cause of plantar pain. *J Belgian Soc Radiol*, 99(2): 98-101.

WILSON DW (1980) Treatment of hallux valgus and bunions. *Br J Hosp Med*, 24: 548-549.

YAMAMOTO H, MUNETA T, ASAHINA S, FURUYA K (1996) Forefoot pressures during walking in feet afflicted with hallux valgus. *Clin Orthop Rel Res*, 323: 247-253.