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Posterior Ankle Impingement: It's Not Only About the Os Trigonum

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Abstract

Background: Os trigonum and Stieda process are common etiologies for posterior ankle impingement syndrome (PAIS), and diagnosis is typically made by radiographs, computed tomographic, or magnetic resonance imaging. However, these static tests may not detect associated soft tissue and bony pathologies. Posterior ankle and hindfoot arthroscopy (PAHA) is dynamic, providing at least ×8 magnification with full anatomical visualization. The primary aim of this study was to report the prevalence of associated conditions seen with trigonal impingement treated with PAHA.

Methods: In this retrospective comparative study, patients who underwent PAHA for PAIS due to trigonal impingement, from January 2011 to September 2016, were reviewed. Concomitant open posterior procedures and other indications for PAHA were excluded. Demographic data were collected with pre- and postoperative diagnosis, arthroscopic findings, type of impingement, location, associated procedures, and anatomical etiologies. Trigonal impingements were divided in os trigonal or Stieda and subgrouped as isolated, with flexor hallucis longus (FHL) disorders, with FHL plus other impingement, and with other impingement lesions.

Results: A total of 111 ankles were studied—74 os trigonum and 37 Stieda. Isolated trigonal disorders accounted for 15.3% of PAIS (n = 17). Cases having associated conditions had a mode of 3 additional pathologies. FHL disorders were found in 69.4%, subtalar impingement in 32.4%, posteromedial ankle synovitis in 25.2%, posterolateral ankle synovitis in 22.5%, and posterior inferior tibiofibular ligament impingement in 19.8% of cases. Associated pathologies were observed in 58.6% of cases when FHL was not considered. Significant differences were noted comparing os and Stieda (isolated: 20.3% to 5.4%, $P = .040$; FHL plus others: 35.1% to 59.5%, $P = .015$).

Conclusion: Trigonal bone (os trigonum or Stieda) was found to cause impingement in isolation in a small proportion of cases even when the FHL was considered part of the same disease spectrum. This should alert surgeons when considering removing trigonal impingement. Open approaches may limit the visualization and assessment of associated posterior ankle and subtalar pathoanatomy, thus possibly overlooking concomitant causes of PAIS.

Level of Evidence: Level III, retrospective comparative study.

Keywords: posterior ankle impingement, endoscopy, hindfoot, os trigonum, trigonal, Stieda, associated, combined

Introduction

Trigonal (Stieda) process and os trigonum are recognized as entities behind posterior ankle impingement syndrome (PAIS).[12,22] Several studies have assigned trigonal disorders as the main etiologies behind this disease.[22,31-33] Usually PAIS is suspected clinically, in a combination of history and careful physical examination, and confirmed with subsidiary tests.[7,25] Conventional radiographs, computed tomography, and magnetic resonance imaging are static modalities that can portray local anatomy and indirect signs of impingement but lack the dynamic capability to indicate which structure is affected and causing symptoms.[4,20,33,35] The high prevalence of os trigonum in imaging examinations also places it as potential incidental finindgs.[2,35] Conversely, ultrasonography provides the ability for dynamic testing (imaging structures

during physical examination maneuvers) that can reproduce clinical symptoms, but visualization can be constrained by depth, definition, and the experience of the operator.[14,23]

Surgery is commonly indicated when nonoperative treatments such as rest/immobilization, injections, and nonsteroidal antiinflammatory medications fail to relieve pain.[9,27] The physical examination finding of posterior impingement, and studies that demonstrate the bony trigonal impingement, focus attention on the removal of these bony structures as the single goal in the treatment.[2,30,37] The concomitant role of the flexor hallucis longus (FHL) tendon as an additional pain generator is described as a common associated finding, which is also treated by resection of the process.[15,30] However, recent literature have been pointing to various other anatomic structures that could be etiologies of PAIS.[5] Ligamentous, peri-articular bony impingement, synovitis, and capsular structures could also cause impingement in isolation or in combination with trigonal disorders.[5] Failure to address these potential disorders could result in residual or recurrent symptoms.[37] A recent systematic review on surgically treated PAIS found mild to poor satisfaction in endoscopic procedures around 9% and 14% in open surgeries.[37]

Although the general reproduction of impingement pain can be clearly diagnosed preoperatively by physical examination, the specific structure(s) causing symptoms are not always evident.[2,3] Posterior ankle and hindfoot arthroscopy (PAHA) is an established approach to treat PAIS.[5,15,37] The increased magnification, field of view, and real-time dynamics that an arthroscope provides allows assessment of other anatomic structures as potential causes of impingement, in addition to Stieda impingement.[5,26,30] The real-time visualization of impingement lesions with joint motion, probe examination, and palpation/pressure over different locations of the posterior ankle is the most direct evidence of impingement.[5,28] Although the general impingement condition can be clearly diagnosed preoperatively, the specific structure causing symptoms is not always evident. Therefore, the primary objective of this study was to report the prevalence of associated conditions in a large cohort of os trigonum and Stieda impingement treated with PAHA. We hypothesized that the proportion of isolated trigonal disorder in PAIS would be low, even when the FHL was not considered a concomitant pathological structure.

Materials and Methods

Design

This was a retrospective comparative study that obtained local IRB approval (IRB 201904825) and followed all requirements from the Health Insurance Portability and Accountability Act (HIPAA) and the Declaration of Helsinki.

Sample

Consecutive patients with clinical diagnosis of os trigonal or Stieda process causing PAIS and a surgical indication for a PAHA from January 2011 to September 2016 were analyzed. The sample was obtained by accessibility. Clinical and radiographic PAIS diagnosis due to trigonal (os or Stieda) impingement that were treated by a PAHA procedure were established as inclusion criteria.[6,21] Subjects were included only if a trigonal process was elected as the primary preoperative diagnosis and resected (Figure 1). Other causes for PAHA, Haglund resections, or associated open posterior procedures to the ankle and subtalar joints were excluded. Bilateral and revision procedures were not removed from the studied cohort.

Surgical Technique

Surgeries were performed by 3 fellowship-trained orthopaedic foot and ankle surgeons each with more than 10 years of experience performing PAHA. The employed arthroscopic assessment and technique were generally the same.[5] Most procedures were preceded with a peripheral block and were performed under general anesthesia. Patients were placed in prone with a tight tourniquet. No traction was used. A 4.0-mm 30-degree scope was used and a 4.0-mm 70-degree arthroscope occasionally used. The posterolateral portal was created at a level just proximal to the tip of the lateral malleolus, paramedian to the Achilles tendon. Initially aiming to the third web space as the deep compartment fascia was perforated with a straight hemostat and then directed toward the posterolateral recess of the subtalar joint; the posteromedial portal was generated paramedian to the Achilles tendon and

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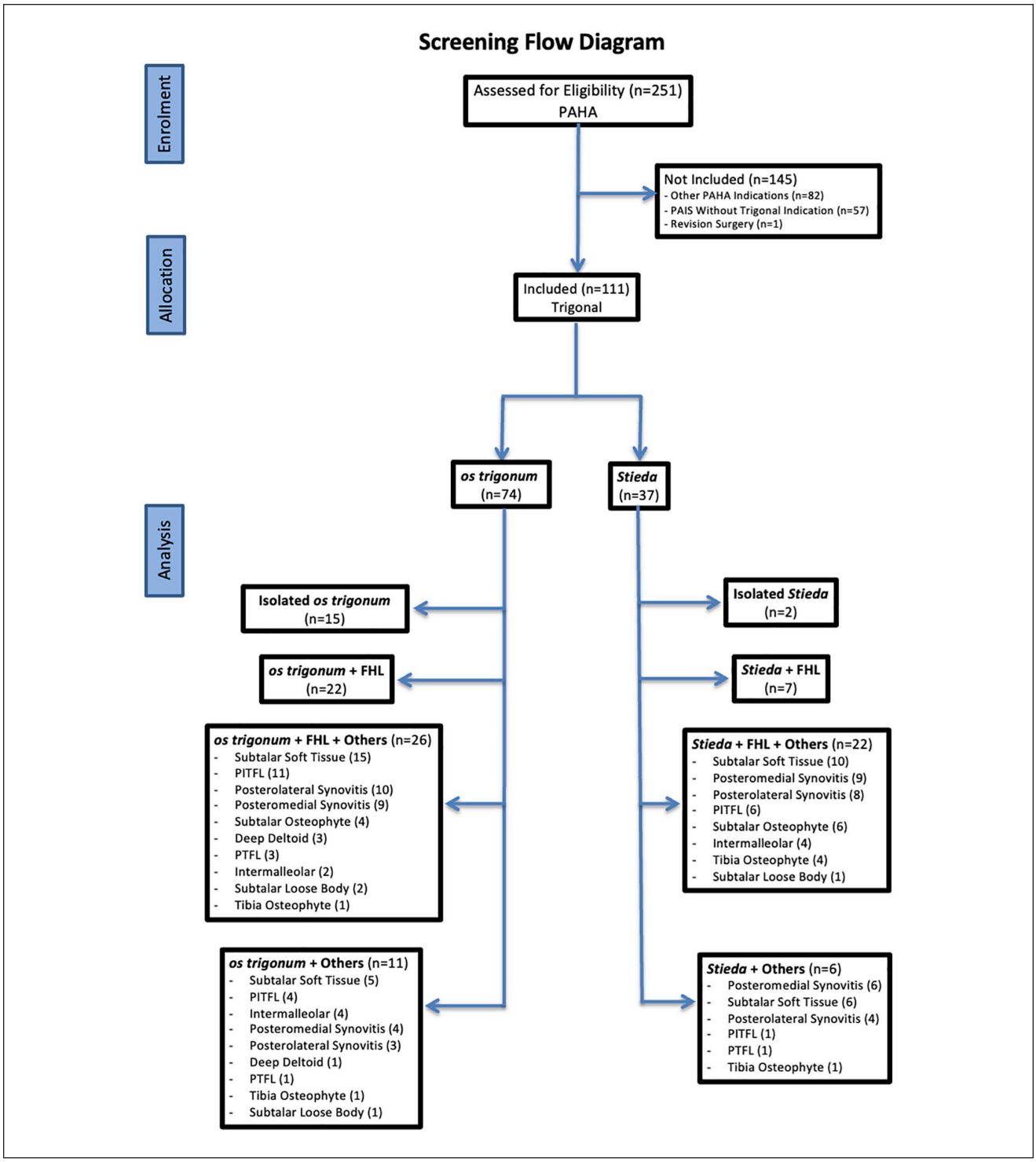

Figure 1. Study's flow diagram. FHL, flexor hallucis longus; PAHA, posterior ankle and hindfoot arthroscopy; PAIS, posterior ankle impingement syndrome; PITFL, posterior inferior tibiofibular ligament; PTFL, posterior talofibular ligament.

directed toward the posterolateral recess of the subtalar joint. Instrument and working portals were interchanged as needed.

Superficial debridement of posterior extraarticular soft tissues (adipose, fibrous scar tissue, and posterior extracapsular ligaments, such as the fibulotalocalcaneal ligament of

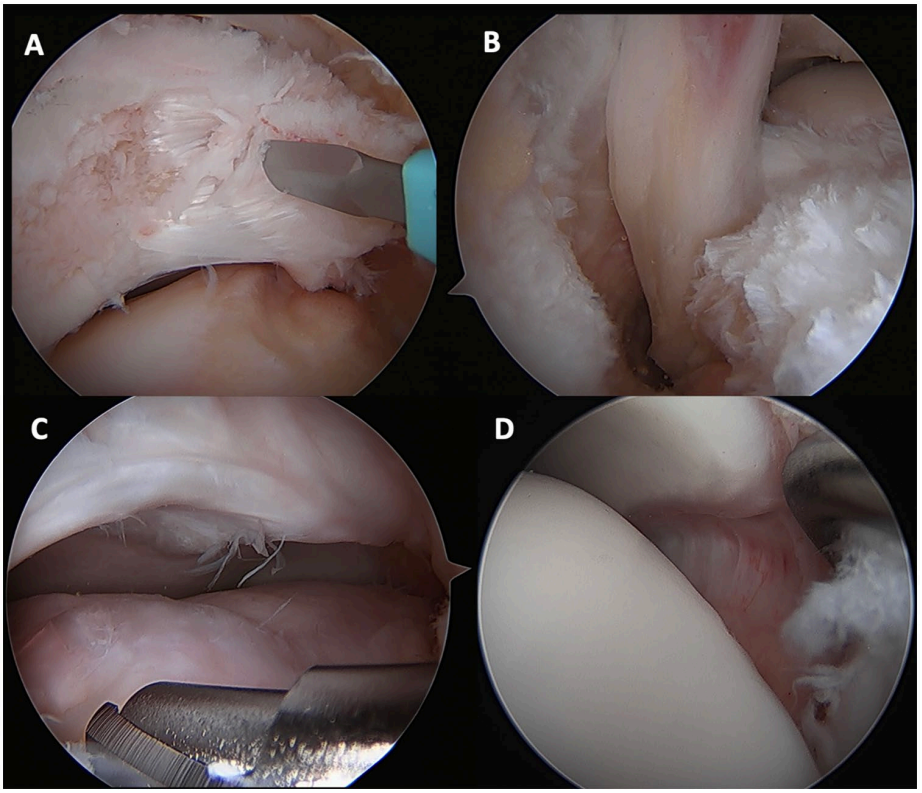

Figure 2. Examples of posterior ankle hindfoot arthroscopy (PAHA) findings associated with a trigonal disorder. (A) Removal of the os trigonum ossiculum, (B) flexor hallucis longus (FHL) stenosis release, (C) posterior inferior tibiofibular ligament (PITFL) impingement, and (D) deep deltoid impingement.

Rouvière Canela Lazaro) was accomplished in a lateral-to-medial fashion to enable visualization of the deep structures.[11] The intermalleolar ligament (posterior capsule of the ankle) was usually torn from chronic trigonal impingement on the posterior tibia in plantarflexion, and it was routinely resected to both the medial and lateral origins. The deep transverse portion of the posterior inferior tibiofibular ligament was tested for instability, and if it was able to be displaced into the ankle joint because of detachment from the posterior tibia, it was also resected. This was often associated with grade 2 chondromalacia at the most posterior articular surface of the talus. The os trigonum or trigonal impingement lesion were then resected, and confirmation of relief of impingement was performed with plantarflexion and direct visualization. The FHL was typically surrounded by tenosynovitis or chronic fibrotic tenosynovium. Stenosis of the FHL muscle was often noted to be low lying with bulging on the proximal retinacular attachment on the talus with dorsiflexion of the ankle and hallux. Release of this was always required for os trigonum excision and often performed with Stieda process excision when muscle stenosis or findings of acute or chronic inflammation were noted. Chronic fibrotic tenosynovium was often resected. Additional minor resection of the extraarticular talar bone posterior to the articular margin was performed when it was noted to be impinging on the tibia as well. Whether this was due to native posterior cam-shaped talar bone, reactive change due to chronic impingement, and/or joint instability it is not clear.[1] Subsequently, posteromedial and posterolateral recesses and gutters of the ankle were inspected for soft tissue impingement, often due to synovitis and/or torn deep deltoid, posterior talofibular ligament and these were resected. The scope placed through the posterolateral portal allowed for direct visualization of the posteromedial recess/gutter and a fine-tipped shaver through the posteromedial portal was sufficient for debriding this area while protecting the FHL with hallux plantarflexion. The posterior subtalar joint is readily visible along with calcaneal articular surface after resection of the os trigonum or Stieda process. The posterolateral recess of the subtalar joint inferior to the PTFL and lateral gutter medial to the calcaneal fibular ligament were inspected and soft tissue impingement debrided as necessary. Throughout the procedure, dynamic examination with digital pressure or probe examination were used to determine the potential for impingement of various soft tissue or bony lesions (Figure 2). The portals were closed and

Table 1. Associated Disorders Distribution by Group (Os Trigonum and Stieda) in the Posterior Ankle Hindfoot Arthroscopy (PAHA) Study's Cohort.[a]

Diagnosis	Os Trigonum (n = 74)		Stieda (n = 37)		*P* Value	Total (n = 111)	
	n	%	n	%		n	%
FHL disorders	48	64.9	29	78.4	.145	77	69.4
Other conditions	37	50.0	28	75.7	**.010**	65	58.6
Subtalar soft tissue	20	27.0	16	43.2	.085	36	32.4
PM ankle synovitis	13	17.6	15	40.5	**.009**	28	25.2
PL ankle synovitis	13	17.6	12	32.4	.077	25	22.5
PITFL impingement	15	20.3	7	18.9	.866	22	19.8
IML impingement	6	8.1	4	10.8	.639	10	9.0
Subtalar osteophyte	4	5.4	6	16.2	.061	10	9.0
Tibia osteophyte	2	2.7	5	13.5	**.027**	7	6.3
PTFL impingement	4	5.4	1	2.7	.518	5	4.5
Deltoid impingement	4	5.4	0	0.0	.150	4	3.6
Subtalar loose body	3	4.1	1	2.7	.719	4	3.6

Abbreviations: Deltoid, posterior deep deltoid; FHL, flexor hallucis longus; IML, intermalleolar ligament; PITFL, posterior inferior tibiofibular ligament; PL, posterolateral; PM, posteromedial; PTFL, posterior talofibular ligament.
[a]*P* values when groups are compared per concomitant finding. Values in bold represent statistical significance.

a bulky dressing with a boot placed. Patients were allowed partial weightbearing and start range of motion at day 3.[5]

Assessments

A fellowship-trained orthopaedic foot and ankle surgeon with no surgical participation retrospectively evaluated the cases. Demographic characteristics were collected. Preoperative and postoperative diagnosis, arthroscopic findings, type of impingement, location of the disorder, associated procedures, and anatomical etiologies were gathered. Trigonal impingements were allocated in 2 groups, os trigonal or Stieda. They were then analyzed accordingly to the adjunctive structures found during the arthroscopies. Thus, the groups were further subgrouped in correspondence to the patterns found: as occurring in isolation, with FHL disorders, with FHL plus other impingement lesions, and with other impingement lesions (Figure 1).

Statistical Analysis

Data normality of continuous variables was assessed by the Shapiro-Wilk test. Descriptive statistics were reported with means and standard deviations for continuous variables and frequency (percentages) for categorical variables. Differences between groups with isolated trigonal impingement and those with associated pathologies were determined by distribution comparison. Pearson χ^2 and Fisher exact test were used to compare subgroups. Significant differences were established for *P* values below .05. Data were analyzed using SPSS software version 29 (IBM Corp, Armonk, NY).

Results

From an initial sample of 251 ankles (Figure 1), 111 ankles (46 right, 55 left, 5 bilateral) were included in the study (106 patients: 58 females, 48 males), having a mean age of 31.0 years (range: 12-70) and a BMI of 30.4 (SD: ±9.2). Athletes accounted for 40.6% and nonathletes 59.4% of subjects (Appendix). After allocation, 74 ankles (66.7%) were placed in the os trigonum group and 37 (33.3%) in the Stieda group ($P < .001$).

Isolated trigonal disorders accounted for 15.3% (n = 17) of the total PAIS occurrences. Inside groups, they were 20.3% (n = 15) in the os group and 5.4% (n = 2) in the Stieda group ($P = .040$). In the entire cohort, cases presenting concomitant disorders (84.7%, n = 94), had a mode of 3 (1-5) additional pathologies. Three or more adjunctive findings needing treatment were seen in 38.8% (n = 42) of all ankles. The most common associated conditions were FHL disorders in 69.3%, subtalar soft tissue impingement in 32.4%, posteromedial ankle synovitis in 25.2%, posterolateral ankle synovitis in 22.5%, and posterior inferior tibiofibular ligament impingement in 19.8% of cases (Table 1). Even when FHL disorders were removed from analyses as an associated condition, 58.6% of the trigonal sample had a concomitant pathologic finding, with a higher percentage in the Stieda (75.7%) compared with os group (50%, $P = .010$).

Comparison between subgroups' prevalence (Table 2) per bone-impingement type (os trigonum vs Stieda) portrayed differences only when assessing the FHL with other conditions (35.1% vs 59.5% respectively, $P = .013$) subgroup. The FHL subgroup (29.7% vs 18.9%, $P = .237$) and the other conditions subgroup (14.9% vs 16.2%, $P = .830$) prevalence were similar amid os trigonum and Stieda.

Table 2. Subgroups Analysis by Bone Type (Os Trigonum and Stieda) in the Posterior Ankle Hindfoot Arthroscopy (PAHA) Study's Cohort.[a]

Subgroups	Os Trigonum (n = 74)		Stieda (n = 37)		*P* Value	Total (n = 111)	
	n	%	n	%		n	%
Isolated	15	20.3	2	5.4	**.040**	17	15.3
With FHL	22	29.7	7	18.9	.222	29	26.1
With FHL and others	26	35.1	22	59.5	**.015**	48	43.2
With others	11	14.9	6	16.2	.852	17	15.3

Abbreviation: FHL, flexor hallucis longus.
[a]*P* values when bone types are compared per subgroup. Values in bold represent statistical significance.

Discussion

This study described a high prevalence of associated pathologic structures involved with a trigonal disorder leading to PAIS in a large cohort. Trigonal bone (os trigonum or Stieda) was found to cause impingement in isolation in a small proportion of cases (15.3%). Even when FHL disorders were removed from the analysis, 58.6% of the total patients still presented other associated impingement conditions. More than 38.8% of our cohort presented at least 3 additional diagnoses to a trigonal disorder. In light of the presented results, our primary hypothesis, that the amount of isolated trigonal disorder in PAIS would be low, was confirmed.

The concept of potential alternative etiologies of PAIS besides an os trigonum or Stieda impingement is not new. Hamilton in 1984 described the FHL stenosing tenosynovitis in ballet dancers and its relation to a low-line muscle belly and os trigonum.[10] A direct relation among trigonal bone impingement and FHL disorders were later described by other authors when treating these conditions.[13,34] Descriptions of the intermalleolar ligament through magnetic resonance imaging (MRI) as a potential PAIS etiology was presented by Rosenberg et al[24] and Fiorella et al.[8] Paterson and Brown[19] presented 6 cases of posteromedial ankle impingement after ankle sprains that produced fibrotic scar tissue in the deep deltoid striking the medial malleolus. Peace et al[20] demonstrated several other MRI findings in patients with PAIS, emphasizing the low trigonal prevalence in their cohort (11 of 25). Other authors also demonstrated numerous other indications for PAHA indications and PAIS etiologies, but few explored their association with trigonal bone impingement.[17,18] In a sample of 78 patients treated with PAHA, Van Dijk reported 56 PAIS due to trigonal-related causes.[30] About 43% of his sample (24 of 56) was isolated os trigonum whereas the remaining (57%, 32/56) had an associated FHL tendinitis.[30] Smyth et al[26] found 20 (of 22) PAHA caused by trigonal bones, all of them with adjunctive FHL tenosynovitis (100%). In a sample of 24 os trigonum, Weiss et al[32] observed 21% happening in isolation, 58% in conjunction with FHL disorders, and 12% with talar osteochondral lesions. Zwiers et al[36] described a 37% FHL tendinopathy prevalence in bony impingement (34% in os; 40% in Stieda) treated with PAHA. In our study, reporting a large number of patients (n=112), a lower occurrence of isolated trigonal impingement was noticed (16.07%), whereas FHL concomitant disorders remained high (68.75%). Interestingly, a considerable rate of associated conditions (58.6%) was still depicted once the FHL was removed from our analysis. This prevalence is noteworthy compared with previous PAIS studies that usually report surgical findings as single or dual entities but may not reflect the broader number of concomitant etiologies. The present study highlights findings of numerous other impingement etiologies in addition to Stieda process and os trigonum impingement and might in part explain the relatively high rate of poor patient satisfaction reported in the recent meta-analysis by Zwiers et al.[37] This information should provide impetus to analyze current diagnosis and planning of surgery for treating PAIS whether with PAHA or by open approaches.

The current medical and scientific scenario expose a low correlation among imaging findings, clinical symptoms, and surgical observations, potentially making our findings more meaningful.[2,3,35] Zwiers et al[35] described os trigonum in 23.7% and Stieda in 34.9% of their asymptomatic bilateral patients undergoing computed tomographic assessment. A higher prevalence was found in patients with PAIS (odds ratio 1.86), which somehow explained why these bone structures are traditionally pointed as PAIS-only etiology.[35] In a case-control study, Baillie et al[2] compared MRI data in 82 athletes with and without PAIS, finding no association ($P > .005$) between positive imaging (edema, trigonal, synovitis, FHL disease) and symptoms (posterior ankle pain or positive plantarflexion test). No correlation ($P > .005$) among MRI and patient-reported outcomes (PROs) was noted.[2] A later systematic review in imaging for PAIS, performed by the same authors, pointed to the lack of control groups (with asymptomatic subjects) in the available studies, making an adequate clinical correlation impossible.[3] These authors support the clinical notion that PAIS diagnosis is very challenging and

etiologic characterization almost impossible.[2,3,35] We described a number of different conditions (n = 11; Table 1) coexistent with a os trigonum or a Stieda process that are likely to contribute to pain and impingement. Trigonal disorders were also found to carry a mode of 3 adjunctive procedures in our cohort, supporting the idea that many pathologic structures might be encountered when treating these conditions through PAHA.

Comparison among Stieda process and os trigonum is very scarce in the literature when considering any variable. Symeonidis et al[29] assessed PROs after bone removal in both groups (n = 26) and found favorable outcomes at 6 months, but no difference at the 12-month period. Zwiers et al[36] found similarity in PROs when comparing os trigonal and Stieda resection. Prevalence of both trigonal bones were described for os trigonum and Stieda, respectively, by Peace et al[20] (7 and 4), Vila et al[31] (31 and 4), Pereira et al[21] (5 and 3), Baillie et al[2] (28.0% and 31.7%), and Zwiers et al[35] (23.7% and 34.9%). Our cohort included 74 os (66.7%) and 37 Stieda (33.3%) ($P < .001$), a proportion in line with previous studies. Although groups and subgroups could be underpowered to demonstrate differences, the os trigonum had a higher prevalence of isolated bone impingement (20.3% vs 5.4%; $P = .040$) but a lower prevalence of association with FHL and others (35.1% vs 59.5%; $P = .015$). A rigid Stieda (when compared to a mobile ossicle) could have more capability in producing other surrounding lesions (Table 1), such as posteromedial synovitis (40.5% vs 17.6%; $P = .009$) and tibia osteophytes (13.5% vs 2.7%; $P = .027$).

The present study has important limitations that must be discussed. First, the study is not an outcomes study, so no conclusion can be made about the potential benefit of treating these multiple impingement lesions. Ideally, having at least short-medium term (2-5 years) patient-reported outcomes would help to validate our hypothesis but as the number of etiologies (variables) increases the ability to discern which specific etiologies that might otherwise be asymptomatic becomes extremely difficult. Additionally, this was a retrospective study of 3 surgeons' experience without controlled diagnostic criteria of physical examination, radiologic studies, ultrasonographic examination, and operative selection criteria. However, the ultimate diagnostic test is direct visualization, for which PAHA provides the most comprehensive and magnified images of the posterior ankle. The assumption that soft tissue displacement into the ankle joint is consistent with symptomatic impingement is central to much of what we do in arthroscopic treatments. It is, however, critical to recognize that not all impingement is necessarily symptomatic as painless crepitus is a common finding on physical examination. In this absence of knowing what might or might not be a cause for ongoing pain after treating a more obvious cause of impingement such as an os trigonum, the concept of treating those pathologies that are likely to be symptomatic postoperatively seems to be a reasonable concept in practice. Conversely, they were removed based on their intraoperative appearance and dynamic behavior with impingement maneuvers or manipulation. It is critical to recognize that we cannot treat what we cannot see.[16] Nevertheless, as previously stated, they could also be clinically dissociated.[2,37] Finally, no sample sizing or power analysis were performed.

Conclusion

In a large PAHA cohort, os trigonum or Stieda process presence in isolation was low. Most cases presented several associated pathologic findings that could be contributing to PAIS. Even when FHL conditions were not considered as associated pathologies, the concomitant disorders' presence was still high. These results, although still not completely clinically correlated, should alert surgeons when assessing PAIS and planning PAHA. Open approaches to PAIS can limit the field of view and do not allow for the magnification and lighting that is possible with an arthroscope. This may impact the ability to fully evaluate additional etiologies of PAIS.

Ethical Approval

University Ethics Committee approved this research under the number 201912144 in accordance with the Declaration of Helsinki. The study complies with the Health Insurance Portability and Accountability Act (HIPAA).

Data Sharing

According to the ICMJE data sharing police, core records will be shared through Mendeley Data and available on request.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article. Disclosure forms for all authors are available online.

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Appendix

Variable	Mean	SD	Range
Age	31.01	15.15	12-70
Body mass index	30.36	9.21	15.2-57.5

Labels	Count of Athlete/Nonathlete
Nonathlete	63
Dancer	10
Gymnast	7
Track	5
Football	4
Soccer	4
Basketball	4
Swimmer	2
Softball	2
Runner	2
Baseball	1
Volleyball	1
Multiple	1
Grand total	106