

REVIEW

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The Stieda process of the talus: the anatomical knowledge and clinical significance of an overlooked protrusion

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Abstract

Background: The Stieda process (SP) is an extended lateral tubercle of the posterior process of the talus. Although there are different classifications for SP in the literature, it is essential to know the differential diagnosis of SP from fractures and accessory ossicles. This review aims to classify the SP and to guide the formation, prevalence, etiopathology, symptoms, differential diagnosis, and treatment.

Main body of the abstract: The authors conducted a literature review that lasted up to September 2022 and used the databases PubMed, Web of Science, and Google Scholar to explore the SP of the talus in all of its aspects. Out of 3802 publications, only 34 could be included, and most of them were studies on posterior ankle impingement syndrome (PAIS). The occurrence, prevalence, etiopathology, clinical significance, symptoms, differential diagnosis, and treatment methods of SP were investigated based on the literature. SP is formed by the fusion of a secondary ossification center at the posterolateral side of the talus with an incidence of 12–36.5%. It is frequently observed in males. It causes reduced plantar flexion, impairment in the inversion of the foot, pain, and swelling in the posterior ankle joint, PAIS, and SP fractures after trauma.

Short conclusion: SP can be diagnosed by lateral ankle radiography or MRI in patients complaining of PAIS. The initial treatment of the SP should include reducing inflammation, swelling, pain, and limiting activities. If neither modality affects the patient, surgical treatment will be performed, and SP will be resected until the impingement disappears.

Keywords: Stieda process, Stieda fracture, Posterior ankle impingement, Talus, Lateral process, Posterior process

Background

The Stieda process (SP) is an elongated lateral tubercle of the posterior process of the talus or astragalus (Moore and Harger 2018). It was first identified by German anatomist and anthropologist Christian Hermann Ludwig Stieda (1837–1918), and he described this anatomical variant in 1869 in the “Ueber secundäre Fusswurzelknochen” (Stieda 1869). His anatomical research covered the comparisons of the central nervous system,

the construction of bone, and the development of the thyroid, carotid, thymus, and lungs (Stieda 1937). SP is more significant than the medial tubercle of the posterior process of the talus, and it is the most varied element of the hindfoot anatomy (Moore and Harger 2018). The posterior talofibular ligament (PTFL), which is the part of the lateral collateral ligament of the ankle, inserts into the lateral tubercle of the posterior process of the talus. The tendon of flexor hallucis longus (FHL) runs between the medial and lateral tubercles of the posterior process of the talus (Moore and Harger 2018). The acute or chronic fractures of SP are relatively unusual lesions, and they give rise to malunion and early degenerative changes in patients with SP (Nyska et al. 1998). SP fractures may

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occur due to tension forces created by fibers of the PTFL, tibial compression, or avulsion fractures (Russell et al. 2010). There are contributing mechanisms for non-avulsion fractures in the literature. These are a gradual detachment of the lateral tubercle caused by repetitive plantar flexion or traumatic sudden plantar flexion of the foot (Russell et al. 2010).

The profile of SP fractures is similar to that of an ankle sprain (Moore and Harger 2018), and it can be misdiagnosed as a sprain or mistaken for a standard variant and affects the management of the patient. The fractures of the entire posterior process of the talus are not known by the same name. The fracture of the medial tubercle is called Cedell fracture (CF), while the fracture of the lateral tubercle is called Shepherd fracture (SF). Therefore, fractures of SP and SF should be distinguished from *os trigonum* (OT) and simple ankle sprains since they involve the articular surfaces of the ankle and subtalar joints (Moore and Harger 2018; Prasad et al. 2007). Although it is classified as a type of OT in the literature, it is a different anatomical variant. There is a lack of studies on the development process of this accessory ossicle and differential diagnosis and management strategies. Therefore, this review aims to classify this anatomical variant and guide diagnosing and treatment-related disorders caused by SP.

Main text

Embryology

The ossification center of the talus is formed during the intrauterine 7–8th weeks. Medial and lateral tubercles of the talus or astragalus appear as a separate ossification center at 11–13 years in males and 8–10 years in females and unite with the body of the talus (Herring 2002). Talus has a retrograde arterial supply; therefore, particular

arterial distribution plays an essential function in developing impairments and complications after trauma. The talus is predisposed to avascular necrosis (AVN) or bone death due to ischemia, owing to its unique structure, extraosseous, and variable intraosseous arterial supply. SP is formed by the fusion of a secondary ossification center at the posterolateral side of the talus, where the fused segment remains more prolonged than the usual profile (Robinson and White 2002). It is formed by a secondary ossification nucleus that appears between 7 and 13 years and joins the talus in the following 12 months (Robinson and White 2002; Sanchez Prida et al. 2016). When the ossification nucleus is large, it forms a prominent lateral tubercle known as the SP (Sanchez Prida et al. 2016). SP is not a sesamoid or accessory bone and is not embedded within the tendon. In some individuals, the lateral tubercle does not unite with the body of the talus and it remains as a separate ossicle due to the unfused ossification center. This accessory bone is called OT with a prevalence of 7–14% in adults (Weinstein and Bonfiglio 1975). Moreover, the repetitive raising of the heels might lead to additional bony growth on the talus in the early years of life (Robinson and White 2002). While the SP is an extension of the posterior process (lateral tubercle part of the posterior process) of the talus, the OT is connected to the posterior process by fibrous, fibrocartilage, or cartilage bridges (DiGiovanni and Greisberg 2007). The difference between SP, OT, SF, absence of lateral tubercle, and normal talus is shown in Fig. 1.

Methods

The authors conducted a literature review that lasted up to September 2022 and used the databases PubMed, Web of Science, and Google Scholar as their sources to explore the SP of the talus in all of its aspects. The MeSH terms that were used were either “Stieda Process” (All

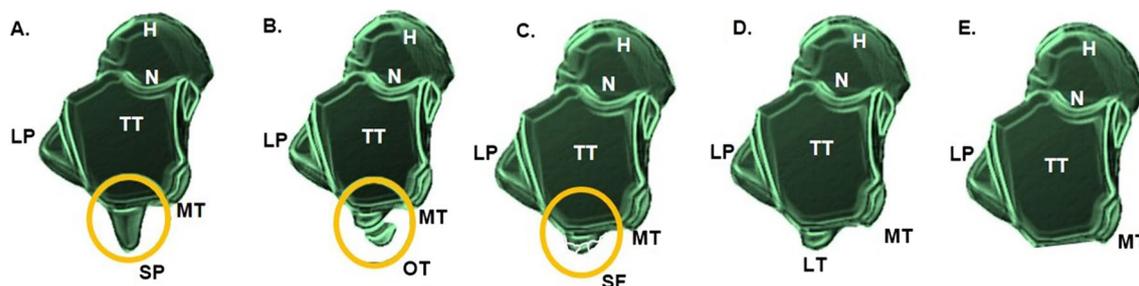


Fig. 1 The superior view of the talus (left side). **A** The Stieda Process (SP) (circle). It occurs as a result of the elongation of the lateral tubercle of the posterior process of the talus. **B** Os Trigonum (OT) (circle). OT occurs as a result of the separation of the lateral tubercle of the talus from the posterior process. OT is an accessory bone, unlike the SP. **C** The Shepherd fracture (SF) (circle) is a fracture resulting from the fracture of the lateral tubercle. **D** The typical talus with a normal lateral tubercle. **E** The figure shows the talus with a missing lateral tubercle (agenesis of lateral tubercle). *H* Head of the talus, *LT* Lateral tubercle, *MT* Medial tubercle, *N* Neck of the talus, *OT* Os Trigonum, *SF* Shepherd fracture, *SP* Stieda process, *TT* Trochlea of the talus

Fields) OR “Stieda’s Process” (All Fields). There were two different search criteria utilized according to the previous methods of literature review (Ogut 2022, Ogut and Armagan 2022; Ogut et al. 2022a, 2022b). The first criterion was exclusion criteria, articles that were eliminated from the list because they did not relate to the topic but included the words “stieda process,” or “stieda’s process” were evaluated according to the first set of criteria. The second criterion was eligibility criteria, articles that were relevant to the topic and included the terms “stieda process,” or “stieda’s process” but instead be editorials, letters to the editor, or commentaries were eliminated from the list. Only research that demonstrated the “Stieda process” or “Stieda’s process” was considered for inclusion in the study. These studies might take the form of original articles, case reports, books, book sections, or systematic reviews with or without meta-analysis. All studies in the literature that describes a “Stieda process” or “Stieda’s process” were included in the current study regardless of language or format. This review did not include articles that looked at the os trigonum, os trigonum syndrome, posterior ankle impingement syndrome (PAIS), variations of the talus, or the foot without the Stieda process.

Results

It was determined that a total of 3802 items existed. Following the elimination of the redundant articles, the total number of unique entries that remained was 1592. However, according to the second search criteria, it was found that 37 publications were pertinent to our topic. There were a total of 24 publications that were included in the research that discussed the “Stieda process” of the talus or “Stieda’s process.” The number of articles that were written on the “Stieda process” or “Stieda’s process” but were not included in the research was 66. This review article consists of a total of 34 articles, excluding any letters to the editor, comments, or proceedings that may have been included. Previous investigations into the patient profile, diagnosis, symptoms, therapy, and clinical importance of SP are outlined in Table 1, which provides a summary of those studies.

Prevalence

Little knowledge about the incidence of SP is available in the literature (Kalbouneh et al. 2021). It might result from the SP that has not part of a standard definition or classification system (Kalbouneh et al. 2021). Studies on SP (mostly case studies) have examined its symptoms, clinical significance, and treatment plan (Table 1). There is no agreement on the prevalence of SP in the literature, as it varies widely from 12 to 36%. Kalbouneh et al. reported that SP was found in 26.1%, OT in 20.5%, typical lateral tubercle in 46.1%, and absent tubercle in

7.3% (Kalbouneh et al. 2021). The literature shows that PAIS caused by SP is frequently seen in dancers and athletes who have to perform forced plantar flexions of the foot, such as soccer, basketball, and volleyball players, and especially among ballet dancers, but this syndrome can occur in many other professions or sports activities (Sanchez Prida et al. 2016). The injury to the lateral tubercle is usually caused by excessive inversion or plantar flexion of the foot; however, the injury to the medial tubercle is mainly caused by the forced dorsiflexion and pronation of the foot. The frequency of SP was evaluated by dividing the posterior talar process into four variants based on the classification of Sarrafian & Kelikian (2011) (Sarrafian and Kelikian 2011). The following types were classified in the literature: type 1 (absent tubercle), type 2 (typical tubercle), type 3 (SP), and type 4 (OT) (Kalbouneh et al. 2021). Fu et al. stated that type 1 was a single bone that was not connected to the talus, type 2 was connected to the posterior process of the talus by hyaline cartilage, and type 3 was the SP (Fu et al. 2019). Although there are different classifications in the literature, SP and OT are entirely different from each other, and their differentiation is necessary to treat caused disorders properly.

Furthermore, a higher prevalence of SP was found in males, while OT was higher in females (Kalbouneh et al. 2021). In the study of Zwier et al., the frequency of SP was found in about one-third of the patients without OT, with an incidence of 35.7% in the Netherlands (Zwiers et al. 2018). In a CT-based study of a Chinese population, SP was identified in 14.7% of patients (Fu et al. 2019). In a study, the prevalence of SP was 16.7–36.5% (20.3% in males and 12.7% in females) (Derin Çiçek and Bankoğlu 2020). Furthermore, they stated that 26% of the Turkish population was predisposed to PAIS (Derin Çiçek and Bankoğlu 2020). Significant disparities between the sexes were detected, but the prevalence of SP in the right and left feet was 17 and 16.4%, respectively, which was not statistically different (Derin Çiçek and Bankoğlu 2020). According to the literature, it can be said that the incidence of SP is between 12 and 36.5%, and it is frequently observed in males.

Etiopathology

The profound knowledge about the pathology of SP is unclear. However, it has been reported that the most frequent causes of bony impingements were OT (47%), Shepherd’s Fracture (2%), SP (24%), loose bodies or avulsion fragments (3%), accessory ossicles (1%), and enlarged calcaneal tuberosity (Ribbans et al. 2014). This extra bone growth, combined with repetitive plantar flexion, can build inflammation and result in PAIS. Several studies have reported that SP might also cause FHL pathology

Table 1 Previous studies on the patient profile, diagnosis, symptoms, treatment, and clinical significance of SP

| Author | Year | Place | Number/age/sex | Symptoms | Study design | Pathology | Treatment | Significant outcomes |
|-----------------------|------|-------------------|-------------------------------------|--|--|--|--|--|
| Yilmaz et al | 2006 | Mersin/ Turkey | 1/19/M | -Posterior ankle pain after trauma -PAIS of SP -Tenderness (posteromedial ankle) | AP Lateral, Mortise Stress Radiographs | Long SP Long calcaneal process | -Conservative (shortleg cast for 6 weeks) (failed) -Arthroscopic excision | -SP is a rare cause of PAIS -Refractory to conservative treatment, arthroscopic excision allows for easy and more satisfactory excision than open excision |
| Perdikakis et al | 2010 | Greece | 1/27/ M | -Progressive posterior ankle pain -Mild swelling and tenderness (posterolateral ankle) -Restriction during plantar and dorsal flexion -Haglund's deformity in calcaneus | Plain X-ray MRI | -Marrow edema and small effusion of tibiotalar joint -Soft-tissue edematous and fibrotic changes in periarticular fat | -Conservative -physiotherapy -arthroscopic -open debridement | Conventional radiographs and CT may show SP, but MRI is required to confirm if these findings are the source of symptoms |
| Sánchez Prida et al | 2016 | Spain | 1/40/ M | -Right ankle pain without trauma -PAIS-related symptoms -Radiating pain -Stiffness -Pain with plantar flexion and palpation | Plain X-ray (Lateral view) | No signs of inflammation, edema or skin changes, or the presence of asymmetries or deformities in ankles | -NSAID, analgesics, joint rest, immobilization -Physiotherapy -Manual mobilization -Kinesio tape -Ultrasound -Transcutaneous electrical nerve stimulation (TENS) -Dry needling -Percutaneous treatment with corticosteroids -Arthroscopy, open surgery | -When the pain appears in the posterior ankle, especially plantar flexion, and there is no previous history of trauma, we must be aware of PAIS caused by SP -Plantar hyperflexion produces the "nutcracker phenomenon," in which the inferior-posterior tibial platform and the posterosuperior part of the calcaneus form a pin-ner that compresses the SP against the surrounding soft tissues |
| Cuéllar-Avaroma et al | 2017 | Mexico | 24/31.8 ± 5.26 years/ M:19, FM:5 | -Chronic pain PAIS-related symptoms -complex regional pain syndrome | X-ray | Wound erythema | -Conservative -posterior ankle endoscopy -physical therapy -immobilization -arthroscopic treatment | The arthroscopic treatment is an ideal option as it presents an excellent postoperative recovery with a swift return to patients' preoperative sports activities |

Table 1 (continued)

| Author | Year | Place | Number/age/sex | Symptoms | Study design | Pathology | Treatment | Significant outcomes |
|---------------|------|----------------|---|--|--|---|---|--|
| Moore et al | 2018 | Oregon/ USA | 1/39/F | -Pain (Posterior ankle) -Swelling after trauma (left ankle) -Impairment in the plantar flexion | Radiographs | Long SP | -Conservative (shortleg cast for 6 weeks) -Serial follow-up radiographs at 2, 4, and 8 weeks | -SP fracture could be misdiagnosed as an accessory ossicle, OT, because of its similar appearance and location -It is essential to recognize and identify the fracture early to ensure proper management |
| Loureço et al | 2018 | Portugal | 1/35/M | -Posterior ankle pain without trauma (right) -Pain anterior to the Achilles tendon | Radiography CT/MRI STIR | -Inflammatory changes with SP -Inflammatory process of soft tissues near SP -Joint effusion -Palpable soft-tissue thickening | -Conservative treatment (local corticosteroid injection and 4–6 weeks of immobilization) -Arthroscopic excision | -SP is a rare cause of PAIS, and radiologists must be aware of this differential diagnosis -Arthroscopic excision can be used in refractory cases, and complete recovery is attained in a shorter time than open excision |
| Martins et al | 2018 | Portugal | 1/38/FM | -Posterior ankle pain without trauma (right) -Pain with forced flexion of the right feet -Swelling (posterior ankle) -PAI syndrome of SP | Lateral Radiography Sagittal STIR/MRI | -Enlarged SP -Bone marrow edema -Slight effusion -Edema of the subcutaneous fat | -NSAID -Cortisone injections -Rehabilitation, physiotherapy (soft-tissue mobilization) -Tape or brace the ankle in dorsiflexion -activity restriction (avoidance of forced plantar flexion) | SP is an under-recognized cause of PAI, but MRI can easily make the diagnosis and guide appropriate treatment |
| Cicek et al | 2020 | Istanbul | 1088/35.86 ± 15.79 (15–95) / M: 576 (52.9%) FM: 512 (47.1%) | -PAIS-related symptoms -Excessive repetitive plantar flexion (ballet dancers, gymnasts, basketball, soccer, and football players) or traumatic events | Lateral ankle radiographs | Prominent posterolateral talar process (7.3 mm) | -Conservative -arthroscopic -open debridement | -¼ of the Turkish population had SP or OT, which made them susceptible to PAIS. The prevalence of SP was higher than that of OT, and both were more frequent in males than in females -Therefore, clinicians should provide early diagnosing and treatment SP for ankle health management |

Table 1 (continued)

| Author | Year | Place | Number/age/sex | Symptoms | Study design | Pathology | Treatment | Significant outcomes |
|------------------|------|----------------|---|--|---------------------------|--|--|---|
| Micheli et al | 2021 | Boston/ USA | 54/22.7 ± 6.1 years/ FM:53, M:1 | -PAIS-related symptoms -Anterior ankle impingement -Recurrent tenosynovitis -complex regional pain syndrome | Lateral Radiography CT | -Abnormalities of the SP -painful OT of the talus | -The posterolateral surgical approach -Posterior ankle decompression with physical therapy -Corticosteroid injections under ultrasound medication -physical therapy -IV antibiotic treatment | The resection of OT and SP via the posterolateral approach is a safe and effective solution |
| Symeonidis et al | 2021 | Greece | 30/31.46 ± 10.70 (17–48) years M:10, FM: 3 | PAIS-related symptoms | MRI and CT | -SP -OT -Lateral ankle instability -Haglund deformity -Osteochondral lesion of the talus | -Conservative (physiotherapy, modification of activities single ultrasound-guided beta-methasone injections) -Posterior arthroscopy -SP resection | -Better FFI outcomes were observed in the resection of the OT group at a 6-month follow-up -Significant AOFAS and FFI improvement were observed from 6-month to 12-month follow-up |

AVN Avascular necrosis, AP Anteroposterior, AOFAS The American Orthopedic Foot and Ankle Score, CT Computed tomography, F Female, FFI Foot function index, FHL Flexor hallucis longus, IV Intravenous, M Male, MRI Magnetic resonance imaging, NSAID Nonsteroidal anti-inflammatory drugs, OT Os Trigonum, PAI Posterior ankle impingement, PAIS Posterior ankle impingement syndrome, PTFL Posterior talofibular ligament, SP Stieda process, STIR Short tau inversion recovery, TENS Transcutaneous electrical nerve stimulation

due to the compression of the SP against the surrounding soft tissue. Other causes of FHL pathology apart from SP were tenosynovitis, stenosis (26%), partial tears (2%), nodules (1%), adhesions (1%), ossicle of FHL, tendinosis (1%), hypertrophied muscle, distal muscle insertion (1%), ganglion/cysts of FHL (1%), accessory muscles (1%), posteromedial impingements and other soft-tissue impingements (1%), and accessory ossicles (Ribbans et al. 2014). The posteroinferior part of the tibia and the posterosuperior part of the calcaneus compress the SP against the surrounding soft tissue by repeated plantar hyperflexion, generating inflammation and pain (Sanchez Prida et al. 2016). On the basis of the data provided, it is possible to say that recurrent plantar flexion produces compression of the SP, and as a result, PAIS-related inflammation and discomfort develop in this region.

Clinical perspective and symptoms

SP plays a role in the weight bearing and plantar flexion of the foot (Moore and Harger 2018). SP fractures are frequently caused by excessive inversion or plantar flexion (Judd and Kim 2002; Moore and Harger 2018; Paulos et al. 1983), which can occur in athletes when running or during plantar flexion of the foot (Berkowitz and Kim 2005; Bureau et al. 2000). It can form a posterior block to plantar flexion when the distal end of the tibia rests against it in forced plantar flexion (Russell et al. 2010). The repetitive plantarflexion/dorsiflexion may disrupt the synchondrosis type of joint between the SP and tibia or cause compression of the SP against the tibia (Kalbouneh et al. 2021). Forced plantarflexion increased pain (Yilmaz and Eskandari 2006). Alterations in various ankle structures can produce the same type of pain, so the differential diagnosis of the ankle anatomy and its functional mechanics have a crucial role (Sanchez Prida et al. 2016). When the pain appears in the posterior ankle, associated with movements of the foot, especially plantar flexion, and there is no previous history of trauma, clinicians should be aware of PAIS and the causes of this syndrome, such as the SP (Sanchez Prida et al. 2016). Forced ankle dorsiflexion may avulse the SP through the PTFL. The SP of the talus, joint capsule, adjacent ligaments, and tendons might have impinged between the distal end of the tibia, posterior malleolus, and calcaneal tuberosity (Yilmaz and Eskandari 2006).

The SP is often asymptomatic, but it might be impinged between the calcaneus and tibia in forced plantar flexion, causing microtrauma to the adjacent bony structures and compressing the soft tissues posterior to the ankle (Hess 2011), and leading to posterior ankle pain (Yilmaz and Eskandari 2006). Acute impact injury may cause SP fracture and PAIS, which seems like the syndrome of OT (Hess 2011). Patients usually report chronic or recurrent

posterior ankle pain increased by forced plantar flexion (Martins et al. 2018). Other causes of PAI may result from FHL tenosynovitis, ankle osteochondritis, and subtalar joint disease (Martins et al. 2018). PAI typically induces posterior ankle pain exacerbated by plantar flexion or dorsiflexion, posterior tenderness anterior to the Achilles tendon, and a palpable soft-tissue thickening (Robinson and White 2002). Physical examination of a patient with a fracture of SP will reveal pain with plantar flexion and weight bearing on the foot that may be aggravated with flexion of the hallux (Moore and Harger 2018). Patients will frequently present with swelling (especially medial to or at both sides of the Achilles tendon) and PAI at the posterolateral side of the ankle (Judd and Kim 2002; Paulos et al. 1983). It can also cause significant disability because of the risk of malunion and degenerative arthritis (Prasad et al. 2007). The presence of SP can narrow the space between the posterior aspect of the talus and the calcaneus, which has led to increased compression and injury to the bony structures, joint capsule, ligaments, and tendons (Berman et al. 2017). Corresponding protrusions of calcaneal processes have also been reported in patients with SP (Yilmaz and Eskandari 2006). In the same manner, OT leads to bony impingement between the posterior edge of the distal tibia and the calcaneal tuberosity (Yilmaz and Eskandari 2006). However, it has been reported that SP causes bony impingement after an injury such as acute or chronic ankle trauma after forced plantar/dorsal flexion (Bureau et al. 2000). Impingement may be elicited with an acute plantarflexion trauma and leads to contusion, compression, or fracture of the SP (Yilmaz and Eskandari 2006). Therefore, SP fractures of the talus can be a source of PAIS, dysfunction, swelling, tenderness at the posteromedial side of the ankle, and mechanical irritation of the heel (Dancer's heel pain) (Yilmaz and Eskandari 2006). PAIS, due to the presence of SP or OT, causes tenosynovitis of the flexor of the first digit, plantar fasciitis, tarsal tunnel syndrome, deltoid ligament injury, and posterior tibial tenosynovitis (Sanchez Prida et al. 2016). It can be said that SP usually causes PAIS with swelling and tenderness, after an injury or trauma during the forced plantar flexion or massive inversion of the foot and leads to contusion, compression, or fracture of the SP.

Differential diagnosis

Differential diagnoses of separate accessory bones, abnormal bone formations, and anatomical variations are essential for treating several disorders (Ecker et al. 1967; Ogut 2022, Ogut and Armagan 2022; Ogut et al. 2022b, 2020; Weinstein and Bonfiglio 1975; Zwiers et al. 2018). SP is not to be confused with the Pellegrini–Stieda lesion. SP refers to an elongated lateral tubercle of the posterior

process of the talus; however, Pellegrini–Stieda lesions are ossified post-traumatic lesions at the medial femoral collateral ligament. The posterior process of the talus is of particular significance in PAI because that leads to both the OT and SP (Ecker et al. 1967). Because of its similar appearance, locations, and symptoms, fractures of SP are misdiagnosed as OT (Kou and Fortin 2009).

Similarly, Fu et al. noted the similarity between the SP and the OT in origin, location, and clinical diseases (Fu et al. 2019). A similar location of the pain is also caused by a tear of the posterior syndesmotic ligament, Achilles tendinopathy, and posterior osteochondral lesion of the talus without impingement symptoms. Serial radiographs and other imaging modalities can distinguish SP from OT, which can be required for accurate diagnosis. Moreover, lateral radiograms should be carefully examined to assess the presence of SP or OT (Berkowitz and Kim 2005). Therefore, understanding various impingement types will provide clinicians prevent, identify, treat, and rehabilitate the affected ankles (Hess 2011). However, several studies stated that SP was classified as a type of OT (Fu et al. 2019; Kalbouneh et al. 2021; Zwiwers et al. 2018). OT has marrow signal alteration with T2-hyperintense and T1-hypointense signals consistent with marrow contusion. Posterolateral capsular thickening and fluid-signal compatibility with synovitis are part of the differential diagnosis of OT. However, SP was characterized by the length of the lateral tubercle exceeding the length of the medial tubercle by more than 5 mm in the lateral view (Derin Çiçek and Bankaoglu 2020). It has a sharp marginated non-corticated irregular outline as opposed to OT, a round/oval accessory ossicle with well-corticated margins, and smooth radiolucent lines between the OT and posterior process.

Imaging modalities such as the anteroposterior, lateral ankle, modified X-ray radiographs, sagittal CT, and MRI can be used to diagnose patients with SP (Yilmaz and Eskandari 2006). CT and MRI arthrography techniques allow the most accurate evaluation of the capsular recesses (Robinson and White 2002). Moreover, local anesthetic injection under fluoroscopy is recommended for diagnostic tests (Yilmaz and Eskandari 2006). MRI is required to confirm if the PAIS caused by SP (Perdikakis and Karantanas 2011). MRI detects the bony and soft-tissue abnormalities related to the PAIS (Bureau et al. 2000), and it is helpful in the assessment of abnormal signal intensity in the SP (Bureau et al. 2000), such as high signal intensity on fat-suppressed T2-weighted images/STIR sequences and low signal intensity on T1-weighted images (Cerezal et al. 2003; Robinson and White 2002). The signal abnormalities concern bone marrow edema, contusions, or hidden fractures resulting from repeated skeletal trauma (Cerezal et al. 2003; Robinson and White

2002). Increased signal intensity was also seen in inflammatory abnormalities in the soft tissues of the posterior ankle, the enlargement of the posterior synovial recess of the tibiotalar joint with the growth of the posterior cavity of the subtalar joint (Bureau et al. 2000), and the sheath of FHL or other internal deformities which can modify surgical attitude (Cerezal et al. 2003; Robinson and White 2002). Bone contusions of the SP are frequently seen in MRI of PAIS (Bureau et al. 2000). It has been reported that signs of instability at the ankle joint and tarsal tunnel syndrome were not seen in most patients with SP (Yilmaz and Eskandari 2006). Bone marrow edema and posterior ankle synovitis also suggest diagnosing PAIS (Cerezal et al. 2003; Robinson and White 2002). The most commonly used methods to diagnose SP include lateral radiography and MRI. Capsular recess, the signal abnormalities like bone marrow edema, contusions, fractures, or soft-tissue abnormalities related to the PAIS can be detected more easily on MRI.

Management

There are currently no formal guidelines with specific recommendations for the treatment of SP, due to the paucity of data in the literature. Several studies reported that the management of accessory ossicles could be due to the clinical condition being rare and the clinical course of accessory variants is not well understood (Ogut 2022; Ogut et al. 2022b; Ögüt et al. 2020). It is essential to recognize and identify the SP and related fractures to ensure proper management and a favorable outcome by preventing complications (Moore and Harger 2018). Moreover, adequate control requires extensive knowledge of hind-foot anatomy (Berkowitz and Kim 2005). Symptoms improve with nonsurgical management, but surgery may be necessary in refractory cases (Martins et al. 2018). Nonsurgical management with cast immobilization is frequently successful when the fracture is correctly diagnosed, and large fragments may be amenable to open reduction and internal fixation (Berkowitz and Kim 2005; Prasad et al. 2007). The patient with SP should be closely monitored with serial lateral radiographs to see the recovery. If there is inadequate resolution on the lateral view, a 30° subtalar oblique view may help differentiate between OT and SP (DiGiovanni and Greisberg 2007). Moore et al. reported that the patient was closely monitored with serial follow-up radiographs at sequential weeks to observe healing. The patient reported painless weight bearing and plantar flexion after 8 weeks (Moore and Harger 2018).

Similarly, in a study, it has been reported that 8 months after open surgical excision, patients could return to sports activities in 8 weeks (Yilmaz and Eskandari 2006). It may be followed by 1–2 weeks in a boot and 4–6 weeks

of physiotherapy. Symeonidis et al. reported that arthroscopic treatment of PAIS provided a significant American Orthopaedic Foot and Ankle Society (AOFAS) hindfoot score and foot function index (FFI) score at a 6-month follow-up (Symeonidis et al. 2021). The same improvement was observed from 6 to 12 months follow-up (Symeonidis et al. 2021), and no significant differences were reported between the groups at the 12-month follow-up (Symeonidis et al. 2021). Compared to the conservative and nonsurgical methods, complete recovery is achieved in a shorter time than the open excision.

A literature review on conservative treatment, like the management of PAI syndrome, includes rest, restriction of activity such as avoidance of forced plantar flexion, technique modification, physiotherapy, orthotics/footwear modification, and NSAID (nonsteroidal anti-inflammatory drugs), except for drug allergies and injections (Ribbans et al. 2014). It has been reported that NSAIDs were prescribed to reduce pain and inflammation (Martins et al. 2018). Conservative treatment of PAI consists of local corticosteroid injection and 4–6 weeks of immobilization (Mouhsine et al. 2004). However, it has been reported that conservative management, early mobilization, and cast immobilization did not change the results (Nyska et al. 1998). A few studies reported that 50% of the patients recovered with conventional treatment methods (DiGiovanni and Greisberg 2007). Physiotherapy, manual mobilization, the application of the Kinesio tape, ultrasound, and transcutaneous electrical nerve stimulation (TENS) shorten the duration of the condition (Sanchez Prida et al. 2016). Preventive taping and dry needling can also be applied in cases where the clinic is prolonged in recurrent cases to decrease its duration and increase the interval between recurrences (Sanchez Prida et al. 2016). If there is no clinical improvement, percutaneous treatment with corticosteroids can be performed, generally with good results (Sanchez Prida et al. 2016). In those cases that are refractory to treatment or have risky professions or activities and present symptoms, repeated procedures, arthroscopy, or open surgery allow resection of these lesions and resolution of the condition (Sanchez Prida et al. 2016).

If neither modality affected the patient, surgical treatment would be performed (Mouhsine et al. 2004), and SP would be resected with a burr until the impingement disappeared (Yilmaz and Eskandari 2006). Refractory to conservative treatment, arthroscopic excision (with standard posterolateral and trans-Achilles portals) under direct and dynamic visualization of the pathology, and open surgical excision are among the preferred treatment methods in the literature (Yilmaz and Eskandari 2006). The literature has stated that the preferred surgical technique for posterior ankle decompression and SP

resection remains controversial; however, the open posterolateral approach resulted in high expectations of a return to daily life with minimal complications (Micheli et al. 2021). It has been reported that endoscopic management is associated with low morbidity and short recovery time and provides better outcomes at 2–5 years of follow-up in most patients (Niek van Dijk 2006). The arthroscopic treatment is the preferred technique for SP as it presents an excellent postoperative recovery with a swift return to the patient's preoperative sports activities (Cuéllar-Avaroma et al. 2017).

Misdiagnosis or poor management of SP fractures includes chronic pain, late arthrosis, painful limitation of ankle movements, and impingement syndromes (Berkowitz and Kim 2005; Nyska et al. 1998; Robinson and White 2002). Moreover, untreated, chronic injuries can cause significant pain and functional impairment that may be improved with late surgical intervention (Berkowitz and Kim 2005). If SP is not recognized and appropriately managed, it may result in complications and long-term morbidity.

Limitations

In the literature, more emphasis was placed on OT and its types, but there was not enough information in the literature about the types of SP. Therefore, it needs to be studied and investigated separately from OT. Moreover, there is a need for 2D/3D studies on large-scale study groups with SP.

Conclusions

This review highlighted the potential etiopathology, prevalence, clinical significance, differential diagnosis, and management strategies of the SP. These unusual processes cause reduced plantar flexion, impairment in the inversion of the foot, pain and swelling in the posterior ankle joint, PAIS, and SP fractures after trauma. Radiologists must be aware of this anatomical variant because it is an under-recognized cause of posterior ankle pain. SP can be diagnosed by lateral ankle radiography or MRI in patients complaining of swelling, tenderness, and increased pain in the posterior ankle by forced inversion or plantar flexion after trauma.

Abbreviations

AVN: Avascular necrosis; AP: Anteroposterior; AOFAS: The American orthopedic foot and ankle score; CT: Computed tomography; FFI: Foot function index; FHL: Flexor hallucis longus; IV: Intravenous; MRI: Magnetic resonance imaging; NSAID: Nonsteroidal anti-inflammatory drugs; OT: Os trigonum; PAI: Posterior ankle impingement; PAIS: Posterior ankle impingement syndrome; PTFL: Posterior talofibular ligament; SP: Stieda process; STIR: Short tau inversion recovery; TENS: Transcutaneous electrical nerve stimulation.

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Author contributions

EO contributed to the conception and design of the study. EO is involved in data collection and acquisition of data, contributing to data management and interpretation of data. EO participated in drafting the article and revising it critically for valuable intellectual content and the writing of the manuscript. All authors have read the final approval of the version to be submitted.

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