The Cause of Pain in Symptomatic Accessory Navicular Syndrome: comparison between Athletes and Non-Athletes

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My disclosure is in the Final AOFAS Program Book.

I have no potential conflicts with this presentation.
Many theories have been put forth to explain the role of an accessory navicular as a pain generator in the foot. But there were few literature that reported the radiologic Cause of pain, especially, focusing on athletic population.

The Purpose of this study

To Evaluate the Radiologic Cause of Pain in the Athletes with Accessory Navicular Syndrome who underwent Surgery for continuing pain after 3 months of conservative treatment on comparison with that in the non-Athletes.
## Material & Methods (I)

### Demographic

- From Aug. 2012 to Aug. 2013
- 64 feet of 60 patients / Men : Women 33 : 27 divide into two groups as below
- Underwent Modified Kinder Procedure after minimum 3 month-conservative treatment

<table>
<thead>
<tr>
<th>Athletes group</th>
<th>Non-Athletes group</th>
</tr>
</thead>
<tbody>
<tr>
<td>31 feet of 27 patients</td>
<td>33 feet of 33 patients</td>
</tr>
<tr>
<td>Mean age at surgery</td>
<td>Mean age at surgery</td>
</tr>
<tr>
<td>16.06 ± 3.52</td>
<td>4.33 ± 11.04 y/o</td>
</tr>
<tr>
<td>Sports activities</td>
<td></td>
</tr>
<tr>
<td>16 soccer, 3 baseball</td>
<td></td>
</tr>
<tr>
<td>4 ballets, 4 others</td>
<td></td>
</tr>
</tbody>
</table>

### Clinical & Radiographic Evaluation

- Trauma History
  - Simple X-ray classify according to Sella classification
  - Talo-1st metatarsal angle on AP & Lateral view
Material & Methods (II)

**Ultrasonographic Evaluation**
- Check Synchondrosis / Posterior tibial tendon
  - heterogenous echotexture of synchonrosis
  - complete/incomplete separation
  - diastasis, fluid collection
  - tendon signal change: hypoechogenic

**MRI Evaluation**
- Bone marrow (BM) edema
- Tendon: signal intensity change
Results (I)

Simple X-ray

- Ankle sprain / classification
  - ankle sprain 36/64 (56.3%) / type II in All

- Size of Accessory navicular bone
  - Length & width measured

<table>
<thead>
<tr>
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<th>Non-Athlete group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Length</td>
<td>11.17±4.95 mm</td>
<td>13.79±5.14 mm</td>
</tr>
<tr>
<td>Width</td>
<td>7.22±2.37 mm</td>
<td>8.41±2.66 mm</td>
</tr>
</tbody>
</table>

- Concurrent flatfoot deformity
  - Total 20 feet of 64 feet
  - 8 of 31 feet in Athlete group
  - 12 of 33 feet in Non-Athlete group
All the patients were checked by ultrasound

Diastasis between Navicular and Accessory navicular

Pathologic posterior tibial tendon

3 of 31 feet in Athlete group
4 of 33 feet in Non-Athlete group
Results (III)

**MRI**

- Fat-suppressed T2 weighted sequences checked in 57
  bone marrow edema 47 of 57 (82.5%)

  A. navicular bone +/AN bone + : 43
  B. navicular bone - /AN bone + : 4
  C. navicular bone +/AN bone - : 0
  D. navicular bone - /AN bone - : 10

  Tendon signal change: only one in 57 feet

In Athletes

- MRI checked in 30 feet
- Bone edema 27 of 30 feet (navicular & accessory: all of 27 feet)

In Non-Athletes

- MRI checked in 27 feet
- Bone edema 20 of 27 feet (navicular & accessory: 16 of 20 feet, only accessory: 4 of 20 feet)

- No significant difference in the presence of bone edema
  \( P = 0.167 \), Fisher’s exact test
- Significant difference in BM edema in both AN & Navicular
  \( P = 0.012 \), Fisher’s exact test
Discussion(I)

✓ Many Theories to explain the role of an accessory navicular as a pain generator

“Microscopic chronic inflammation”

Geist et al. JBJS Am 1925;7(3):570-574

“The navicular showed an incompletely fused physeal structure with resultant synchondrosis”

Zadek et al. JBJS Am 1948;30(4):957-968

“Symptoms were caused by sprain & contusion in the foot in 7 of 29 athletes”

Nakayama et al. AJSM 2005;33(4):531-535

✓ In this study, All of the patients had type II AN, and there was history of trauma in 23 feet (74%) of the athlete group. The remaining 8 feet were also very active young athletes. This study and previous reports strengthen the theory that clinical pain is due to movement at or repetitive trauma to unintended articulation, which resulted in a stress fracture.
Synchondral disruption and movement between the two bones could be a source of pain.

In this study, synchondral disruption was noted in all of the feet on USG. In addition, 18 feet (58%) in the athlete group and 11 feet (32%) in the non-athlete group showed a movement between the two bones. (p=0.047, Pearson’s chi square test).

Bone marrow edema of the navicular bone was more common in the Athlete group.

In this study, 47 of 57 feet (82%) showed BM edema including 4 feet showing only AN bone BM edema. There was no significant difference in the presence of BM edema in any of the two bones. But there was a significant difference in BM edema in the navicular and accessory navicular bone between the two group. (p=0.012, Fisher’s exact test)
Conclusion

All of the patients with symptomatic ANS were classified as type II. All of the patients had synchondral disruption on ultrasound examination. Bone marrow edema was also seen in the navicular and accessory navicular bone in most of the patients. However, posterior tibial tendon pathology was not highly prevalent as the symptoms.

Therefore, clinical pain was due to movement at or repetitive trauma to the accessory navicular and navicular and their articulation, which resulted in a 'stress fracture'.

There were significant differences in age, history of trauma, movement between the two bones, and bone marrow edema of the navicular bone between athletes and non-athletes. However, there were no differences in the flatfoot deformity and size of the accessory navicular bone.


