Talar Avascular Necrosis following Subchondroplasty in the Setting of Antiphospholipid Syndrome

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Statement of Purpose

This case presentation highlights the first report of talar avascular necrosis (AVN) in the setting of Antiphospholipid Syndrome (APS) following attempted repair of an osteochondral defect by subchondroplasty.

Literature Review

Antiphospholipid syndrome (APS) represents a significant challenge in the setting of orthopedic surgery, even when medically anti-coagulated. The auto-antibodies generated in APS have pro- and anti-coagulant effects, however, pro-coagulant properties typically dominate leading to thrombosis. Prior reports of APS complications noted that 13% of cases are initiated by surgery.

Illustrated in **Figure 1** (left image) are several previously identified areas where APS is a concern. Mechanistically, the pro-coagulative nature of the disease process is highlighted (right image), characteristically falling into five categories, which include:

1. Platelet activation
2. eNOS inhibition causing endothelial dysfunction
3. Neutrophil activation and adhesion
4. Accelerate atherosclerosis
5. Sensitivity to inflammatory triggers (i.e. infection, **surgery**)
6. Intimal hyperplasia vasculopathy

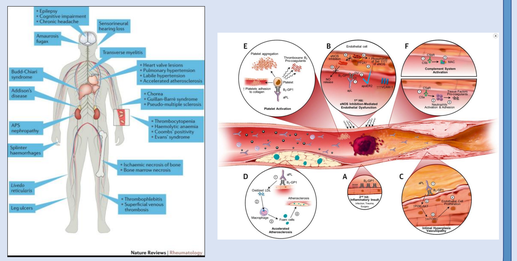

Figure 1: *(Left) Systems and disease processes associated with APS, (Right) Mechanisms driving APS pathology.*

Case Study

Pre-operative Evaluation

33-year-old male patient with known history of APS initially presented with a twisted ankle and continued pain. Following failed conservative care which consisted of 6 weeks non-weight bearing, patient obtained MRI (see **Figure 2**). Radiological report identifies a non-contained, lateral talar shoulder osteochondral defect with underlying medullary edema.

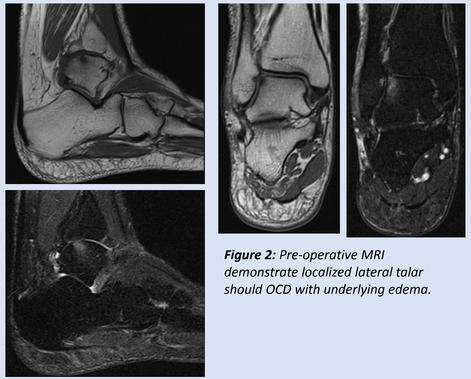

Figure 2: *Pre-operative MRI demonstrate localized lateral talar should OCD with underlying edema.*

Pre-operative blood testing revealed elevated but still within-normal-range lab values for β-2 glycoprotein, anticardiolipin and low levels of hexagonal phase phospholipid and lupus anticoagulant. Because the patient was known to have APS, the patient's long-term warfarin medication (used to maintain INR between 3.0 and 3.5) was bridged with enoxaparin during the operative period.

Per manufacturer protocol, patient underwent subchondroplasty of the talar osteochondral defect. Post-operative x-rays were taken and the patient had temporary pain reduction (See **Figure 3**).

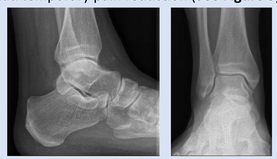

Figure 3: *Post-operative x-rays demonstrating intact ankle joint.*

Case Study (Continued)

Post-Operative Complications

Within five months of the initial surgery, patient's pain began to increase (VAS 6/10), creating difficulties with ambulation. After failure to have functional improvement, patient was sent for a post-operative MRI. MRI demonstrated AVN of the lateral half of the talus – beyond the boundaries of the initial lesion with additional loss of bone structural integrity laterally and as well as medially through access drill site (See **Figure 4**).

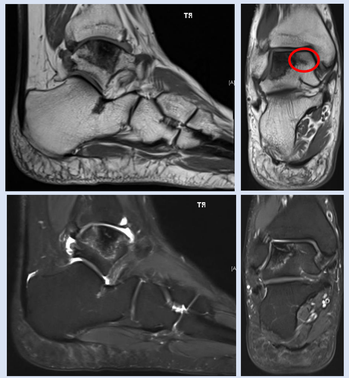

Figure 4: *Post-operative MRI demonstrating severe AVN of the lateral half of the talus. (Top Right) Noted red circle demonstrates how the drill hole used for the subchondroplasty was subject to localized AVN.*

Revision Surgery

Eight months following the initial surgery, the patient was dissatisfied with the outcome of the subchondroplasty and sought care from an orthopedic surgeon. After evaluation, the patient was offered a modified Brostrum lateral ankle stabilization procedure.

Case Study (Continued)

Further Post-Operative Complications

Following the Brostrum repair, the patient continued with non-weight bearing protocols for another two months. During the same period, patient was started on a bone stimulator before being transitioned to an Arizona brace. Repeat MRI was taken 8 months following the initial surgery (See **Figure 5**). The second MRI revealed further flattening of the talar dome, with no reduction or improvement of the previously identified AVN.

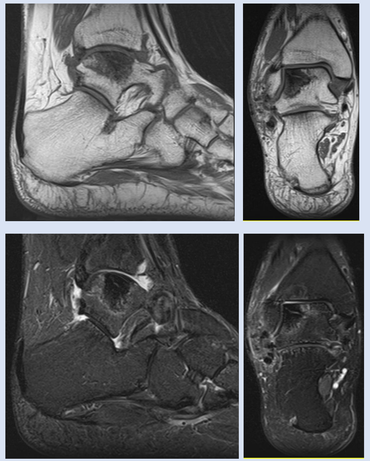

Figure 5: *Second post-operative MRI demonstrating severe AVN of the lateral half of the talus with consequential joint collapse.*

Repeat Lab Testing

Patient during these intra-operative periods were tested. Labs included: Anti-thrombin III Antigen 31/112, β-2 Glycoprotein IgG/IgA/IgM, Anti-cardiolipin IgG/IgA/IgM, Lupus anticoagulant, Factor V Leiden and Prothrobin mutations – **all of which were within normal ranges. However, the auto-anti-bodies were all at the extreme of the normal range**.

Analysis & Discussion

Despite the patient's repeated lab testing demonstrating normal values in terms of the activity of his APS status, the patient suffered catastrophic AVN of the talus following subchondroplasty. As detailed earlier, environmental triggers such as surgery can initiate the dysregulated inflammatory processes that lead to AVN. The talus itself is a vulnerable target in these situation due to the normally large amount of surface area covered in cartilage, limiting vascular access.

As mentioned, prior to surgical intervention, the patient's INR was between 3.0 and 3.5 per warfarin protocol and was bridged during the operative period with enoxaparin. Despite these efforts, the talus was not spared by the pro-coagulative events in the setting of this patient's APS.

This works' analysis suggests strongly the following key findings in terms of surgical management of OCD in the setting of APS:

1. APS auto-antibody levels serially tested and found to be in the normal range is likely insufficient in terms of indicating surgical candidacy for APS patients to receive foot and ankle surgery.

2. Conservative measures, emphasizing non-weight bearing protocols, may be the only treatment options for APS patients with OCDs.

3. The calcium phosphate material used in the subchondroplasty may exacerbate the coagulative dysregulation given the AVN tracking along drill access points and may need review in patients with similar coagulation syndromes.

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Financial Disclosure: The authors have no financial disclosures to make.