Reiter’s Syndrome: A Case Report on an Atypical Cause for Ipsilateral Ankle Edema

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Background: A 22 YO male Division I intercollegiate tennis player reported to the athletic training room with left ankle edema subsequent to a 14 hour plane flight. The patient neither recalled any specific mechanism of injury prior to or during the flight nor any previous history of injury to this limb. He was recently treated for a streptococcus infection; however, no residual symptoms of the illness were present at the time of our exam. His left ankle was red and swollen with palpable pain present at the insertion for the Achilles tendon. The patient’s gait was antalgic. Range of motion (ROM) for dorsiflexion and plantar flexion of the left ankle was limited. Conversely, the right ankle was injury-free. Differential Diagnosis: Achilles tendon rupture, Achilles tendon strain, lateral ligament sprain. Treatment: The patient was referred for definitive evaluation. Diagnostic ultrasound, X-ray, and magnetic resonance imagery findings were unremarkable. The initial course of treatment prescribed was rest, ice, compression, and elevation and an appointment with a team physician was scheduled. Re-examination focused more on systematic causes for the edema including inquiry about risk behaviors associated with sexually-transmitted diseases (STDs). Blood chemistry analyses were performed. Hematocrit (39.5% - normal = 41%-50%) and hemoglobin (13.9 g/dL - normal = 14.5-16.8 g/dL) values were slightly decreased whereas the Westergren Sedimentary rate (55 mm/hr - normal = 0-10 mm/hr) was elevated. The patient tested positive for the human leukocyte antigen-B27 (HLA-B27)(found in ~5% of Caucasians). All other blood counts were within normal ranges. A diagnosis of Reiter’s syndrome was made. Diclofenac (an NSAID) was prescribed and instructions to proceed with conservative treatment were given. Effleurage massage and intermittent compression treatments were initiated to reduce edema. A routine of 3 X 25 repetitions of clockwise and counterclockwise rotations on a balance board were performed to increase ROM, and wooden boxes were used for toe-heel raise exercises and Achilles stretching. A compression sleeve was worn throughout activities of daily living until his edema subsided. By week 2 subsequent to the initial evaluation, normal ambulation was restored; however, plantar flexion ROM was limited (10°). By week 3, full ROM was restored and jogging progressions were initiated. By week 4 the patient returned to normal practice and competition using a brace without residual complications. Uniqueness: When ipsilateral edema in a limb occurs, the cause is generally acute injury and orthopedic in nature. Systemically-evoked peripheral edema is seldom confined to one limb. This case presents a competitive athlete who experienced spontaneous ipsilateral ankle edema evoked by Reiter’s syndrome. Reiter’s syndrome is a rare condition where the body’s autoimmune system invades healthy tissue, typically an asymmetric joint, causing a profound inflammatory reaction. The condition tends to afflict males between 20 and 40 YO. The HLA-B27 is found in ~80% of individuals with Reiter’s syndrome, although not all individuals with the gene express it. Expression is believed to require some trigger such as contracting an STD. Interestingly, our patient had no history or outward signs of an STD (e.g., conjunctivitis, urethritis) and the link with his recent streptococcus infection serving as the trigger is purely speculative on our part. Indeed, the origin of Reiter’s syndrome remains elusive. Conclusions: This case presents a rare systematic cause for ipsilateral ankle swelling: Reiter’s syndrome. Despite the peculiarity for the cause of edema in this case, the standard course of treatment was effective in returning this patient to his chosen sport activity. Word Count: 600.