Rapporteur Report
23rd November 2024: Hall A

Foot and Ankle in Rheumatology

1) RA: Foot is the first to be affected and last to be treated and is the 2nd most common area for RA affection after hands.
2) Intra-articular injection: Sinus tarsi, Retrocalcaneal bursitis.
3) Widening of the forefoot can occur due to metatarsal synovitis, can lead to functional impairment
4) Collapse of the medial arch difficult to correct with orthoses or surgery.
5) Psoriatic arthritis: Rear foot involved first, more than forefoot/midfoot.
6) Four common sites of enthesitis in PsA: Tendoachiles, Peroneus brevis, Tibialis posterior and plantar fascia.
7) Sesamoiditis can be a manifestation of PsA.
8) Red flag in PsA and Ankylosing spondylitis:: bilateral plantar fasciitis with PD signal.
9) Be careful not to inject the tendo achilles.
10) Bauer digit: IP involvement + nail dystrophy in the foot, can be the first manifestation as mono/ oligo arthritis in PsA
11) Enthesitis: at various sites and may be resistant to treatment. TNFi and Tofacitinib have shown to be useful
12) Chronic tophaceous gout can also mimic PsA, and they may co-exist as well
13) Scleroderma: Can have refractory ulcers which may involve feet more than hands and need to be monitored regularly
14) Correcting the biomechanics is as important as systemic treatment in foot pain

SLE Symposium

15) Tyk2 inhibitors: Potential in moderately active non organ threatening lupus
16) TLR 7/8 antagonists and RNase IgG1-Fc: potential future agents based on ongoing trials.
17) Voclosporin: Significant benefit as add on therapy to background treatment with MMF.
18) JAK inhibitor Upadacitinib - marked improvement in remission of flare but high proportion of HZ in SLE

19) Tyk 2 ihibitor: Deucravacitinib trial showed low risk of HZ and an outcome driven by skin response. However, it was linked to severe COVID 19 infection and susceptibility to Mycobacterium tuberculosis.
20) Phase 1 trials TLR 7 / 8 antagonists showed markedly reduced IFN signals in SLE
21) Lupus clinical trials need to be bold: active disease needs better representation in RCTs.
22) Non immunosuppressive to be given to all SLE and LN- not based on class & not time dependent,
23) Even after remission, maybe life long continuation of non- immunosuppressives
24) Established agents: HCQ, RAAS inhibitors, statins, sun screen -
25) RAAS inhibitors: if proteinuria >=0.5 and above or in hypertension should be used as first line. They Works even in class 6 LN
26) RAAS inhibitors: delay the occurrence of lupus nephritis in those without kidney involvement.
27) Transient rise in creatine is expected and the agent should not be stopped in SLE
28) Emerging non-immunosuppressives: SGLT2 inhibitors, MRAs, GLP-1 receptor agonists, especially in those with refractory proteinuria
29) Early biologic treatment may help to disrupt the immunologic mechanisms of SLE and change the course
30) Vaccination is an important strategy to improve outcomes. Low vaccination rates are reported in SLE.

Vasculitis symposium

31) one should always be vigilant for other causes before treating definitively as vasculitis,
32) Red flags for alternate etiology: cytopenias, extremes of age, underlying other systemic diseases
33) Some common examples of mimics are TB, infectious endocarditis and rarely, syphilis
34) Malignancies including hematological, paraproteinemias, drugs, collagen disorders, APS

35) JAKi in Aortoarteritis: There is increased IFN signature and JAK STAT pathway in takayasu arteritis. •

36) JAKinibs act by inhibiting Th1 and Th17 derived cytokines

37) Tofacitinib inhibited neo-angenesis and vascular intimal hyperplasia in animal models of GCA

38) CD4+ and CD8+ T lymphocytes in TAK have ISG -Ruxolitinib inhibits these cells while increases Treg cell population

39) Jakinibs have shown promise in refractory as well as new patients with Takayasu in case reports- the response ranges between 70% to 85%

40) A subset of patients especially with extravascular manifestation may benefit more by these medications.

41) High serum levels of IL-23 in patients of Takayasu and differentiation of Th17 cells.

42) Both IL-12 and 23 activate JAK2 and Tyk2, hence Baricitinib may act better than Tofacitinib.

43) ndian data: Tofacitinib has been shown to be useful in difficult to treat Takayasu - in 69% in one study.

44) The middle part of the tongue, due to its high vascularity, is a typical area for vasculitic ulcers, especially medium vessel.

45) Initial trials, including CYCLOPS had a high mean dose of steroids.

46) Progressive trials, including the RAVE trial and finally the ADVOCATE trial included a lesser mean doses of steroids and faster taper.

47) ADVOCATE was the first trial to measure steroid toxicity with glucocorticoid toxicity index (GTI) which measures change in toxicity over time.

48) Targeted biologic therapy has improved AAV outcomes.

Clinico-pathological correlation

Differential diagnosis of vascultis with cytopenias:

VEXAS syndrome

- Vasculitis associated with lupus.
- Vasculitis associated with antiphospholipid antibodies.
- Infection-associated vasculitis.
- Malignancy-associated vasculitis.
- Drug-induced vasculitis.
- Hairy cell leukemia.

Tissue diagnosis is the cornerstone for diagnosis and treatment guidance in these situations.

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