Introduction

- Infective endocarditis can have a varied clinical presentation due to involvement of multiple organs with associated vascular and immunological phenomena.
- Rheumatological manifestations of infective endocarditis have been underestimated and their pathogenesis remains poorly understood.

Case

- A 55-year-old woman with PMH of hypertension, achalasia and carotid artery dissection presented with new-onset knee, shoulder, hand and bilateral wrist pain associated with facial flushing which worsened with stress and sun exposure.
- Physical exam was benign except tenderness to palpation on bilateral wrists.

Hospital course

- Patient spiked fever to 101°F on Day 2 of admission.
- Echo: 1 cm vegetation on the mitral valve (See Fig 2). Had normal echo one week prior.
- Found to have septic emboli to the bilateral cerebral hemispheres as well as an epidural abscess of her lumbar spine.
- Underwent an urgent mitral valve replacement (See Fig 3) as well as drainage of the epidural abscess.
- Blood cultures grew Group B Streptococci.

Post discharge

- Joint pains failed to improve several weeks after completion of treatment, and she was subsequently referred to Rheumatology.
- Work-up revealed a mildly positive RF, negative CCP, negative extractable nuclear antigen antibodies (ENA) panel and normal complement level.
- Unfortunately, the patient continues to have persistent arthritis requiring NSAIDs despite completing treatment for infective endocarditis over 8 months ago.

Discussion

- Severe infections have been known to generate an exaggerated immune response by various mechanisms including molecular mimicry, cytokine dysregulation or an immune complex-mediated process.
- Immunological cross-reactions between streptococcal and host molecules have been identified involving antibodies or T cells that react with streptococcal components and tissue antigens by means of molecular mimicry.
- Renal immunofluorescence studies in IE have shown the presence of granular IgG and CIC deposits on the membranes possibly due to the presence of circulating immune complexes. If the correlation between renal damage and musculoskeletal involvement is confirmed, it would probably indicate that immune complexes are also involved in development of the latter phenomenon.

Conclusion

- This case illustrates the importance of recognizing rheumatological manifestations as both presentation and/or complications of infective endocarditis.
- Further studies are needed to establish a possible immunological relationship between the two and to consolidate our understanding of the role of infections in the pathogenesis of rheumatological diseases.

References


Lab Data

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Fig 1: Hand X-ray with mild degenerative changes of the triscaphe joint and first carpometacarpal joint and small osteophytes.

Fig 2: Echocardiogram with Mitral Vegetation.

Fig 3: Mitral Valve Intraoperative.