HKCD Scientific Live Webinar

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Pioneering JAK science: How do JAK inhibitors fit in the PsA treat-to-target paradigm?

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Psoriatic arthritis (PsA) is a distinct disease encompassing various clinical manifestations including peripheral arthritis, enthesitis, dactylitis and axial disease. PsA can be underdiagnosed or misdiagnosed due to its heterogenous clinical presentations and overlapping symptoms with other inflammatory and rheumatological conditions. Apart from a lack of diagnostic laboratory markers, the number of well-validated tools for PsA screening is also limited. In a population-based survey of patients in North America and Europe, of 712 patients who reported a diagnosis of PsA, 72% reported developing skin signs and symptoms first. In the pathogenesis of PsA, various cytokines can signal through different Janus kinase (JAK) combinations. Tofacitinib targets the JAK intracellular signalling pathway. Pathogenic pathway cytokines in psoriatic arthritis are mediated or modified by tofacitinib. Targeting JAK pathways may enable simultaneous treatment of multiple PsA symptoms. Studies showed that tofacitinib improved joint symptoms and other relevant clinical domains including skin symptoms. Adverse events of special interest were infrequent and generally similar to those observed with biologic disease-modifying antirheumatic drug (DMARD), with the exception of an elevated incidence of herpes zoster. In the 2019 update of the EULAR recommendations of PsA, after failure of conventional synthetic DMARDs (e.g. methotrexate) and first-line biologics, switching to alternative biologics (TNF inhibitors, IL-17, IL-12/23, abatacept) or Janus kinase inhibitors (JAKi) is suggested. Tofacitinib may be considered if the patient prefers oral therapy.

Learning points:

With the potent efficacy and relative safety of JAKi, along with ease of oral administration, JAKi will be used more extensively and earlier in the treatment ladder of PsA.