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"Inflammatory Schmorl's Nodes" as Subtle Early Manifestations of Inflammatory Spinal Disorders

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Editorial

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In general, most of disorders of spine are classified as mechanical, inflammatory, traumatic, developmental, infiltrative and metabolic disorders. They can be usually diagnosed by using detailed medical history, thorough physical examination and other para clinical investigations in most instances. Nevertheless, differentiating inflammatory spinal diseases from mechanical disorders might become challenging particularly in an environment oversensitive to malpractice issues.

Rheumatologists are seemed to be mostly the sole spine specialists who are keener to differentiate those two disorders because they are more frequently confronted with missed cases of Ankylosing Spondilitis (AS) as degenerative or other mechanical disc disorders. This is partly due to taking more careful medical history and a comprehensive physical examination.

We believe some very early radiographic features of inflammatory spinal diseases (including sacroiliac joints) could be very similar to mechanical disorders, causing them to be misdiagnosed as degenerative changes. It is well known that inflammatory joint diseases are common causes of early progeric degenerative joint diseases in all joints universally including weight bearing and non-weight bearing articulations among all nations and worldwide. Typical and classic radiographic manifestations of rheumatic disorders are attributable to classic full-picture features of specific rheumatologic disorders such as AS or rheumatoid arthritis. We also believe that low grade systemic inflammation could explain polyarticular disorders, which were previously, were labeled as generalized osteoarthritis (OA). In a "continuous rather dichotomous viewing" to rheumatologic disorders, typical and classic features of rheumatologic conditions are at the end extreme of the spectrum of idiopathic joint diseases, while OA standing on the other side of this spectrum.

Clues for differentiation degenerative versus inflammatory conditions should not only be based solely on classic and traditional descriptions based on textbooks reproduced for decades but also there is important information closely related to patient's age, childhood rheumatologic history, family history of inflammatory joint disorders in ages bellow 40, occupation, relieving and exacerbating factors of pain, possible dramatic response to low dose anti-inflammatory drugs, strong serological history (rheumatoid factor, anti-nuclear antibody and so on) of close relatives, current and old serological profile and of course paraclinical imaging investigation at the end of individual "illness script".

In a longitudinal observation during more than 15 years and reviewing our big registry ("unpublished data") of more than 40,000 patients in our rheumatology clinic in Yazd, central Iran, we observed hundreds of young to middle age cases (mostly men) with typical inflammatory spinal pain, including low back, back and neck pain with typical morning stiffness and/or nightly accentuation of pain who labeled as back strain, discopathies, psychologic pain by several specialists for years but their history was against these diagnoses. In careful and thorough physical examination some had subtle features of seronegative Sponyl Arthritis (SpA) such as nail or skin changes in favor of psoriasis or gastrointestinal symptoms suggestive of inflammatory bowel disease (enteropathi carthropathy). Retrograde examination of old MRIs years before establishing frank "clinical and imaging" AS in some of our patients disclosed early changes of signal intensity in T2 W images in their old MRI films that simply reported as Schmorl's node or degenerative endplate diseases.

In laboratory investigation some of them had increased titers of C-reactive protein (CRP) or less commonly increased ESR as the most popular acute phase reactants. Beside from clinical clues to inflammatory spine disorders such as limited range of motion in lumbar or cervical spine, some had subtle or typical radiographic changes suggesting and or confirming sacroiliitis addressing early AS or other kinds of seronegative spondylarthropathies (SpA). More interestingly beside from increased signal intensity at spinal corners "shiny corner sign", or "Romanus lesion" there were other patients who displayed some subtle changes in magnetic resonance imaging (MRI) in form of "inflammatory Schmorl's node" destructive lesions at endplates which are less addressed as inflammatory lesions in radiographic reports (Figure 1). These are clearly distinguishable from classic degenerative Schmorl's nodes seen frequently in spine MRIs as a co-incidental findings. These findings are increased signal intensity "hallo" in T2Weighted MRI images around Schmorl's nodes. In some others, kissing lesions are evident. Multifocal lesions in a non-infective clinical setting argue against infective origin of spondylodiscitis. So non-contrast enhanced MRI could be a very sensitive imaging tool in



Figure 1: Inflammatory schmorl's nodes.

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displaying early inflammatory changes in SpA provided be combined with a careful clinical investigation [1]. Based on our search, the single study addressing to similar terminology is belong to Wu HT, who described evolving "edematous Schmorl's nodes" over time, without inferring to clinical implication and possible inflammatory nature of these lesions [2].

There is a similar story regarding Osteitis Condensance Ilii (OCI) and osteitis pubis which are classically, attributed to an accidental finding in multiparus women secondary to repeated micro trauma. We observed that our cases (mostly women) had typical "inflammatory" spinal or pelvic pain. Interestingly; however, none of them had more than two pregnancies and there were some single girls who showed radiographic features typical of OCI (Figure 2). So in the best scenario we could consider a subset of cases with OCI which cannot fulfill the classic definitions of OCI in our big registry in Iran. We hypothesize that these radiographic features are "feminine" version of subtle SpA rather than pathologies related to gestation and micro trauma. Jenks K et al. [3] simply emphasized strong association of sacroiliac joint tenderness and OCI supporting our idea without making any association with clinical SpA [3]. Sparing joint space is in fact a finding against traumatic and de-generative joint disease and we believe that osteitis per se could be a subtle feature of SpA bases on clinical background.

In a biased segregation of diseases solely based on recognition of classic features of them, most of cases of abortive or premature cases of inflammatory conditions can be misdiagnosed as degenerative



conditions. As our insight increases with better understanding of subtle features related to inflammatory spinal (and also non-spinal) disorders, especially by using more sensitive laboratory techniques indicative of occult systemic response to inflammation (as reflected by serum protein electrophoresis, acute phase reactants, and circulatory or local cytokines) our concepts may evolve from a simple consideration of most joint diseases to possible systemic diseases affecting joints. Continuing to rely only on observations and conceptualizations of pioneers for description and definitions of diseases while they were armed with very preliminary armamentarium could cause limitations in our modern era. There are small studies which have argued on the matter of differentiation of "inflammatory-spectrum" spinal disorders from degenerative one in a logic manner. Resnick et al. [4,5] published articles dealing with this topic with some critical limitations in 1976. They argued degenerative sacroiliac disease and full-blown AS which has a serious misleading point causing misinterpretation and underestimating premature or abortive inflammatory sacroiliac joint disease as local degenerative diseases. This might be somehow related to specialty bias due to the non-clinical nature of radiology field.

Finally putting together, from the point of an academic rheumatologist with more than 12 years of experience in clinical field, I believe we have possibly underestimated a huge amount of evidence that points toward the "bottom" of inflammatory "ice berg"; and looking "beyond the surface" is strongly advised. Unfortunately the major body of knowledge and imaging descriptions of rheumatologic conditions are built up by respected pioneer radiologists who might not have sufficient professional expertise and analytical skills in clinical setting of highly sophisticated rheumatology field. So a major revision in current concepts could be emphasized.

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