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CLINICAL AND LABORATORY PROFILE OF POST FEBRILE ARTHRITIS

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ABSTRACT: We have studied 47 cases out of hundreds of cases with poly arthralgias without signs and symptoms suggestive of typical arthritis over a period of one year i.e., January 2013 to January 2014 out of which 28 cases are women, 19 are men, with history of fever and signs and symptoms of URTI followed by polyarthritis involving small to medium sized joints, and in few with large joints with underlying osteoarthritis with sinovitis. All these patients were evaluated for routine as well as HLAB27, ANA and other viral markers for epidemic arthritis, out of which one case each of HLAB27 and Chikungunya Elisa positive, incidentally Rheumatoid factor positive in 3 cases.

KEYWORDS: poly arthritis, Reactive arthritis, post streptococcal arthritis, TNF (Tumor necrosis factor) - Non steroidal anti-inflammatory drugs (NSAIDs), Glucocorticoids, HLAB27.

DEFINITIONS AND BACKGROUND: Arthritis denotes an inflammatory reaction in the joint space. The inflammatory reaction is designated by activation of the innate immune defense mechanisms by e.g., microbial invasion, autoimmune reaction, or synovial deposition of crystals, such as monosodium urate monohydrate or calcium pyrophosphate crystals.

INTRODUCTION: There is an illness usually called post-streptococcal reactive arthritis, meaning only arthritis is present following streptococcal infection. This may be part of the many clinical features of post febrile arthritis.

Post-Streptococcal Arthritis: In adults and youngsters, cases of streptococcal associated arthritis are described that do not fulfill the criteria of acute rheumatic fever. Arthritis develops in the earlier phase of the disease and may involve joints of the hands. It responds poorly to anti-inflammatory treatment and, usually, lasts for months. These features resemble other forms of arthritis. The diagnosis relies on clinical findings, in association with evidence of recent streptococcal infection

Reactive arthritis denotes sterile joint inflammation, which is preceded by an infection elsewhere in the body and is typified by the presence of bacterial structures (Lipopolysaccharides, called also Endotoxin, DNA), but no multiplying bacteria, within the joint space.

Immune response to infection, such as hepatitis B virus infection, elicits circulating, complement-binding immune complexes, which may enter the joint and trigger immune complex arthritis.

The inflammatory reaction may be confined to the joint compartment, or it may be part of a systemic (Generalized) inflammatory reaction going on in the body. Systemic inflammation derives from the activation of the innate immune mechanisms in the circulation and thereby

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throughout the body.¹ Systemic inflammation may be secondary to a spill-over of pro-inflammatory cytokines from an inflamed joint into the circulation ², or it may represent a primary reaction triggered for instance by bacteria, which have entered the circulation and secondarily invade the joint tissue eliciting septic arthritis. Fever is a common clinical sign of systemic inflammation.

Although joint inflammation is frequently associated with fever, inflammation confined to the joints does not necessarily elicit fever, or elevation of body temperature may be prevented by the use of non-steroidal anti-inflammatory drugs (NSAIDs), immunosuppressive drugs, such as glucocorticoids and TNF (Tumor necrosis factor)-blocking agents, or by age.¹ Indeed, elderly patients with systemic inflammation associated with sepsis not so infrequently present with normal body temperature. Finally, febrile patients with severe sepsis may present with vital organ dysfunction so that the physician does not necessarily pay attention to "less urgent" problems such as the presence of infectious arthritis. In this study, we focus on adult patients with acute arthritis and with fever and without fever.

INCLUSION CRITERIA: 1. Patients with signs and symptoms of arthritis, with short duration fever in the past 2 weeks and above.

EXCLUSION CRITERIA: 1. Patients without fever at present and in the past. 2. Patients with history of trauma, and past history of arthritis on treatment.

METHODS: Participants are from outpatient department of GGH Kakinada, some from other hospitals, were screened for HBV, HCV and HAV in addition to Arthritis profile including HLAB27, ANA and Chikungunya Elisa. The data collected and statistically analyzed.

RESULTS: The data collected and distributed in sex, Age and laboratory results, represented in tabular and graphic pattern.

Sex Distribution	No. of patients
Male	19
Female	28
Total	47

Table 1

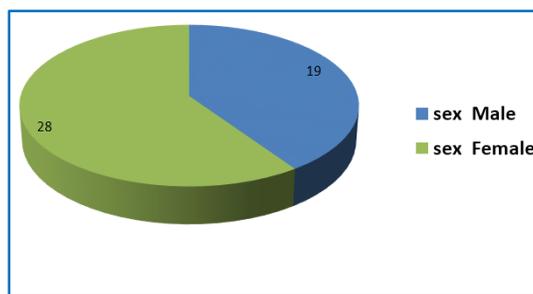


Fig. 1

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Age distribution	No. of patients
21-24	3
25-37	18
38-50	19
51-63	5
64-76	2
Total	47

Table 2

The P-Value is 0.000556

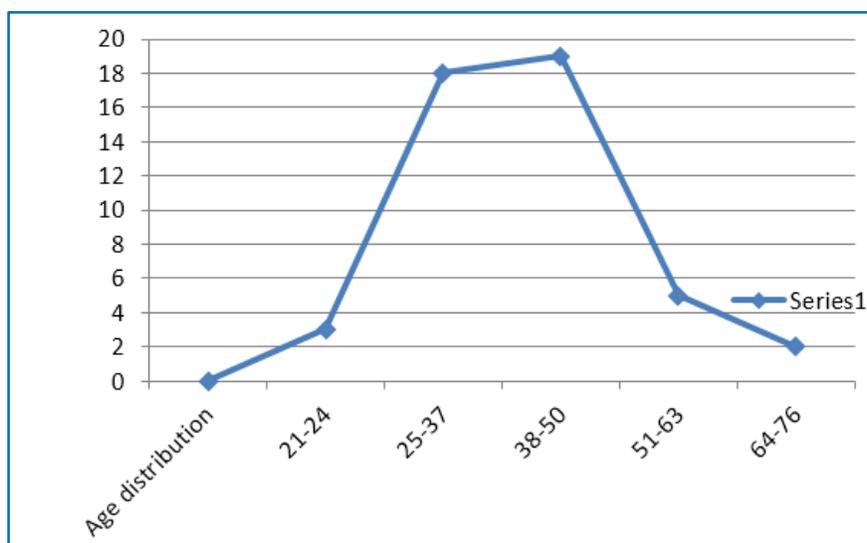


Fig. 2

ESR	35
CRP	21
R.FACTOR	3
ASO	11
ANA	0
HLAB27	1
CHIKUNGUNIYA	1
URIC ACID	1
UTI	3
OTHERS (T2DM)	1
Total	47

Table 3

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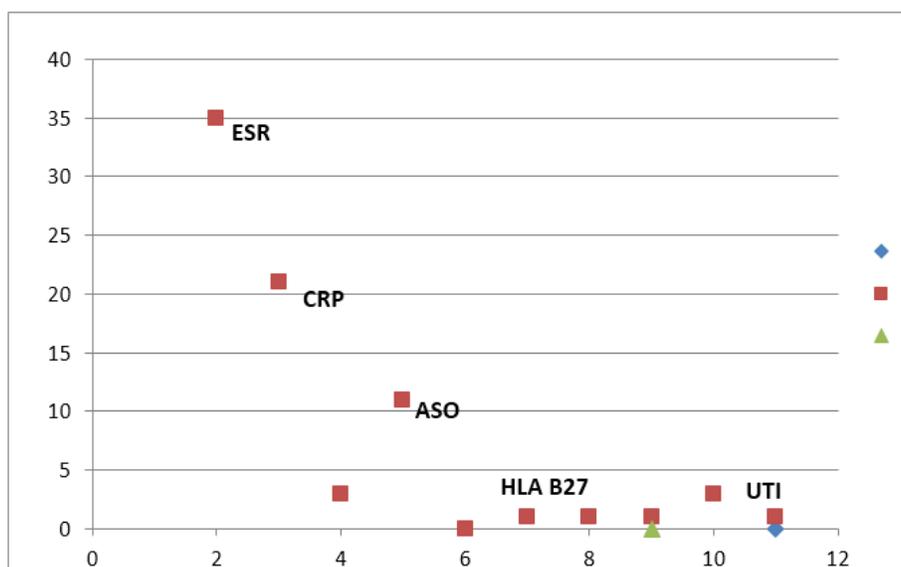


Fig. 3

DISCUSSION: It's an observational, analytical, prospective Evaluator case control study. Clinical and laboratory profile of 47 cases febrile and afebrile polyarthritis presented typically involving small to large joint pain with underlying co-morbid conditions like T2DM, O.A with mild hyperurecemia. Out of 47 cases, 28 cases are females and 19 cases are males; 19 cases are in the age group of 38-50 years; 18 cases in 25-30 years; 7 cases in 51-76 years. All the cases are screened for viral markers like HBsAg, HCV and HAV. In routine laboratory tests ESR is raised in 35 cases, CRP and ASO are 21 and 11 cases respectively. 1 case each of HLA B27, chikungunya are positive. 3 cases of Rheumatoid factor are positive; may be incidentally positive. In 3 cases of UTI patients we could find no microbiological or serological evidence of Chlamydia, mycoplasma and other viral infections.³

Since these are important causes for reactive arthritis. No case of lupus is found in this study. The raising acute phase reactants are even in afebrile patients; this indicates post infectious poly arthritis may not be due to common pathogens responsible for Reiter's disease or reactive arthritis.²

According to literature, HLA B27 is positive in 60-70% of cases of reactive arthritis; here we could find 1 case positive for HLA B27.⁴ Hence we ruled out Reiter's disease or reactive arthritis, since ASO titers are raised in significant number of patients we thought it may be non-Rheumatic post streptococcal arthritis.

Many of the patients responded to NSAIDS, HCQS, Quinolins and other non-narcotic analgesics, and other measures like rest followed by physiotherapy.

CONCLUSION: Polyarthritis following short febrile illness like common Flu, and viral fevers presented as URTI, after laboratory evolution and clinical presentation found to have secondary bacteriological involvement evidenced by elevated ASO titres ,nonspecific laboratory findings like HLAB27,RAF,CHIKUNGUNIYA may are may not be responsible for this clinical picture. All of them

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responded well to the Quinolines, NSAIDS, HCQS and non-narcotic analgesics followed by rest and physiotherapy, very few cases with co morbid conditions get partial response.⁵

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