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Original Article

High sensitive C-reactive protein and interleukin 6 in atrial fibrillation with rheumatic mitral stenosis from Indian cohort

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ABSTRACT

Introduction: Presence of chronic low grade inflammation has often been implicated in the etiology of atrial fibrillation (AF). Whether pre-existing inflammatory state promotes AF or initiation of AF activates inflammation is a dilemma among clinicians. This study investigates the role of high sensitive C reactive protein (hs-CRP) and interleukin 6 (IL-6) in AF with rheumatic mitral stenosis (Rh-MS) as markers of chronic inflammation.

Methods: This case control cohort included sixty five (n = 65) Rh-MS patients having other valve lesions as trivial to mild. Out of them twenty nine (n = 29; group C) had baseline AF and rest were normal sinus rhythm (NSR). A 24 h holter recording was done in NSR patients to diagnose paroxysmal AF/tachyarrhythmia forming group B (n = 12) and not having any tachyarrhythmia were designated as NSR; group A (n = 24).

Results: hs-CRP and IL6 showed statistically significant increase in group C (permanent AF) compared to group A (95% CI: 4.2–0.9, p = 0.007; 95% CI: 1.2–0.89; p = 0.05 respectively), while it was non significant between group A and group B (p > 0.05). A weak positive correlation was observed with hs-CRP and left atrial volume index (LAVi) (r = 0.45, p = 0.06) in AF group as compared to NSR group. 68.2% of patients in AF group (27/41) had moderate to severe spontaneous echo contrast (SEC) as compared to 37.5% (10/24) in NSR group.

Conclusion: Increased hs-CRP and IL-6 levels in the paroxysmal and permanent AF group may favour the hypothesis that low grade chronic inflammation could be the cause of atrial fibrillation than a consequence.

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1. Introduction

The alarming prevalence to 2.2/1000 population of rheumatic heart disease (RHD) continues to be a serious health burden in developing countries like India,[1] fuelling interest in clinicians and researchers to study its epidemiology, pathogenesis and prevention.[2] The clinical events leading to atrial fibrillation (AF) are multiple which include hemodynamic, electrophysiological and metabolic abnormalities, in addendum with genetic factors.[3,4] The dogma of inflammation is in quest since a decade whether initiation of AF activates inflammatory effects or the presence of pre-existing inflammatory state promotes persistence of atrial arrhythmias. It has been cited that low grade inflammation is not

Abbreviations: AF, atrial fibrillation; BMI, body mass index; CI, confidence interval; ECG, electrocardiography; ELISA, enzyme linked immunosorbent assay; hs-CRP, high sensitive C reactive protein; IL, interleukin; LA, left atrium; LAVi, left atrial volume index; LVEF, left ventricular ejection fraction; MMP, matrix metalloproteinases; MR, mitral regurgitation; MVA, mitral valve area; NSR, normal sinus rhythm; PHT, pressure half time; RHD, rheumatic heart disease; Rh MS, rheumatic mitral stenosis; ROC, receiver operating curve; RVSP, right ventricular systolic pressure; SD, standard deviation; SEC, spontaneous echo contrast; SVE, supraventricular ectopics.

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only a response to the underlying arrhythmic process but also an integral part of it .[5,6]

Biomarkers act as surrogate markers in understanding the biological, pathogenesis, clinical states of a disease in response to an intervention or identifying patients at high risk.[7] Inflammatory biomarkers could potentially refine clinical risk stratification for stroke and thromboembolism. The association of inflammation with non rheumatic AF has been demonstrated in recent studies with significantly raised high sensitive C reactive protein (hs-CRP) and interleukin (IL)6 in both paroxysmal and permanent AF with marked inflammatory infiltrates and myocyte necrosis in atrial biopsies.[8] It was observed that CRP was two-fold higher in AF patients when compared with control group having no history of atrial arrhythmia.[9]

The role of biomarkers in chronic low grade inflammation in atrial fibrillation in rheumatic mitral stenosis (Rh-MS) has not been addressed. The mechanism of this chronic process is debatable and is thought to be due to a continuing low-grade rheumatic process or due to hemodynamic stresses on the damaged valve.[10,11] The objectives of this research were a) to investigate the role of inflammatory cytokines– hs-CRP and IL-6 in isolated Rh-MS and their association with atrial fibrillation; b) to detect episodes of atrial arrhythmias in patients of Rh-MS in normal sinus rhythm (NSR) and their association with chronic inflammation.

2. Methods

This prospective observational case control study included patients with isolated Rh-MS with other valve lesions as trivial to mild (except tricuspid regurgitation without organic involvement). Patients of either sex, between 18 and 45 years were recruited from the cardiology clinics of the institute over a period of one year. Age matched patient's relatives or local hospital and community dwellers formed healthy controls. Patients with known supraventricular tachyarrhythmias with left ventricular ejection fraction (LVEF) <50%, coronary artery disease, recent cerebro-vascular accident within 3months), hyperthyroidism, diabetes mellitus, fasting blood sugar >126 mg/dl or on drugs/insulin), obesity – body mas index (BMI) >30, acute or chronic infections, malignancy, liver dysfunction, major surgical procedure in the last 3 months and renal failure (creatinine > 2.5 or on dialysis) were excluded. The study had ethics committee approval and written informed consent was obtained from all eligible patients prior to recruitment. Baseline demographic data, prior medications, clinically relevant history and examination was performed in all patients. All patients underwent routine hematological, biochemical and cardiac investigations like echocardiography (ECG) and doppler.

2.1. Echocardiographic evaluation

Echocardiography was performed on Philips IE 33(Philips Ultrasound, Bothell, WA) machine. Mitral valve area (MVA) was calculated by pressure half time (PHT) as well as by 2D planimetry. Lowest of the two was taken as actual MVA. Patients with mitral valve area $<1\,cm^2$ were classified as severe MS, between 1.0–$1.5\,cm^2$ as moderate and between 1.5–$2.0\,cm^2$ as mild MS.

2.2. Measuring LA size

Left atrium (LA) end-systolic diameter was measured in the parasternal long axis and apical 4-chamber view in M-mode at end systole. Antero-posterior, medio-lateral and apico-basal diameters

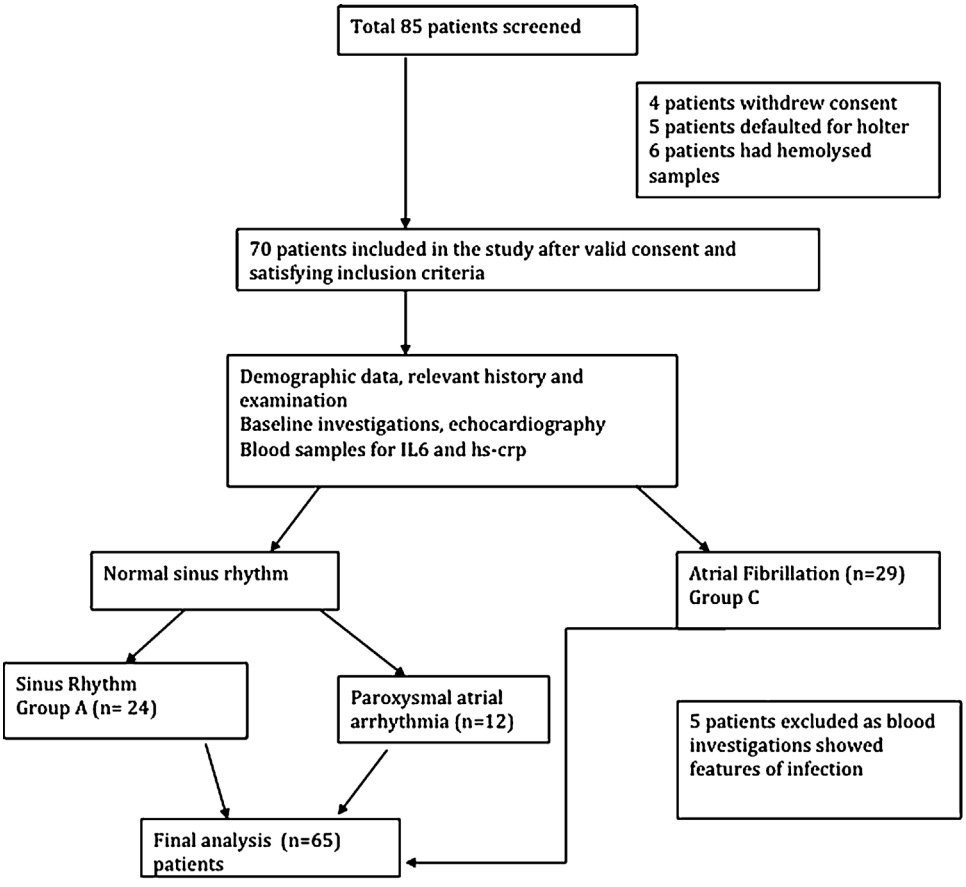

Fig. 1. Flow chart depicting the study design.

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