Original article

Electrocardiographic p-wave changes as marker of myocardial stress in male patients of chronic plaque psoriasis

[1]Dr Arun Kumar Metta, [2]Dr Sandhya Metta, [3]Dr Imran Ali, [4]Dr Pisati Navaneetha Reddy, [5]Dr Nunavath Madhukar

[1]Professor and Head, Department of DVL, Kamineni Institute of Medical Sciences, Narketpally, Nalgonda, Telangana.
[2]Ph.D Scholar, Department of Physiology, Gandhi Medical College, Secunderabad, Telangana.
[3]Department of DVL, Kamineni Institute of Medical Sciences, Narketpally, Nalgonda, Telangana.
[4]Post graduate, Department of DVL, Kamineni Institute of Medical Sciences, Narketpally, Nalgonda, Telangana.
[5]Post graduate, Department of DVL, Kamineni Institute of Medical Sciences, Narketpally, Nalgonda, Telangana.
Corresponding author : Dr Arun Kumar Metta

Abstract

Background: Plaque psoriasis is one of the most common forms of psoriasis. Since psoriasis is an inflammatory condition with high oxidative stress, the patients with psoriasis seem to be vulnerable for the development of atherosclerosis, hypertension and coronary artery disease.Electrocardiographic P-wave changes are considered as significant marker of myocardial stress.

Methods and materials: A total of 120 male subjects aged 20 to 50 years, out of which 60 were patients diagnosed with moderate to severe form of chronic plaque psoriasis were enrolled for our study. They were subsequently examined for electrocardiographic p wave changes by a 12 lead electrocardiogram and lipid profiles were evaluated.

Statistical analysis: The results obtained were statistically analyzed by using the Student's 't' test. The probability (p-value) was calculated

Results: The lipid profile of patient group indicated significantly high TC, LDL, VLDL, TG ($p<0.001$) and significantly low levels of HDL in comparison to controls P wave duration (PWD) were significantly higher in patient group ($p<0.001$) than control group.

Conclusion: Along with conventional markers of dyslipidemia, higher PWD should be considered as markers of hemodynamic stress for screening of chronic plaque psoriasis patients at the risk of developing cardiovascular diseases.

Keywords: P wave duration, electrocardiography, plaque psoriasis

Introduction

Psoriasis is a chronic and recurrent T cell-mediated disease involving CD4 and CD8 lymphocytes[1,2] and a known oxidative stress condition[3]. These cells, upon encounter with an antigen become activated and migrate to skin where they release a plethora of cytokines including TNF-∞, IFN-γ and IL-2, that initiate and perpetuate inflammatory response in skin [3]. There is proliferation and accumulation of monocytes and macrophages in blood and tissues [4].The monocytes and macrophages release oxygen metabolites and proteases which cause oxidative and proteolytic damage to plasma constituents and red blood cells [5]. Psoriasis is not just a skin disorder, it has also been shown to be a systemic inflammatory condition, similar to other inflammatory immune disorders such as rheumatoid arthritis and systemic lupus erythematosus [6]. Most of the cardiovascular

disorders, including atherosclerosis, hypertension, insulin resistance, and arrhythmias, share the same pathogenic mechanisms such as chronic inflammation, endothelial dysfunction, and increased oxidative stress [7]. Studies have shown that chronic psoriasis is associated with excessive cardiovascular morbidity and mortality [8]. Patients with psoriasis have a much higher risk of developing hypertension, atherosclerosis, and heart valve abnormalities [10]. Recently, Ahlehoff et al. demonstrated that psoriasis is associated with increased risk of atrial fibrillation (AF) [10]. As the most prevalent arrhythmia, AF is associated with increased risks of ischemic stroke, heart failure, coronary artery disease, and cardiovascular death. Since psoriasis is an inflammatory condition with high oxidative stress, the patients with psoriasis seem to be vulnerable for the development of arrhythmias. An electrocardiogram is a simple representation of the electrical activity of the heart muscle during the cardiac cycle. Recording of ECG is one of the easiest, cheap and reliable methods of assessing cardiovascular function. P wave dispersion (PWD) can be easily measured using a single ECG and is regarded as an electrocardiographic marker of prolongation of intra-atrial and inter-atrial conduction time in addition to heterogeneous and discontinuous propagation of sinus impulses [11,12] Studies have shown that chronic inflammatory conditions like lupus erythematous, meditterian fever etc induces changes in the p wave duration in normal ECG pattern[13,14]. Since there are very few studies on p-wave changes in psoriasis and specifically plaque psoriasis, the present study was undertaken to evaluate the lipid profile, and PWD in chronic plaque psoriasis, so as to identify the hemodynamic markers of cardiac stress for early screening of plaque psoriasis patients at the risk of developing cardiovascular diseases.

Materials and methods

Patients

The study comprised of total 120 male subjects aged 20 to 50 years, out of which 60 were patients diagnosed with moderate to severe form of chronic plaque psoriasis in the outpatient Department of Dermatology, Kamineni Institute of Medical Sciences, Hospital, Narketpally over a time period of eighteen months. Patients were evaluated clinically by the dermatologist based on history, cutaneous and systemic examination including site of the lesions, severity of erythema, amount of scaling and thickness of psoriatic plaques. Sixty age matched healthy controls were enrolled for this study, recruited from healthy volunteers and patients attending skin outpatient department for cosmetic problems like acne and pigment disturbances. The study was approved by institutional ethics committee. Informed consent were taken from the patients as well as the controls, before collection of blood sample and recording of ECG . All the patients included were having a PASI score of 8-12.[psoriasis area and severity index score (PASI score 8-12 for moderate psoriasis)].[15] Patients with diabetes mellitus, renal diseases, hepatic diseases and any other neuro-degenerative disorders were excluded from the study. Ten ml of overnight fasting blood sample was collected from all the subjects for the evaluation of lipid profile.

Methods

Body mass index (BMI):

The body mass index of all the subjects was calculated by the accepted formula

weight (kg)/[height(meter)^2.

Lipid profile:

Serum total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low density lipoprotein (LDL) were done by auto-analyser (Hitachi 912). Very low density lipoprotein

(VLDL) was calculated by Friedewald´s formula[16].

Blood pressure:

The systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded by sphygmomanometer in the morning, prior to collection of blood sample. Mean arterial blood pressure (MABP) was calculated with the formula: Diastolic pressure+1/3rd of Pulse pressure (DBP+1/3rdPP).

Heart rate:

Recording of pulse was done by palpating the radial artery for full one minute .

ECG Recording:

ECG recording was carried out in all the 120 subjects after thorough clinical and systemic examinations were done. With the subjects in the resting supine position, a 12 lead electrocardiogram was recorded by using a single channel ECG cardiant (heart view 1200 ECG recorder- manufactured by Brown Dove Healthcare Pvt Ltd). ECG recordings were obtained at a paper speed of 50 mm/s and 10 mm/mV amplitude. The beginning of the P-wave was defined as the point where the first atrial deflection crossed the isoelectric line, and the end of the P-wave was defined as the point where the atrial deflection returned to the isoelectric line. The P-wave durations (Pmax, Pmin) were calculated in all 12 ECG leads. The difference between Pmax and Pmin was defined as PWD [17]. Two ECG readers, who were blinded to the study, evaluated the ECGs. Initially, the measurements were performed manually with the help of callipers and a magnifying glass to define the electrocardiographic deflection.

Statistical analysis

The data was expressed as mean ± standard deviation. The results which were obtained were statistically analyzed by using the Student's 't' test. The probability (p-value) was calculated. A p-value of < 0.001 was taken as highly significant, a p-value of <0.05 as significant and a p-value of >0.05 as non- significant.

Results

The comparison of baseline data indicated no significant change in HR,SBP,DBP and MABP among both the groups. The lipid profile of patient group indicated significantly high TC, LDL, VLDL, TG ($p<0.001$) and significantly low levels of HDL in comparison to controls (Table 1).Upon clinical examination, all the study subjects were found to be in sinus rhythm. Pmax and PWD were significantly higher in patient group ($p<0.001$) than control group (table-2). Pmin was significantly lower ($p<0.001$) in patient group compared with controls.

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Table No 1: Baseline data and lipid profile of psoriasis patients and controls

(Data expressed as mean ± SD)

	Controls N =60	Psoriasis patients N =60	P-value
AGE (yrs)	42.9 ± 1.02	43.2 ± 1.64	>0.05
BMI(Kg/m^2)	19.72 ± 1.87	19.34 ± 1.41	>0.05
Total Cholesterol (up to 200 mg/dl)	162.65 ± 16	198.72±23.45	0.000 **
HDL(30-60 mg/dl)	46.07 ± 6.98	44.64 ± 3.56	>0.05
LDL(80-150 mg/dl)	97.1± 24.7	132.59 ± 26.58	0.000**
VLDL(10-30 mg/dl)	22.73 ± 5.72	27.36 ± 4.41	0.000**
Triglycerides (upto150 mg/dl)	115.04 ± 26. 57	138.29 ± 34.76	0.000**

**$P < 0.001$. BMI = Body mass index, HDL = High-density lipoprotein, LDL = Low density lipoproteins, VLDL = Very low-density lipoproteins, TC = Total cholesterol

Table-2 Comparison of P-wave values among psoriasis and patients.

	Controls (n=60)	**Psoriasis patients (n=60)**	**P-Value**
Heart rate (beats per min)	70.35±7.37	72.1±9.06	0.248(NS)
SBP (mm of Hg)	128 ±12.59	130.81±20.1	0.347(NS)
DBP (mm of Hg)	80.25± 4.39	79.34±6.95	0.380(NS)
MABP (mm of Hg)	96 ± 6.09	95.83±9.35	0.903(NS)
P max (ms)	97.2 ± 15.9	109.8 ± 26.9	0.002*
P min (ms)	45.9 ± 16.1	39.02 ± 14.3	0.014*
PWD (ms)	48.9 ± 20.4	58.9 ± 28.7	0.029*

Data expressed as mean ± SD, NS-Not significant, *p<0.05, SBP-Systolic blood pressure, DBP-Diastolic blood pressure, MABP-Mean arterial blood pressure

Discussion

Chronic plaque psoriasis is one of the most common form of psoriasis. This inflammatory skin disease often encountered clinically, seems to impose an oxidative stress condition. The lipid profile of patient group indicated significantly high TC, LDL, VLDL, TG ($p<0.001$) and significantly low levels of HDL in comparison to controls (Table 1).Dyslipidemia was found to be significantly associated with psoriasis as reported by us in the previous study published elsewhere [18]. Our study is concurrent with that of Lateef et al, (2011) who has reported similar findings in their study on lipid profile and protein fractions in psoriasis [19]. The association of dyslipidemia may be due to chronic inflammation, which is a characteristic feature of psoriasis, plays a role in the initiation and progression of atherosclerosis and coronary artery diseases [20]. While analysing the hemodynamic markers we did not observe any significant differences in HR, SBP, DBP and MABP(Table 1) among both the groups.While analyzing the p wave duration in patients and controls, Pmax and PWD were significantly higher in patient group ($p<0.001$) than control group (table-2). Pmin was significantly lower ($p<0.001$) in patient group compared with controls. The differences between the maximum (Pmax) and minimum (Pmin) P-wave duration on standard 12-lead electrocardiogram (ECG) are defined as P-wave dispersion (PWD). PWD can be easily measured using a single ECG and is regarded as an electrocardiographic marker of prolongation of intra-atrial and inter-atrial conduction time in addition to heterogeneous and discontinuous propagation of sinus impulses [11]. Prolongation of PWD has been demonstrated to be an independent risk factor for the development of atrial fibrillation (AF), which is the most common sustained arrhythmia in the general population that increases cardiovascular morbidity and mortality and decreases quality of life [12]. The maximum P-wave duration and PWD are commonly used to determine the risk of AF in several patient populations with inflammatory diseases [13,14]. In the present study, it was observed that the ECG markers of atrial conduction are altered in patients with plaque psoriasis. Enhancement of PWD strengthens the role of chronic inflammation for the provocation of atrial conduction abnormalities and consequently, the creation of the necessary pre-requisite for the development of atrial arrhythmia in these patients. We demonstrate in the present study that patients with psoriasis had impaired atrial conduction of sinus impulses. Prolongation of PWD, is an independent risk factor for the development of AF, could predispose these patients to other cardiovascular disorders as well [21]. The estimation of PWD, which is a reliable, non-invasive, and feasible variable with good reproducibility of intra and inter-atrial heterogenicity, could be a method useful to differentiate the group of patients prone to suffer from arrhythmias.

Therefore the present study concludes that patients with chronic plaque psoriasis have higher PWD, indicating an increased risk of atrial fibrillation in comparison to healthy controls. Although the exact mechanism still remains unclear, chronic inflammation may be responsible for increased PWD in these patients. Further, long-term prospective studies are needed to clarify the clinical utility and prognostic importance of PWD in patients with psoriasis.

Conclusion

Along with conventional markers of dyslipidemia, higher PWD should be considered as markers of hemodynamic stress for screening of chronic plaque psoriasis patients at the risk of developing cardiovascular diseases. The clinicians caring for the patient should inform their patients that they may have an increased risk of cardiovascular disease. Risk factors such as smoking, dyslipidemia, hypertension, and obesity should be assessed regularly in all psoriasis patients and should be advised for lifestyle modification including diet, exercise and smoking cessation in appropriate cases.

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