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SPECIAL COMMUNICATION

VENTRICULAR LATE POTENTIALS

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VENTRICULAR LATE POTENTIALS

Ventricular late potentials are low amplitude, high frequency wave forms appearing in the terminal part of QRS complex that may extend up to a variable length in ST segment. Ventricular late potentials are frequently seen in patients with sustained ventricular tachycardia[1]. They are noninvasive markers of an electrophysiological substrate for ventricular tachycardia that escort to sudden cardiac death[2]. Beriberi and Simson discovered ventricular late potentials in dogs for the first time in history. Initially ventricular late potentials were obtained directly from the endocardium or epicardium, but later on it was found that they can be recorded from the body surface[3].

PATHOPHYSIOLOGICAL BASIS

The heart myocardium has three morphological compartments: the muscular compartment comprises 30% consisting of myocytes, interstitial compartment produced by fibroblast and collagen, and the vascular compartment formed by smooth muscles and endothelial cells[4]. Ventricular late potentials are supported by modifications in these three morphological compartments due to necrosis, fibrosis or dystrophy to provide substantial anatomical substrate for reentry. The prerequisites for reentry are unidirectional block, slow conduction and revival of tissue ahead of the wave front of excitation, leading to serious ventricular arrhythmias[5]. Different diseases involve different mechanism to trigger electrophysiological changes in ventricular myocardium leading to potentially fatal arrhythmias[6]. Hypertension through increase in ventricular wall stress lead to gap junction and ion channel remodelling. This results in myocardial fibrosis escorting to early after depolarization and triggered activity to sustain the arrhythmias[4]. In myocardial infarction, ventricular late potentials arise through the peri-infarct zone, an area of myocardium with slow conduction. This means when depolarization in rest of the healthy myocardium is complete, this area is still depolarizing thus providing a substrate for arrhythmias[7]. In heart failure, interstitial fibrosis interdigitates with viable myocardium resulting in abnormal impulse conduction with slow ventricular activation. These complex electrophysiological changes are responsible for ventricular arrhythmias[6,8]. Thus delayed myocardial activitation through regional conduction block, long conduction delay and slow non uniform and discontinuous conduction affect the electrocardiographic signals between the end of QRS complex and the initial part of ST segment thus producing low voltage fractionated signals called ventricular late potentials[9,10].

DETECTION

Ventricular late potentials are detected through high resolution electrocardiography called signal averaged ECG. It is not possible to record them by surface ECG due to their amplitude in microvolt[11]. Signal averaged ECG was first introduced in 1970 and is recorded by using three standard orthogonal leads X, Y and Z[12]. These leads are combined in a vector magnitude called filtered QRS complex[13]. Signal averaging of all the leads is done and the system is able to average at least 100 beats per minute. Signal to noise ratio increases with the number of averaged beat. Filter frequencies of 25 to 100 Hz have been scrutinized but most recent systems used a 40 Hz high pass filter to decrease the electrical and skeletal muscle noise[14]. Ventricular late potentials are labelled if two of the following criteria are positive:

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1. Signal averaged ECG – QRS duration > 114 ms
2. Low amplitude signals under 40 μv in the terminal QRS complex for > 38 ms
3. Root mean square voltage in the terminal 40 ms < 20 μv

Besides time domain analysis, frequency domain analysis is also used to recognize ventricular late potentials in the terminal part of QRS complex but most signal processing systems used time domain analysis[15].

CLINICAL IMPLICATIONS

Sustained ventricular tachycardia and ventricular fibrillation result in sudden cardiac death. It is essential to lessen the risk of sudden cardiac death by accurate identification of risk factors in patients[16]. Driven by the need, signal averaged ECG is a noninvasive marker to identify ventricular late potentials in high risk patients and has been widely used since the last two decades[13]. Ventricular late potentials are of great importance for the diagnosis, risk stratification, therapy and prevention of patients with sustained ventricular arrhythmias[6,13].

MYOCARDIAL INFARCTION

Signal averaged ECG is a very useful noninvasive method for identification of patients with MI who are at risk of fatal arrhythmias[12]. Patients suffering from MI were more prone to sudden cardiac death due to ventricular tachycardia and fibrillation and had high mortality, Yodogawa Kenji et al demonstrated this fact in his study conducted on 50 patients with previous MI. 50 patients were included in control group. The signal averaged ECG record confirmed the presence of ventricular late potentials in 28 out of 50 MI patients (56%) versus 2 out of 50 control (4%)[17]. Percutaneous coronary intervention was associated with a significant reduction in prevalence of ventricular late potentials.10 Reperfusion of rigorously ischemic myocardium may also lead to haemorrhage in the infarct zone by extravasations of red blood cells through the damaged myocardium. Patients with myocardial haemorrhage had significantly prolonged filtered QRS duration on signal averaged ECG which may be related with serious malignant arrhythmias in the post infarction period[18]. MU Rabbani et al showed the utility of thrombolytic therapy in reducing the frequency of ventricular late potentials in patients with MI. The incidence of late potentials was 62.5% in inferior wall MI and 17.5% in anterior wall MI. Thrombolysed group with anterior wall MI had enormous chance of negative late potential than the inferior wall MI patients[7].

CARDIOMYOPATHIES

Cardiomyopathies are also one of the important causes of death in young people 19. Ventricular late potentials were observed in more than 50% of patients with arrhythmogenic right ventricular dysplasia[10]. A close correlation was revealed between signal averaged ECG and extent of disease[20]. Signal averaged ECG abnormalities were significant in cardiomyopathy related right ventricular outflow tract arrhythmias[21]. A considerable linear correlation was found between the extension of right ventricular outflow tract scar and all the three signal averaged ECG parameters[22]. Distinct scar distribution was found in non-ischemic cardiomyopathy. Ventricular late potentials were found in the anteroseptal group (11% versus 74%, $p < 0.001$) whereas late potentials were more frequent in inferolateral group (81% versus 4%, $p < 0.001$)[23]. In ischemic cardiomyopathy scar related reentry is the most common mechanism for ventricular tachycardia. Surviving myocytes may form channels that provide substrate for ventricular tachycardia. These channels were of greater length, had higher and longer filtered QRS, longer conduction time and slower conduction velocity than non ventricular tachycardia channels[24].

HYPERTENSION

Hypertension has a significant correlation with arrhythmias and sudden cardiac death.25, 26 Ventricular late potentials have a noteworthy prognostic value as an arrhythmogenic marker in hypertensive patients[27]. The most important mechanism by which hypertension predisposed to arrhythmias is related to the degree of left ventricular hypertrophy[28,29]. The prevalence of

left ventricular hypertrophy was higher in patients with secondary arterial hypertension than with essential hypertension.30 The prevalence of left ventricular hypertrophy in women was found to be 68% versus 30% in men. Angiotensin converting enzyme inhibitors were the only anti-hypertensive drugs related with lower risk of left ventricular hypertrophy[31].

DIABETES MELLITUS

Increased prevalence of ventricular late potentials was found in 72 children with type 1 diabetes mellitus. Increased duration of diabetes, cardiac autonomic neuropathy and thicker left ventricular posterior wall were the strongest risk factors for occurrence of ventricular late potentials[32]. Sanjuan *et al* found an association between hyperglycemia, acute ST elevation MI and arrhythmias[33]. Diabetes mellitus in patients with arterial hypertension altered the electrophysiological properties of myocardium thus providing a substrate for arrhythmias[34].

HEART FAILURE

Patients with heart failure have a high risk for sudden cardiac death despite therapeutic devices. Endomyocardial biopsies in patients with congestive heart failure point up interstitial fibrosis and hypertrophy. This can result in multifarious electrophysiological changes responsible for arrhythmias[6]. Matsuzaki *et al* demonstrated the usefulness of 24 hours ambulatory monitoring of ventricular late potentials to predict the prognosis in patients with heart failure. Using left ventricular ejection fraction, filtered QRS and low amplitude signals as the three predictors, the specificity and accuracy was found to be 83% and 82% respectively[15].

OTHER DISEASES

Signal averaged ECG parameters were deranged in patients with chronic renal failure. Hypokalemia provoked arrhythmogenecity results in slow conduction providing a substrate for ventricular late potentials[35,36]. Chronic obstructive pulmonary disease is an independent risk factor for arrhythmias[37]. Carjea *et al* studied the prevalence of ventricular late potentials in 90 patients with chronic obstructive pulmonary disease compared to healthy subjects and found significant difference[38]. A noteworthy correlation was found between dyslipidemia and filtered QRS, low amplitude signals and root mean square

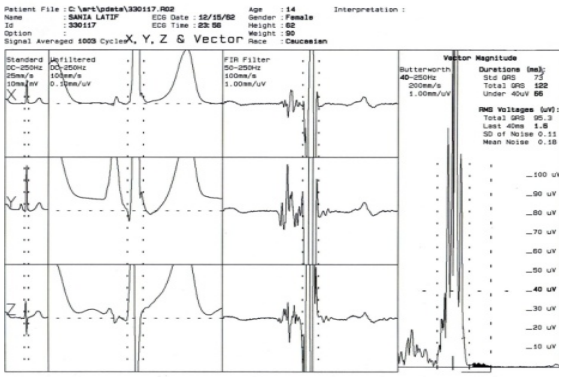

Figure-1: Ventricular late potential detection through signal averaged ECG.

voltage[39]. Thalassaemia patients need multiple blood transfusions due to severe anemia resulting in iron over load. This iron over load provides a substrate for reentry and fatal arrhythmias[40]. Ventricular late potentials were also found to be positive in patients with bundle branch block, structural heart diseases, brugada syndrome, acromegaly and schizophrenia[6,41,42].

Sudden cardiac death caused mainly by lethal ventricular arrhythmias, can be predicted by using noninvasive and low cost tool i.e. signal averaged ECG. Ventricular late potentials detected through signal averaged ECG help to better stratify the patients with risk of arrhythmias, alone or in amalgamation with other methods, in several clinical settings.

Conflict of Interest

This study has no conflict of interest to declare by any author.

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