Vol 3, Issue 30, 2022

CARDIOMAG NEWSLETTER

Volume 3| Issue 30| 2022

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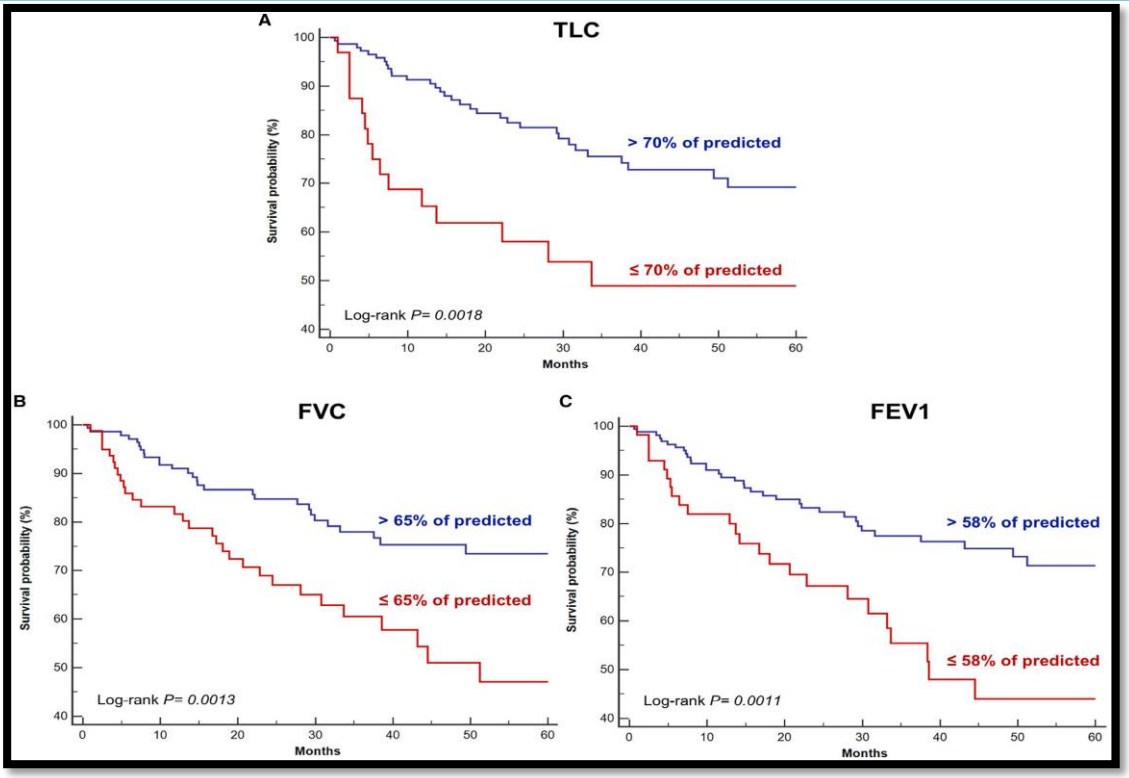
Association Between Pulmonary Hypertension and Ventilatory Defects in patients with heart failure with Reduced Ejection Fraction

Patients with Heart failure with reduced ejection fraction usually have functional decline and impaired quality of life. In patients with heart failure, dilated left ventricle and elevated left ventricular end-diastolic pressure worsens the normal ventilation leading to cardiopulmonary interaction.[1] Abnormalities in lung function both at rest and during exercise, are mostly seen in patients with heart failure.[2] According to studies, abnormalities in ventilation and diffusion with decreased in lung volumes was observed in up to 60% of patients with end-stage reduced ejection fraction.[2] Increased in Left ventricular filling pressure not only causes ventilatory abnormalities, but also lead to pulmonary hypertension, due to the backward pressure transmission.[1] Study says that presence of Pulmonary hypertension may affect the pulmonary function but the correlation between ventilatory abnormalities and the Pulmonary hypertension attributable to left heart disease is yet to be cleared.[1]

In patient with Pulmonary Hypertension, ventilatory abnormality was observed more common with heart failure with reduced ejection fraction compared to patients without Pulmonary Hypertension

A study conducted by Chang H, et al aimed to investigate the association between ventilatory abnormalities and Pulmonary hypertension in heart failure patients with reduced ejection fraction and enrolled 440 ambulatory patients having left ventricular ejection fraction ≤40% and underwent comprehensive echocardiography and spirometry. Pulmonary function test like total lung capacity, forced vital capacity, and forced expiratory volume in the first second were conducted. Primary outcome of the study was mortality of all-causes at 5 years. Decreased in total lung capacity, forced vital capacity, and forced expiratory volume in the first second was observed in patients with Pulmonary hypertension.

Fig:1 Kaplan-Meier survival curves for 5-year all-cause mortality in patients with heart failure with reduced ejection fraction without pulmonary hypertension (pulmonary arterial systolic pressure [PASP] ≤50 mm Hg), stratified by total lung capacity (TLC) (A), forced vital capacity (FVC) (B), and forced expiratory volume in the first second (FEV1) (C).



Around 111 deaths were recorded during a follow-up of 25.9 months. Left ventricular ejection fraction, functional capacity, total lung capacity forced vital capacity and forced expiratory volume in the first second were all significantly correlated with mortality in patients without Pulmonary hypertension. According to Kaplan-Meier curve, impaired pulmonary function was associated with higher mortality rate in patients without Pulmonary hypertension, regardless of their functional status **(Fig:1).** Patients with Pulmonary hypertension had significantly enlarged left atrium, increased mitral inflow E/A ratio, and increased E/e' ratio compared to patients without Pulmonary hypertension. Lower total lung capacity was observed in patients with Pulmonary hypertension than those without Pulmonary hypertension, but FEV1/FVC were about the same between with Pulmonary hypertension and without Pulmonary hypertension group. Though patients were having normal ventilatory function but the restrictive ventilatory abnormality was more prevalent in patients with Pulmonary hypertension

Conclusion:This study demonstrated that in patient with Pulmonary Hypertension, ventilatory abnormality was observed more common with heart failure with reduced ejection fraction when compared to patients without Pulmonary Hypertension. Regardless of the functional status, ventilatory defects were associated with long-term survival only in patients without Pulmonary Hypertension

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Involvement of bone marrow activity in the association between vascular inflammation and atherosclerosis.

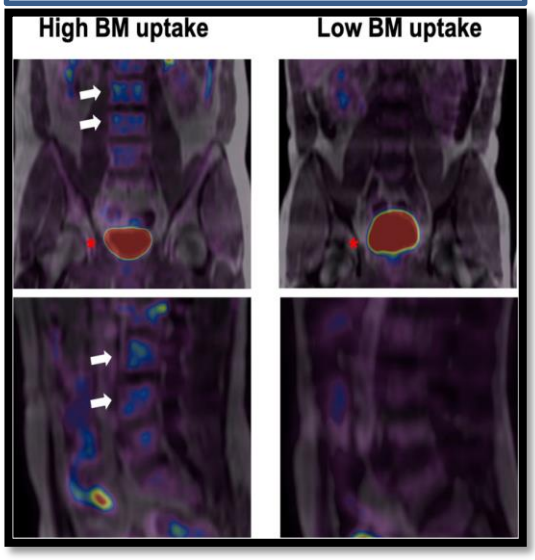
Linked between inflammation and atherosclerosis have been established, and it is demonstrated that inflammation is an important mediator of atherosclerosis, from initiation to progression and the development of thrombotic complications.[1] Various deleterious effects such as promoting plaque activation can be found in systemic inflammatory process associated with the metabolic syndrome.[2] Association between the innate immune system and lipid-derived products are the important factors in the pathophysiology of atherosclerosis in relation with the metabolic syndrome.[2] On the other hand, bone marrow is known as the primary site of haematopoiesis, and various physiological and pathological stimuli regulates the proliferation and migration of haematopoietic progenitors.[1] According to the studies, increased in the activity of bone marrow haematopoietic is linked between Cardio metabolic risk factors and exacerbated inflammation in atherosclerosis.[1]

In the development of atherosclerosis, Bone marrow activation appears to be an early phenomenon

A study by Devesa A et al, aimed to study the association between the cardiovascular risk factors, BM activation, and subclinical atherosclerosis and enrolled 745 apparently healthy individuals.[1]

All patients were asked to perform whole body vascular 18F-fluorodeoxyglucose positron emission tomography/magnetic resonance imaging. In the lumbar vertebrae, bone marrow activation was assessed. Circulating biomarkers were used to detect systemic inflammation Early atherosclerosis was identified by observing the uptake of 18F-FDG in five vascular regions while late atherosclerosis was detected by observing the MRI showing fully formed plaques.

Fig: 2:High and Low BM uptake



In patients with Metabolic syndrome, bone marrow activation was significantly associated. At the same time, bone marrow activation was associated with increased leucocyte (mainly neutrophil and monocytes) and erythrocyte counts, increased high-sensitivity C-reactive protein, ferritin, fibrinogen, P-selectin, and vascular cell adhesionmolecule-1. However, in the subgroup of participants with no systemic inflammation, associations between bone marrow activation and Metabolic syndrome and increased erythropoiesis were maintained. Elevated arterial metabolic activity i.e. 18F-FDG uptake was linked with bone marrow activation and the co-occurrence of bone marrow activation and arterial 18F-FDG uptake was associated with advanced atherosclerosis (Fig:2)

Conclusion:This study highlighted that in patients with no systemic inflammation, uptake of bone marrow ^{18}F-FDG is associated with Metabolic syndrome and its components. Early atherosclerosis is linked to Bone marrow activation characterized by high arterial metabolic activity

Reference

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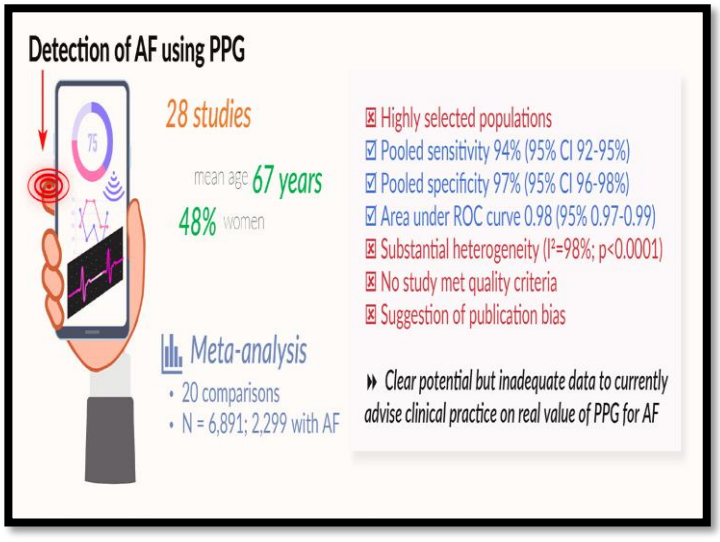
Diagnosis of Atrial Fibrillation using smartphone camera photoplethysmography: A systematic review and meta-analysis

Atrial fibrillation is the disorder of cardiac rhythm associated with increased morbidity and mortality.[1] Early detection of Atrial fibrillation is important in the prevention of primary and secondary stroke, as it is the leading risk factor for cardio embolic stroke.[2] Atrial fibrillation is encountered by healthcare professionals with increasing prevalence specially in elderly population with comorbidities. Electrocardiogram Interpretation by a trained cardiologist is the gold standard for detecting Atrial fibrillation.[2]According to the European Society of Cardiology guidelines, elderly patients can be benefitted from the early screening of Atrial fibrillation with the help of single time point, repeated or ambulatory electrocardiogram recordings.[1]As Atrial fibrillation is brief and sporadic, there might increases the chances of missing paroxysmal episodes. However, diagnosing atrial fibrillation on time is essential to decrease complications.

Atrial fibrillation detection with high sensitivity and specificity can be possible with Photoplethysmography technology

A study conducted by Gill S et al, aimed to analyse the diagnostic accuracy of smartphone camera photoplethysmography compared with conventional electrocardiogram for the detection of Atrial fibrillation. This systematic review included 28 studies (11404 participants), where comparison was done between smartphone photoplethysmography and reference ECG (1, 3 or 12-lead). Primary and secondary outcomes were validation of Atrial fibrillation detection by examining: sensitivity, specificity, positive predictive value, negative predictive value and overall accuracy.

Fig:3 Detection of AF using PPG



Of the total studies,10 studies were full-text publications and 18 were abstracts providing 31 comparisons of smartphone photoplethysmography versus ECG for the detection of Atrial fibrillation.**(Fig:3)** For the detection of Atrial fibrillation, sensitivity and specificity were high ranging from 81% to 100%, and from 85% to 100%, respectively.Twenty comparisons from 17 studies were meta-analysed, including 6891 participants (2299 with AF), the pooled sensitivity was 94% and specificity 97% with substantial heterogeneity . Accuracy was reported in 18 comparisons and ranged from 61% to 99%. Overall, the area under the receiver operating curve was 0.98, with substantial significant heterogeneity .

Conclusion: With the growing demand of smartphones, photoplethysmography provides a non-invasive, patient-led screening tool for the detection of Atrial fibrillation. Atrial fibrillation detection with high sensitivity and specificity can be possible with Photoplethysmography technology

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Association between very Low Carbohydrate Ketogenic Diet and Cardiovascular Risk: A Case Report

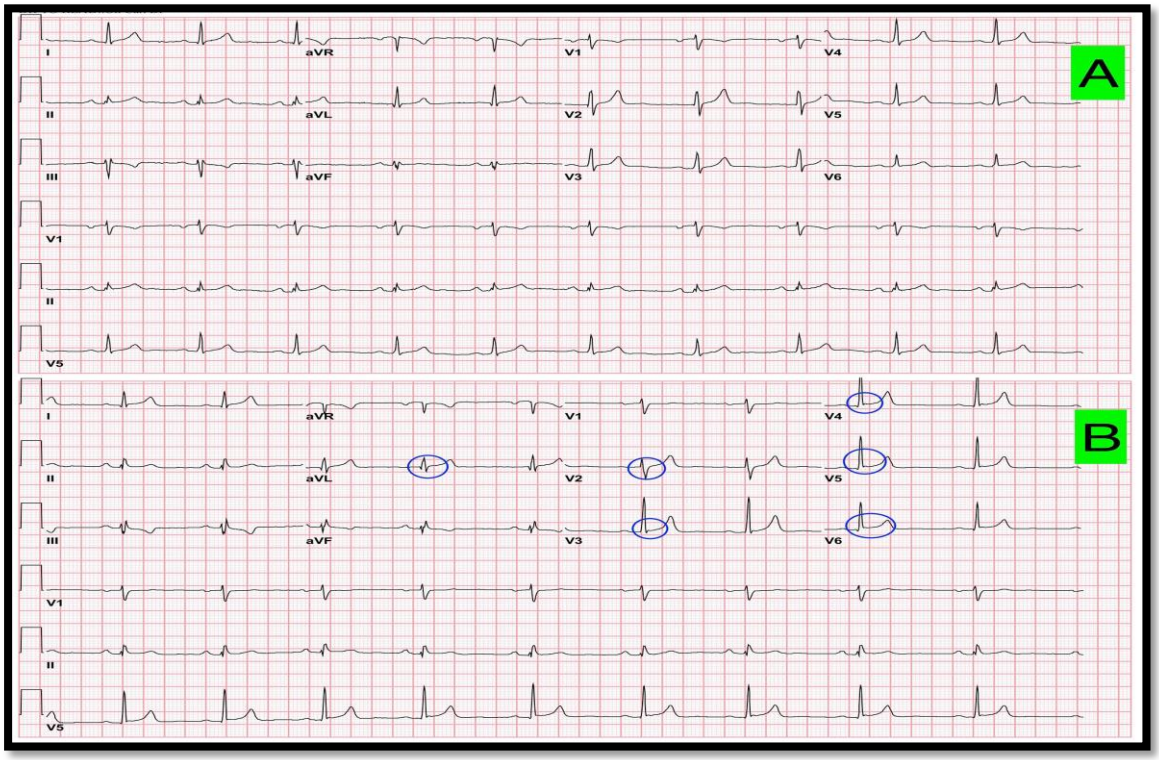
It is common for people in very-low-carbohydrate ketogenic diet to have rapid metabolic changes at the cellular level such as depletion in glycogen stores, decreased insulin level and mobilization of free fatty acids to the liver creating ketone bodies. The physiologic and metabolic changes are directly proportional to the severity of carbohydrate dietary restriction.[1] In very-low-carbohydrate ketogenic diet, carbohydrate is restricted to less than 50 grams/day (or < 200 kcals/day, since each gram of carbohydrates provides 4 kcals of energy). This indicates that the carbohydrate consumed is < 10% of total daily energy for the average 2,000 kcal/day diet, which is very less than the recommended and acceptable macronutrient ranges for carbohydrate, i.e. 45 - 65% of total calories or 225 - 325 grams of carbohydrate/day for a 2,000 kcal/day diet.[2] This case report demonstrated a strong temporal association between very-low-carbohydrate ketogenic diet and type 2 myocardial infarction.[1]

Middle age male patient was presented with severe, dull, continuous, retrosternal chest pain radiating to the neck, and upper back associated with dyspnoea and nausea that started 6 hours prior to the presentation. Patient's past medical history revealed obesity (body mass index = 33 Kg/m2) and seasonal asthma. Physical examination did not show any palpitations, lower extremity swelling, dizziness, loss of consciousness, fever, chills, or sweating. Patient did not have the history of respiratory symptoms, recent travel, trauma, overexertion, or contact with sick individuals, neither have any habits of smoking, drinking or drug abuse. No family history of premature cardiovascular disease was detected.

Although there is no clarity in the pathogenesis, but the temporal association between very-low carbohydrate ketogenic diet and type 2 myocardial infarction increased some concerns about very-low-carbohydrate ketogenic diet's cardiovascular safety profile

Patient had initiated a carbohydrate ketogenic diet of 15 grams carbohydrates/day and lost 25 lbs, 4 weeks prior to the admission. Daily carbohydrate intake was further decreased to 10 g/day, 1 week prior to the onset of chest pain. Patient had stable Vital signs with normal blood pressure and pulse rate. But, slight reduction in serum bicarbonate level was observed while complete metabolic panel was normal. Patient was asked for urinalysis, which revealed ketonuria (80 mg/dl) and urine specific gravity > 1.050 but urinary tract infection was not detected.

Fig: 4 Dynamic ST-segment changes in electrocardiogram (EKG). (A) normal EKG upon admission. (B) 1- mm ST elevation in anterolateral leads,1 day after admission (blue circles).



Creatinine kinase level was 539 U/L. On electrocardiogram, changes in ST-T waves were observed along with elevation of troponins, ketonuria, and mild rhabdomyolysis **(Fig:4).** Transthoracic echocardiogram showed mild inferior wall hypokinesia and cardiac catheterization showing normal coronaries and the treatment was initiated with aspirin, carvedilol, atorvastatin, morphine, sublingual nitro-glycerine, and heparin infusion. Chest pain got resolved immediately after the administration of sublingual nitro-glycerine which was there for continuous 6 hours prior to the admission. Cardiac catheterization ruled out coronary atherothrombosis and heparin infusion was then discontinued and the patient was discharged on aspirin and moderate-intensity statin therapy was strictly advised for the prevention of primary Coronary Artery Diseases.

Conclusion: Although there is no clarity in the etiopathogenetic mechanisms of type 2 Myocardial Infraction in very-low-carbohydrate ketogenic diet, it is to be believed that oxidative stress, nutritional ketosis, and cardiac muscle degradation plays an important role in causing type 2 Myocardial Infraction

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