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Case Report

Non Angiographic Prediction of Coronary Artery Disease in Obese Patients with Prominent Thick Layer of Pericardial Fat: A Case Report

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Abstract

In this report, we present a case of middle aged obese women with no coronary artery disease risk factors, who presented with recurrent angina and was admitted for work up and possible coronary intervention. During the work up we found that the patient has a prominent thick layer of pericardial pad of fat. The patient underwent coronary angiogram and percutaneous coronary intervention with angioplasty and coronary stent. We believe and also as shown in previous studies that the thickness of this layer could correlate with the coronary artery disease and can be used for risk stratification.

Introduction

Epicardial fat is in direct contact with coronary arteries. Pericardial fat is the visceral fat of the heart deposited under the visceral layer of the pericardium and has the same origin as abdominal visceral fat, which is shown to be strongly related to the development of Coronary Artery Disease (CAD) [1]. The accumulation of epicardial and pericardial fat is known to be a rich source of free fatty acids and a number of inflammatory cytokines [1-3]. Inflammatory mediators that originate in vascular and extravascular tissues promote metabolic syndrome and coronary lesion formation. Adipose tissue may function as an endocrine organ that contributes to an inflammatory burden in patients at risk of cardiovascular complications.

Transthoracic echocardiogram can measure the subepicardial adipose tissue on the free wall of right ventricle at end-diastole from parasternal long- and short-axis views of three cardiac cycles [4,5].

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Herein we present a case of 48 year-old with pericardial fat pad and coronary artery disease.

Case Presentation

A 48 years old female obese patient non smoker, non diabetic, non hypertensive and no other coronary artery disease risk factors, she had cardiac angiography 2 years ago for chest pain revealed normal coronary arteries. She presented to the outpatient clinic with recurrent angina increasing for the last 2 months, so performed nuclear stress test which showed mild anteroseptal ischemia (Figure 1) and was kept on anti ischemic treatment. She continued to have recurrent chest pain so was admitted 7 months later for coronary angiogram and possible coronary intervention.
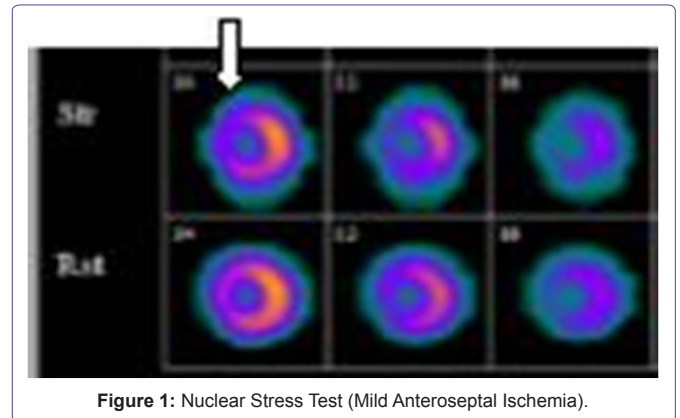

Figure 1: Nuclear Stress Test (Mild Anteroseptal Ischemia).

Vital signs: Blood pressure 111/64 mmHg, pulse rate 75 beat/minute, height 160 cm, weight 109 kg (BMI 38.5).

Cardio vascular system: normal S1S2 no added sound and no mummer, Chest: good air entry bilaterally with no added sound, Abdomen: soft, lax, lower extremities: lipodema.

Laboratory investigations showed normal complete blood cell count & renal function and lipid profile test. Initial high sensitivity C-Reactive Protein (hs-CRP) was elevated 9.9 mg/ml (normal <5) and in follow up normalized 4.0 mg/l.

Electrocardiography showed normal sinus rhythm and heart rate 67 beat/minute and nonspecific anterior ST-T changes.

Chest x-ray showed well defined faint large opacity/shadow project over the right paracardiac and cardiophrenic angle area composed of thickened wing like most likely presenting prominent pericardial fat pad (Figure 2), CT Chest was done and showed exaggerated pericardial fat (Figure 3), Epicardial fat measured 8 x 4 cm in thickness, abdominal CT showed fatty liver.

Echocardiography showed prominent thick layer of pericardial pad of fat with no other significant abnormalities (Figure 4).

She underwent cardiac angiography which showed: left main: mild plaque; Left Anterior Descending (LAD): proximal mid 70% and mid 80-90% stenosis (Figure 5), left circumflex artery: mid 30%, right coronary artery: mid 30%. So successful direct stenting was done
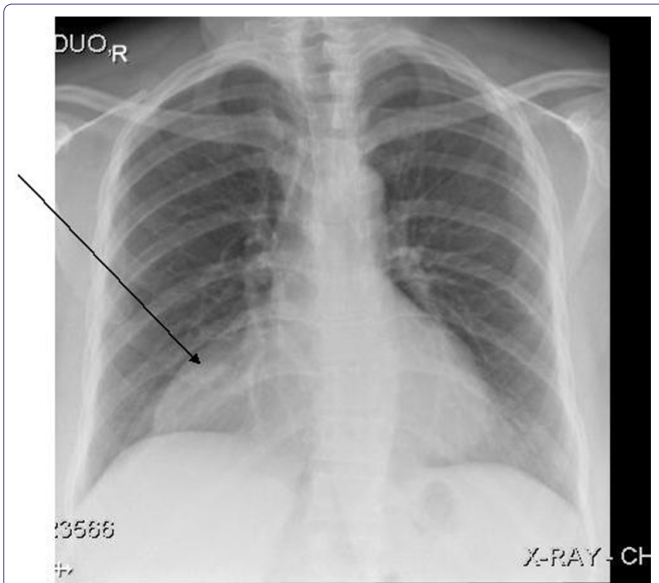
Figure 2: Chest x ray (Large shadow over the right paracardiac and cardiophrenic angle).
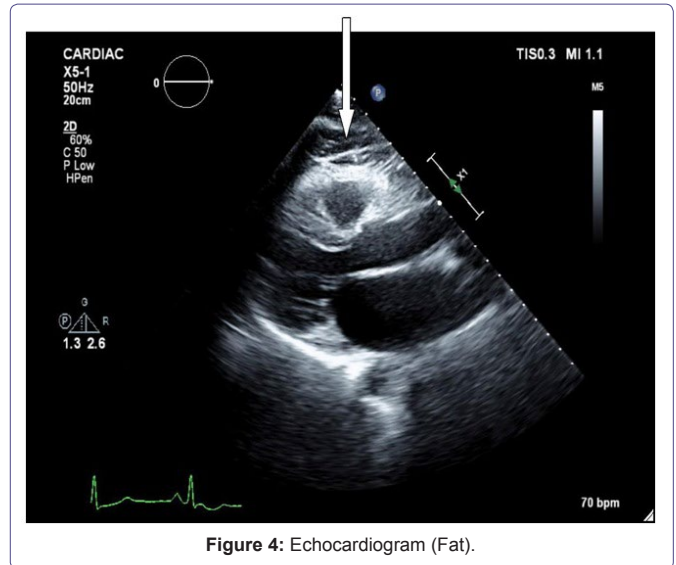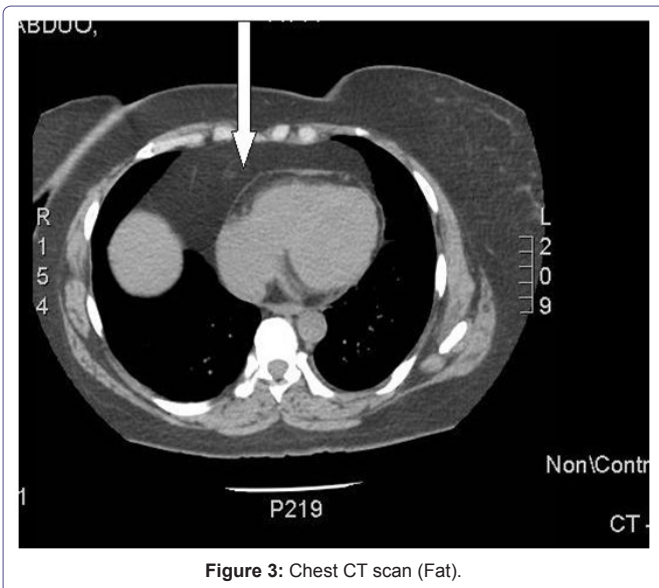

Figure 3: Chest CT scan (Fat).

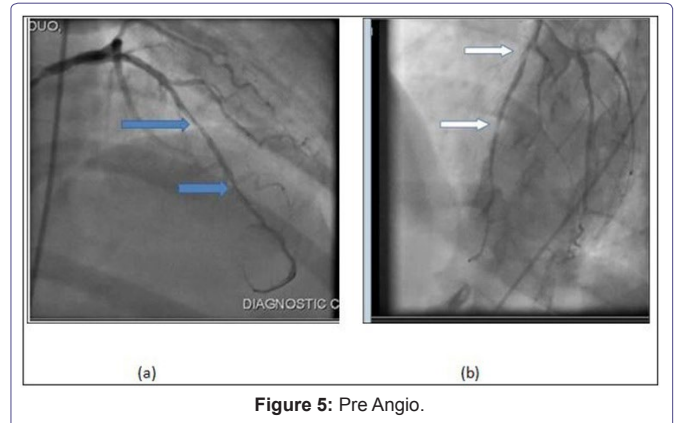**Figure 4:** Echocardiogram (Fat).

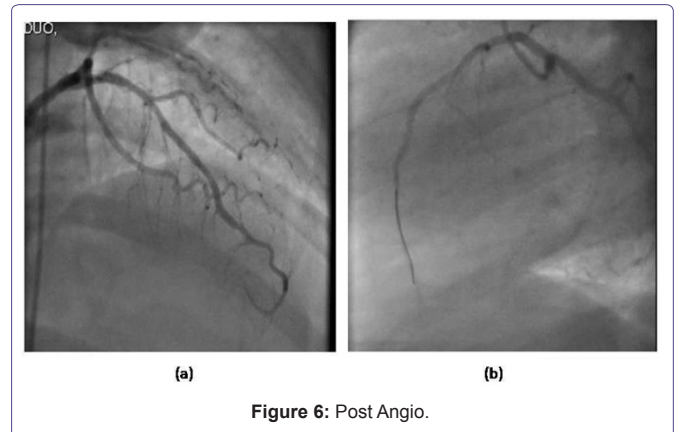

Figure 5: Pre Angio.

Figure 6: Post Angio.

to left anterior descending with 2 stents (Figure 6), the patient tolerated the procedure well without any complication. The patient transferred to floor for 24 hour observation, she did well and She was discharged on the following medications: Acetylsalicylic acid 81 mg daily, Clopidegrol 75 mg daily, Atrovastatin 20 daily, Metoprolol 50 mg daily, Perindopril 5 mg daily. She has been followed up in cardiology clinic for 8 months so far and asymptomatic.

Discussion

Many studies showed that the pericardial adipose tissue measurement could be used as a quantitative indicator of metabolic disorders and systemic atherosclerosis and also as a key for CAD risk stratification. Inflammatory mediators that originate in vascular and extravascular tissues promote coronary lesion formation. Adipose tissue may function as an endocrine organ that contributes to an inflammatory burden in patients at risk of cardiovascular complications. Previous studies examined the expression of inflammatory mediators in epicardial and subcutaneous adipose stores in patients with critical CAD where samples were taken at the outset of elective coronary bypass surgery, showed higher levels of chemokine (Monocyte Chemotactic Protein [MCP]-1) and inflammatory cytokines (Interleukin [IL]-1beta, IL-6, and Tumor Necrosis Factor [TNF]-alpha) and protein were observed in epicardial adipose stores. These proinflammatory properties of epicardial adipose tissue were noted irrespective of clinical variables (diabetes, body mass index, and chronic use of statins or ACE inhibitors/angiotensin II receptor blockers) or plasma concentrations of circulating biomarkers [1-3].

Our case report supports this association as our obese patient with recurrent chest pain who developed significant LAD artery disease over 2 years period and she underwent coronary angioplasty and stents implantation, and after investigations was found to have very thick pericardial fat as shown on the chest x ray, echocardiogram and chest CT scan.

Other ways to measure pericardial and visceral fat include; waist-hip ratio, BMI, Dexa scan of visceral fat, and MRI of visceral fat need to be compared in a large study to determine the best modality for CAD risk stratification in obese patients [6].

Conclusion

Epicardial adipose tissue is a source of several inflammatory mediators in high-risk cardiac patients. We believe that the thickness of this layer could correlate with the coronary artery disease and can be used for risk stratification.

References

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