

Dilated Psoriatic Coronopathy: A Novel Association

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Abstract Coronary artery ectasia (CAE) is defined by the Coronary Artery Surgery Study (CASS) registry as the aneurysmal dilatation 1.5 times the diameter of a coronary artery compared to the adjacent normal coronary artery. CAE is reported with a prevalence of 1.2% - 4.9%. Most CAEs are attributed to atherosclerosis or post-percutaneous coronary intervention (PCI) vessel injury. Vasculitides and infection are uncommon etiologies. A review of 59,423 patients from the Danish registry demonstrated a 3-fold increase in the prevalence of abdominal aortic aneurysms in patients with concomitant severe psoriasis. We present a case of a 64-year-old male with severe plaque psoriasis complaining of substernal chest pain whose coronary angiography demonstrated CAE of the left anterior descending and circumflex arteries. Due to its pro-inflammatory state, psoriasis is associated with various systemic manifestations including cardiac and vascular complications. With possibly a similar underlying pathophysiological mechanism, we describe to the best of our knowledge the first case of CAE in a patient with severe psoriasis.

Keywords: coronary artery ectasia, psoriasis, inflammation, atherosclerosis, abdominal aneurysms

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1. Introduction

Defined by the CASS registry as the aneurysmal dilatation of the coronary artery 1.5 times the diameter when compared to an adjacent normal artery, coronary artery ectasia (CAE) is prevalent in 1.2% - 4.9% [1] individuals and has a male to female prevalence of 3:1 [2,3]. Very commonly, the terms coronary artery aneurysm and coronary artery ectasia are used interchangeably. The difference in terminology depends upon the length of the vessel affected. CAE is used to describe diffuse dilatation (>1.5 times the normal diameter) of the coronary arteries that involve 50% or more of the length of the vessel. Coronary artery aneurysm is defined as a localized dilatation that exceeds the diameter of normal adjacent coronary artery segments, or the diameter of the patient's largest coronary vessel by 1.5 times. Herein, coronary artery involvement is less than 50% of the total length of the vessel [4,5]. CAE and coronary aneurysm are frequently detected incidentally during coronary angiography or computed tomography. CAE may occur due to a variety of underlying etiologies. Atherosclerosis accounts for more than 50% of all patients

with CAE [6]. Less commonly encountered etiologies include Kawasaki disease and other congenital causes (17%), mycotic and infectious septic embolism - including syphilis and borreliosis (11%), connective tissue disease (<10%), arteritis (e.g., polyarteritis nodosa, Takayasu's disease, systemic lupus erythematosus) and iatrogenic causes (coronary interventions such as angioplasty, stent placement or even directional coronary atherectomy) [7]. A population based cohort study of 34,301 patients with psoriasis demonstrated an increase in incidence of aneurysms of the abdominal aorta (adjusted hazard ratio [HR] 1.80; 95% confidence interval 1.25-2.61) [8]. There have been however no reported cases associating psoriasis with CAE. We hypothesize that a similar pathological process may lead to CAE in a patient with psoriasis and report to the best of our knowledge, the first such case.

2. Case Description

A 64-year-old Caucasian male presented with 3 hours of severe, substernal, squeezing, non-radiating, non-pleuritic chest pain with associated shortness of breath and diaphoresis. His past medical history was significant for coronary artery disease with a prior inferior

myocardial infarction, chronic total occlusion (CTO) of the right coronary artery (RCA), hypertension, type 2 diabetes mellitus, hyperlipidemia, obesity, hypothyroidism and a 20-year history of plaque psoriasis. His medications included aspirin 81 mg, prasugrel 10 mg, atorvastatin 40 mg, metoprolol succinate 50 mg and metformin 1000 mg BID. He had only received regular psoralen ultraviolet A (PUVA) phototherapy along with topical low potency steroid creams for psoriasis. He was a lifetime non-smoker and he denied any family history of premature coronary artery disease.

On initial assessment, his vital signs revealed a temperature of 97.3°F, blood pressure was 124/85 mm of Hg and heart rate, 80 bpm. Physical exam revealed a middle-aged male in respiratory distress saturating at 90% on ambient air. On examination of the cardiovascular system, jugular venous distention at 10 cms above the sternal angle, bibasilar crackles, and S3 were present. No pedal edema was present. The skin was warm, diaphoretic with psoriatic plaques covering both the flexor and extensor surfaces of the upper and lower extremities, the abdomen including the periumbilical region, upper back and nape with relative sparing of the chest and lower back. The PASI score was 32.4 points. On closer inspection, pitted nails with transverse ridges but no onycholysis or dactylitis were seen.

The electrocardiogram (ECG) revealed sinus rhythm, a heart rate of 75 bpm, Q waves in II, III and AVF and nonspecific ST-T changes which were also noted on his prior ECGs. His blood tests were significant for an initial troponin I of 0.30 ng/mL (ref. <0.15 ng/mL), low density lipoprotein level of 180 mg/dL and a triglyceride level of 480 mg/dL. The patient was given 324 mg of aspirin, 600 mg of clopidogrel and was started on an intravenous heparin infusion for non-ST elevation myocardial infarction. Cardiac catheterization was performed from the right radial artery using the Judkins Right (JR) 4 and Judkins Left (JL) 3.5 catheters which revealed triple vessel disease. CTO of the RCA with collaterals from the left coronary circulation was re-visualized. A 100% occlusion of the 1st diagonal branch of the left anterior descending (LAD) artery was identified as the culprit vessel for his acute coronary syndrome. Interestingly, both the proximal LAD and proximal left circumflex arteries were noted to be dilated and ectatic consistent with aneurysmal dilatations [Figure 1 and Figure 2]. Given these findings, intervention at the time was deemed high risk and coronary artery bypass graft surgery was planned. His troponin levels peaked at 44 ng/mL after 48 hours of hospitalization and trended downward in the coming days. He remained chest pain free and was discharged with a plan for coronary artery bypass graft surgery in 2 weeks.

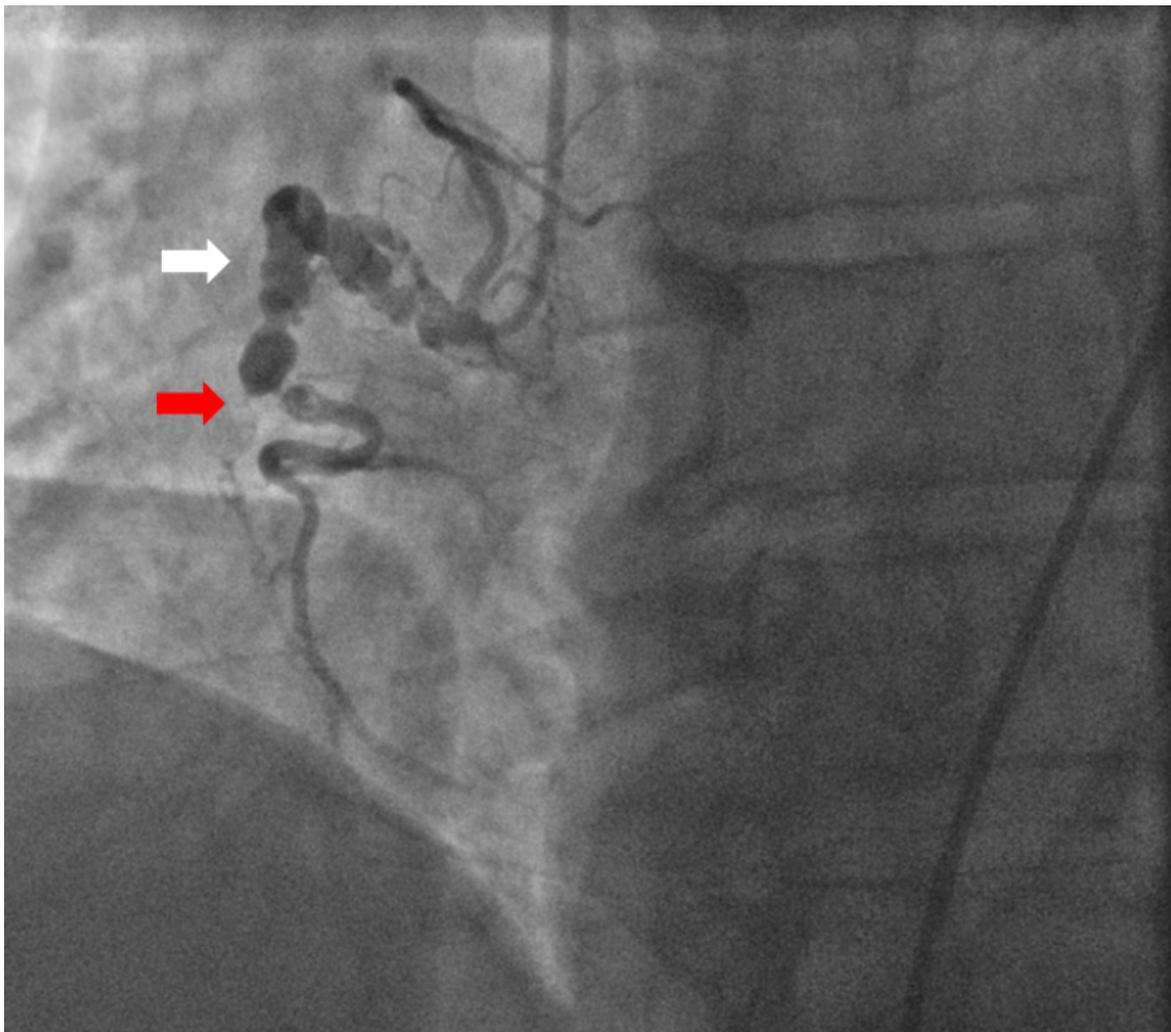


Figure 1. Coronary angiogram of right coronary artery showing large vessel with multiple aneurysmatic dilation in the proximal segment (red arrow) and chronic total occlusion of the middle right coronary artery (white arrow)

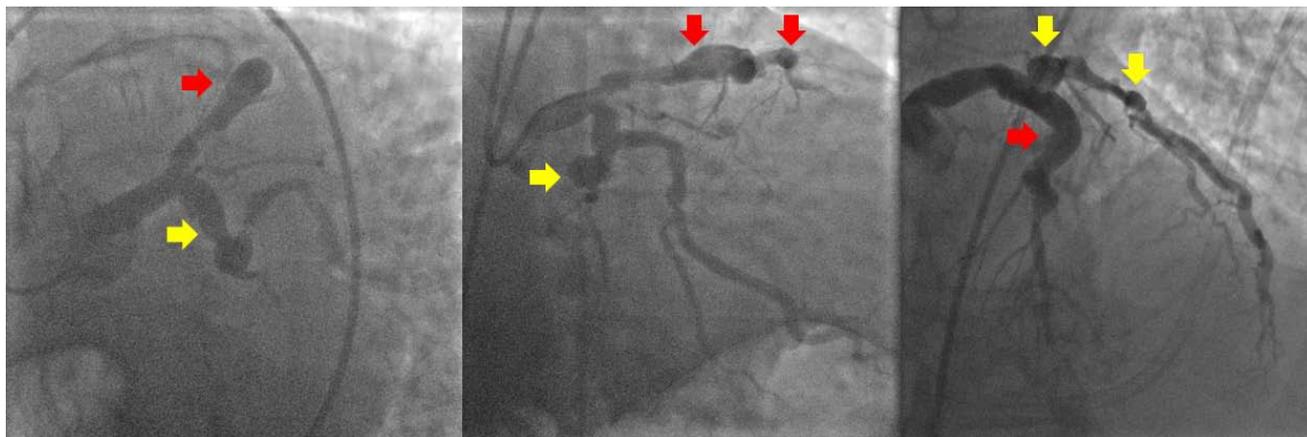


Figure 2. Left coronary system in left anterior oblique (LAO) caudal (left), LAO cranial (middle) and right anterior oblique (RAO) caudal views (right). Red arrows indicate aneurysmal dilation of the proximal left anterior descending artery with 70% tubular stenosis and yellow arrow indicates aneurysmatic dilation of proximal circumflex with 70% stenosis

3. Discussion

Psoriasis is a chronic, complex autoimmune disease characterized by erythematous, scaly patches over the extensor aspect of skin and is associated with joint involvement in about one-third of patients [9]. It affects nearly 3.2% of the American population [10]. It is considered a systemic condition similar to other common autoimmune conditions such as lupus and rheumatoid arthritis. There is increasing evidence that patients with psoriasis have a higher incidence of disorders such as hypertension, diabetes, obesity, and dyslipidemia, thus increasing the risk of cardiovascular disease [11,12,13,14]. Adverse cardiac outcomes including thrombus formation with embolization leading to myocardial infarction have been well documented in literature. In a case-control observational study conducted to determine the association between psoriasis and vascular disease, Prodanovich *et al* found that patients with psoriasis were significantly more likely to have ischemic heart disease (odds ratio [OR], 1.78; 95% confidence interval [CI], 1.51-2.11), cerebrovascular (OR, 1.70; 95% CI, 1.33-2.17) and peripheral vascular (OR, 1.98; 95% CI, 1.32-2.82) diseases when compared with controls. Psoriasis was also found to be an independent risk factor for mortality (OR, 1.86; 95% CI, 1.56-2.21) [11]. This relationship is of particular importance as there is a clear link between atherosclerosis and aneurysmal dilations of the arterial system. In a Danish cohort, Khalid *et al* reported that people with mild psoriasis had a 1.2 times higher incidence of abdominal aortic aneurysm when compared to those without psoriasis (95% confidence interval 1.03 to 1.39). The risk was significantly (67%) higher in those with severe psoriasis [15].

An important risk factor for both abdominal aortic aneurysms and coronary artery ectasia is atherosclerosis. Previous literature has also supported a link between the pathogenic mechanism of psoriasis and atherosclerosis. Ghazizadeh *et al* highlighted that both psoriasis and atherosclerosis share inflammatory mechanisms, such as T-helper 1 (Th1) cell-mediated pathways, T-cell activation and expression of adhesion molecules [16]. Psoriasis involves Th1/Th17 pathways and in turn, Th17 has an important role in several cardiovascular diseases. Vascular cell adhesion molecule -1 (VCAM-1), intercellular cell

adhesion molecule-1 (ICAM-1), and L-selectin also play a crucial role in atherosclerosis and plaque instability, and similarly ICAM-1 and VCAM-1 are upregulated in psoriasis [16]. Interleukin-17 (IL-17), Monocyte Chemoattractant Protein-1 (MCP-1) and tumor necrosis factor- α (TNF- α) inflammatory markers in psoriasis promote vascular inflammation and thrombosis as well [17]. The observed association of psoriasis, obesity and hyperlipidemia is further supported by the demonstration that low density lipoprotein (LDL) particles infiltrate the blood vessel walls leading to oxidation in the tissue macrophages which in turn results in the typical macrophage foam cells of atherosclerosis [17].

As the process advances, monocytes adhere to the activated endothelium and are activated via pattern recognition receptors favoring the production of pro-inflammatory cytokines (IL-6) and chemokines that further contribute to the tissue damage [17]. The interplay of inflammatory markers that participate in the pathogenesis of psoriasis and atherosclerosis, ultimately drives the production of acute phase reactants such as C-reactive protein, marker of cardiovascular disease and inflammation [17]. Furthermore, psoriasis and atherosclerosis share features of infiltrating T-cells, monocytes/macrophages, neutrophils, dendritic cells, and mast cells. Both processes are predominantly characterized by Th1 type cytokines (IFN γ , IL-2, and TNF α) [16]. Angiogenesis and upregulation of vascular endothelial growth factor (VEGF) can also be seen in both psoriasis and atherosclerosis [16].

In adults, most CAEs are attributed to atherosclerosis or vessel injury after coronary intervention. Vasculitides and infections make up the majority of the other cases. Our patient was incidentally found to have CAE on coronary angiography. In addition to diabetes and hypertension, he also suffered from severe, widespread psoriasis. Psoriasis unlike other spondyloarthropathies is a rare cause of vasculitis (associated with mutations of the IL12B locus) with only 5 documented cases of aortitis [18]. We hypothesize a similar pathophysiology as aortitis involving infiltration of lymphocytes, plasma cells and macrophages combined with development of fibrotic tissue rich in type I collagen and fibroblasts of the coronary arteries as the cause of CAE. We surmise that there must be an association between psoriasis and CAEs, similar to that of psoriasis and abdominal aortic

aneurysms, a consequence of its pro-inflammatory state [19]. We performed a thorough search of the PubMed/Medline and Google scholar medical databases to determine the relationship of psoriasis with CAEs. No citations were found after limiting the search to English language, humans and case reports. To the best of our knowledge, this is the first case to describe psoriasis as a potential etiology for CAE.

4. Conclusion

Psoriasis, a common skin condition should be considered an atherosclerotic risk factor along with diabetes, hypertension, smoking and obesity. Due to its pro-inflammatory state, there may be various systemic manifestations including cardiac and vascular complications. While there are more common contributing factors to CAE, we postulate that patients with psoriasis are also at increased risk.

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