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CASE REPORT

Coronary Artery Dissection and Myocarditis Caused by Eosinophilic Granulomatosis with Polyangiitis (EGPA): A Case Report

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Abstract

Eosinophilic granulomatosis with polyangiitis (EGPA) also referred to as Churg-Strauss syndrome is a rare vasculitis of the small to medium vessels. We present a rare case of acute coronary artery dissection brought on by EGPA, which generally has a poor prognosis. A 41-year-old male with history of bronchial asthma presented to the emergency room with a 2-week history of dyspnea, cough with clear phlegm, and fever. For the past eight months he had experienced episodes with similar symptoms relieved by steroids. CT chest showed bilateral upper lobe patchy opacities with extensive workup for infectious etiology being negative. He had peripheral eosinophilia with sinusitis. He had acute coronary syndrome and Coronary angiogram showed Right coronary artery dissection. After making a diagnosis of EGPA based on American college of Rheumatology criteria, he was successfully treated with high dose immunosuppression. Coronary artery dissection is a fatal and uncommon complication of EGPA which is usually diagnosed postmortem. Early recognition of this condition ante mortem and aggressive treatment can be lifesaving as demonstrated in our case.

Keywords: Eosinophilic granulomatosis with polyangiitis, EGPA, Churg-Strauss syndrome, Antineutrophil cytoplasmic antibody, ANCA, Vasculitis, Coronary artery vasculitis, Glucocorticoids/therapeutic use, Immunosuppressive agents / therapeutic use, Granulomatosis with polyangiitis / diagnosis, Granulomatosis with polyangiitis / drug therapy, Humans

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1. Introduction

Eosinophilic granulomatosis with polyangiitis (EGPA) also known as Churg-Strauss syndrome is an uncommon small to medium vessel antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis and multisystem disorder with hypereosinophilia-related target end organ damage.[1-3] Clinicopathologically there are two different subtypes of EGPA, ANCA-positive in 40–60% of cases, are associated with vasculitis; ANCA-negative subsets are associated with eosinophil-related organ injury.[3,4] The presenting features of EGPA can be divided into three phases.[3,4] The prodromal phase is characterized by adult-onset asthma with or without rhinosinusitis. The second or eosinophilic phase is associated with marked peripheral blood eosinophilia. The third or vasculitis phase develops after 3–8 years and involves organ systems such as lungs, skin, gastrointestinal tract, heart and blood vessels, kidneys, and peripheral nerves. Cardiac involvement occurs in about 17–29% of cases.[3] Coronary artery dissection is a rare catastrophic complication, which is usually discovered postmortem.[5] Here we present a rare case of myocarditis and coronary artery dissection due to EGPA successfully treated with immunosuppressive therapy.

2. Case presentation

A 41-year-old male with no significant past medical or family history, with occasional cigar and alcohol use, presented to the emergency room with a 2-week history of dyspnea, cough with clear phlegm, and fever. He endorsed having chronic joint pain and body aches. He had normoxemia and was hemodynamically stable with nonspecific T-wave abnormalities on his electrocardiogram. Splinter hemorrhage was noted with the rest of the clinical examination being normal. For the past eight months he had similar symptoms and was treated with oral and inhaled steroids, along with bronchodilators resulting in intermittent relief, followed by a recurrence when steroids were discontinued. He had an outpatient spirometry revealing moderate obstructive airway disease with a significant bronchodilator response. Outpatient computed tomography (CT) scan of thorax demonstrated bilateral ground glass opacities suspicious for infectious/inflammatory opacities. His early hospital workup was significant for leukocytosis with a white cell count of 18,000, absolute eosinophil count of 10.1, troponin of 64,000 and BNP of 4000 (Table 1).

CT thorax showed bilateral upper lobe patchy opacities with an extensive respiratory and infectious disease workup was negative. He was treated for non-ST elevation myocardial infarction (NSTEMI) with dual antiplatelet therapy and therapeutic doses of low molecular weight heparin (LMWH). Troponin trended upwards to 84,000 but he had a normal transthoracic echocardiogram with no regional wall motion abnormalities. There was a strong clinical suspicion of viral myocarditis. Coronary catheterization study revealed coronary artery dissection of the mid right coronary artery (Fig. 1). There was significant stenosis in all three coronary arteries, nevertheless LMWH was discontinued due to the risk of expansion of intramural hematoma in the RCA. Aspirin and Ticagrelor were continued.

Differential diagnosis included occult systemic infectious disease, systemic connective disease, and hematological malignancy. Rheumatology serological work up and Infectious disease work up was negative. Serum protein electrophoresis ruled out the presence of monoclonal proteins. Peripheral smear showed marked eosinophilia with no increase in blast cells or morphologic dysplasia. CT head showed pansinusitis. After ruling out many of the aforementioned differential diagnosis, Eosinophilic granulomatous polyangiitis (EGPA) or neoplastic hypereosinophilic syndrome (HES) was considered likely.

Cardiac MRI revealed large, scattered areas of left ventricular subendocardial enhancing scars (late gadolinium enhancement) with myocardial hypoperfusion and smaller areas of necrosis. No large thrombus was identified. Diffuse edema of the myocardium was noted with Septal extracellular volume abnormally elevated at 32%.There was reduced systolic function with Left ventricular ejection fraction (LVEF) of 42% and borderline thickened left ventricular myocardium. The constellation of MRI findings was highly suggestive of eosinophilic myocarditis. Bone marrow biopsy demonstrated normocellular marrow (50% cellularity) with trilineage hematopoiesis, expanded eosinophils (41% of marrow cellularity) with no increase in blasts. The FISH panel for common diagnostic/prognostic chromosomal abnormalities associated with eosinophilia and chromosome karyotype was normal (negative for rearrangement of PDGFRA, PDGFRB, FGFR1 or BCR/ABL1). Bronchoscopy revealed normal endobronchial anatomy, endobronchial mucosa, and no evidence of diffuse alveolar hemorrhage (DAH) or eosinophilia.

A diagnosis of Eosinophilic myocarditis secondary to EGPA satisfying American College of Rheumatology (ACR) criteria was made based on elevated eosinophilic count, asthma of 8 months duration, chronic sinusitis, nonspecific migratory pulmonary

Table 1. Laboratory results.

Basic labs	Results	Reference Range	Serology	Results	Reference Range
WBC (K/mcl)	18.6	4.2–11	ANA screen with antibody, IFA reflex	Not detected	
HGB (g/dl)	14.6	13–17	ANCA	Not detected	<1:20
Platelet (K/mcl)	226	140–450	Myeloperoxidase antibody	0.2	<1
Absolute eosinophil count (U/L)	10.1	0–0.5	ACE	21	16–85
ESR (mm/hr)	43	0–20	QuantiFERON Tb Plus	Negative	
CRP (mg/dl)	7.5	<0.3	Hepatitis B surface antigen	Negative	
CPK (units/L)	497	39–308	HIV Viral load (Log)	Non detected	
NT Pro BNP (pg/ml)	4000	≤125	Strongyloides antibody	Negative	<0.6
Troponin high sensitivity I (ng/L)	84,000	<77	Toxoplasma antibody IgG (U/ml)	<0.50	<0.6
Creatinine (mg/dl)	1.18	0.6–1.17	Aspergillus antibody (mcg)	<90	<90
GFR (ml/min)	>90	>60	*Aspergillus fumigatus* IGE allergen (Ku/l)	<0.35	<0.5
AST (U/L)	59	<37	Blastomyces antigen EIA	<1:2	<1:2
ALT (U/L)	31	<64	Coccidioides antibody	Negative	
ALP (U/L)	102	45–117	Serum protein electrophoresis	Alpha 1–0.6 alpha 2–1.1 Kappa/lambda ration-1.5 No monoclonal proteins	Alpha 1–0.2 alpha 2–0.5 kappa/lambda ratio 0.2–1.6
Total bilirubin (mg/dl)	1.18	0.2–1.0	FISH hypereosinophilia panel	Negative	
Anion gap (mmol/L)	19	7–19	IGG (mg/dl)	1040	700–1600
			IGE (IU/ml)	783.3	<99
			Peripheral smear	Moderate leukocytosis with marked eosinophilia with no blasts/dysplasia	

White Blood Cell (WBC), Antinuclear Antibody (ANA), Hemoglobin (HGB), Antineutrophil cytoplasmic antibodies (ANCA), Angiotensin Converting Enzyme (ACE), Erythrocyte Sedimentation Rate (ESR), C-Reactive Protein (CRP), Creatine Phosphokinase (CPK), Human Immunodeficiency Virus 1 (HIV 1), N-terminal-pro hormone BNP (NT ProBNP), Glomerular Filtration Rate (GFR), Immunoglobin E (IGE), Immunoglobin G (IGG), Aspartate Aminotransferase (AST), Enzyme Immunoassay (EIA), Alanine transaminase (ALT), Alkaline phosphatase (ALP), Fluorescence in situ hybridization (FISH).

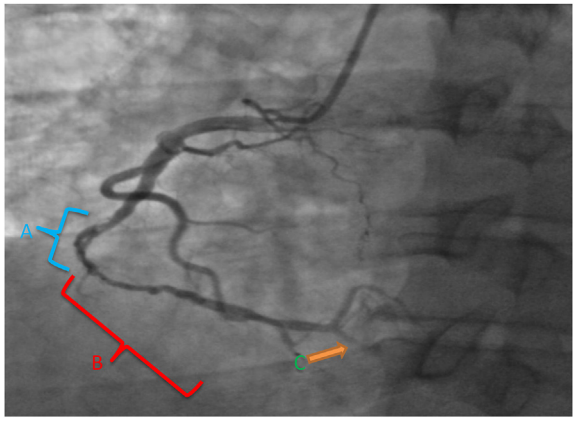

Fig. 1. A) Flow separation due to non-flow limiting dissection of mid to distal right coronary artery B) Long segment of diffuse disease involving distal vessel C) Partly occluded posterior descending artery.

infiltrates on CT imaging, eosinophilic myocarditis type findings on cardiac MRI, and splinter hemorrhages on exam. The bone marrow biopsy/unremarkable FISH results made neoplastic hypereosinophilic syndrome unlikely.[6] The presence of coronary artery dissection in the RCA, and myocarditis in our patient led to administration of pulse doses of Methylprednisone, and systemic Cyclophosphamide followed by tapering oral prednisone therapy. He was noted to be doing well about a year later in the outpatient when last seen.

3. Discussion

The ACR criteria are the preferred method of diagnosis for EGPA and have a sensitivity and specificity of 85% and 99.7%, respectively.[2] In EGPA

Table 2. Case listing from literature search for EGPA and associated coronary arteritis or coronary artery dissection.

SN	Author/Country	Year	Age	Sex	Asthma/ Rhinosinusitis	Peripheral Blood Eosinophilia	EGPA or HES	Coronary Arteritis	Coronary artery Dissection	Treatment received	Outcome
1.	Lin et al./China[5]	2022	45	F	Yes	Yes	EGPA	Unknown or not reported	Yes	Steroid pulse therapy + Rituximab followed by tapering steroid and cyclosporine	Alive at 20 month follow up
2.	Sato et al./Japan[16]	2020	33	M	Yes	Yes	EGPA	Coronary Aneurysm	No	Steroid 1 mg/kg + Cyclophosphamide	Alive at discharge
3.	Karthikeyan et al./USA[17]	2019	Middle aged	M	No	Yes	EGPA	Noninfectious vegetations involving aortic valve, mitral valve, and chorda tendineae	No	Valve replacement and 1 mg/kg steroid followed by tapering dose of steroid	Alive at 10-month followup and then died of cardiogenic shock
4.	Chai et al./UK[18]	2018	42	M	Yes	Yes	EGPA	Widespread coronary lesion	No	Steroid pulse therapy + Cyclophosphamide every 3 weeks for cycle followed by tapering dose of steroid and azathioprine/ mycophenolate	Alive at 1-year followup
5.	Matsuda et al./Japan[19]	2017	48	F	Yes	Yes	EGPA	Suspected coronary vasculitis	Unknown or not reported	Steroid and cyclophosphamide followed by tapering dose of steroid and azathioprine	Alive on discharge
6.	Schiefermueller et al./UK[20]	2017	45	F	Yes	Yes	EGPA	Suspected coronary vasculitis	No	Pulse dose steroid followed by 1 mg/kg steroid	Alive at 1-year followup
7.	Correia et al./ Portugal[21]	2013	22	F	Yes	Yes	EGPA	Diffuse coronary vasculitis	No	Pulse dose steroid followed by 1 mg/kg steroid + cyclophosphamide followed by tapering dose of steroid	Alive at 1-year followup
8.	Riksen et al./ Netherland[22]	2010	18	M	Yes	Yes	EGPA	Multiple coronary aneurysms and stenotic lesions	No	Pulse dose steroid followed by 1 mg/kg steroid + cyclophosphamide followed by tapering dose of steroid	Alive at 1-year followup
9.	Tuzov et al./Israel[23]	2012	Young	F	Unknown	Unknown	Unknown	eosinophilic inflammation of Left coronary artery with thrombosis	No	None	Death
10.	Puri et al./Australia[24]	2009	72	F	No	Yes	HES	multiple coronary aneurysms	No	Imatinib followed by aspirin and clopidogrel	Alive at 6 weeks followup
11.	Lepper et al./Germany[25]	2005	45	F	No	No	No	No	Yes	None	Death

(*continued on next page*)

Table 2. (continued)

SN	Author/Country	Year	Age	Sex	Asthma/ Rhinosinusitis	Peripheral Blood Eosinophilia	EGPA or HES	Coronary Arteritis	Coronary artery Dissection	Treatment received	Outcome
12.	Taira et al./Japan[26]	2005	52	F	Yes	Unknown	Unknown	Yes, in autopsy	No	None	Dead on presentation
13.	Hunsaker 3rd et al./USA[27]	1992	57	F	Yes	No	EGPA likely	Yes, in autopsy	No	None	Death
14	Lie et al./USA[28]	1989	39	M	Yes	Unknown	EGPA likely	Yes, in autopsy	No	None	Death
15.	Asatani et al./Japan[29]	2022	38	F	Yes	Yes	EGPA	Yes	No	Pulse dose Steroid followed by 1 mg/kg/day followed by cyclophosphamide	Alive at discharge

the most common organ involved are the lungs, nervous system and ear, nose and throat[3] but the morbidity and mortality associated with EGPA are often due to vasculitis of organ systems other than the lung, such as the heart[7,3]. The exact pathophysiology of organ damage in EGPA has yet to be elucidated. It is thought that inappropriate TH2-mediated immunity causes hyperproliferation and decreased apoptosis of eosinophils, which in turn release fibrogenic cytokines, transforming growth factor β (TGF- β), IL-1α, and IL-1β leading to fibrosis and organ damage.[8]

Cardiac involvement is associated with high degree of mortality and morbidity, accounting for half of the deaths in EGPA.[3] The presence of Ear, nose and throat involvement is protective against cardiac pathology in EGPA.[3] Cardiac involvement is more common in ANCA negative individuals.[3] In EGPA, subclinical cardiac involvement is common, with active cardiac involvement noted in about 16–29% of patients.[3,9] Cardiac involvement occurs more frequently in patients with eosinophilia.[10] Cardiac involvement in EGPA can have protean manifestations, including cardiomyopathy, myocarditis, heart failure syndromes, arrythmia, pericardial involvement including effusion, valvular involvement, endomyocardial fibrosis and intracardiac thrombus.[3] In a retrospective study by Comarmond et al. with a cohort of 383 patients with EGPA, 15% had pericarditis, and 16% had cardiomyopathy.[4] In a cross-sectional analysis by Neumann et al. of 22 patients with EGPA with cardiac involvement, all patients had abnormal ECGs. Additionally, other cardiac complications such as valvular insufficiency (73%), pericardial effusion (50%), and heart failure (41%) were also noted.[10]

Our patient did not get a cardiac biopsy, which is the gold standard for diagnosing Eosinophilic myocarditis.[11] However, Cardiac MRI findings have been found to have high concordance with biopsy findings, although large scale studies are lacking.[11,12] Our patient demonstrated diffuse subendocardial areas of high signal intensity in LGE images, a feature highly suggestive of eosinophilic myocarditis (EM), with the additional findings of extracellular edema and reduced ejection fraction of left ventricle, providing high specificity and also satisfying updated Lake Louis criteria for acute myocarditis.[13] Endocardial biopsy has low sensitivity due to skip areas and it is associated with high-risk complications.[11,12] In the appropriate clinical setting with supporting echocardiographic and coronary angiogram findings, cardiac MRI can not only be helpful for early diagnosis but can also be used for follow up relating to disease progression

and thereby likely reduce the high mortality and morbidity associated with EM.[11-13]

Coronary artery vasculitis (12%), valvular involvement (12%), and coronary artery dissection are very rare presentations.[3] To our knowledge, there is only one known published case of EGPA with an ante mortem diagnosis of coronary artery dissection,[5] and 10 cases of EGPA-associated vasculitis and aneurysm; these cases are outlined in Table 2. In their literature review from 1987 to 2011, Kajihara et al. documented 15 cases of coronary artery dissection associated with eosinophilic coronary periarteritis diagnosed at autopsy.[14] All these patients had died of sudden cardiac death. This unfortunate occurrence explains the severity of the disease and the need for early diagnosis of EGPA to begin prophylactic treatments and prevent complications.[15]

4. Conclusion

Coronary artery dissection is a rare and fatal complication of EGPA. EGPA can mimic other vasculitides and HES, hence a thorough diagnostic approach is needed to make a correct diagnosis. Organ involvement and vasculitis usually occur after several years of diagnosis of asthma but can also happen early in disease. Early treatment can have favorable outcomes in achieving remission. Therefore, clinical vigilance and early detection of EGPA maybe crucial in preventing severe disease and rare complications such as coronary artery dissection. Aggressive immunosuppressive treatment and multi-disciplinary input to achieve remission and limit damage is needed in severe cases. Further large population studies will be needed to understand this disease entity better.

Conflict of interest

The authors have no conflict of interest to declare.

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