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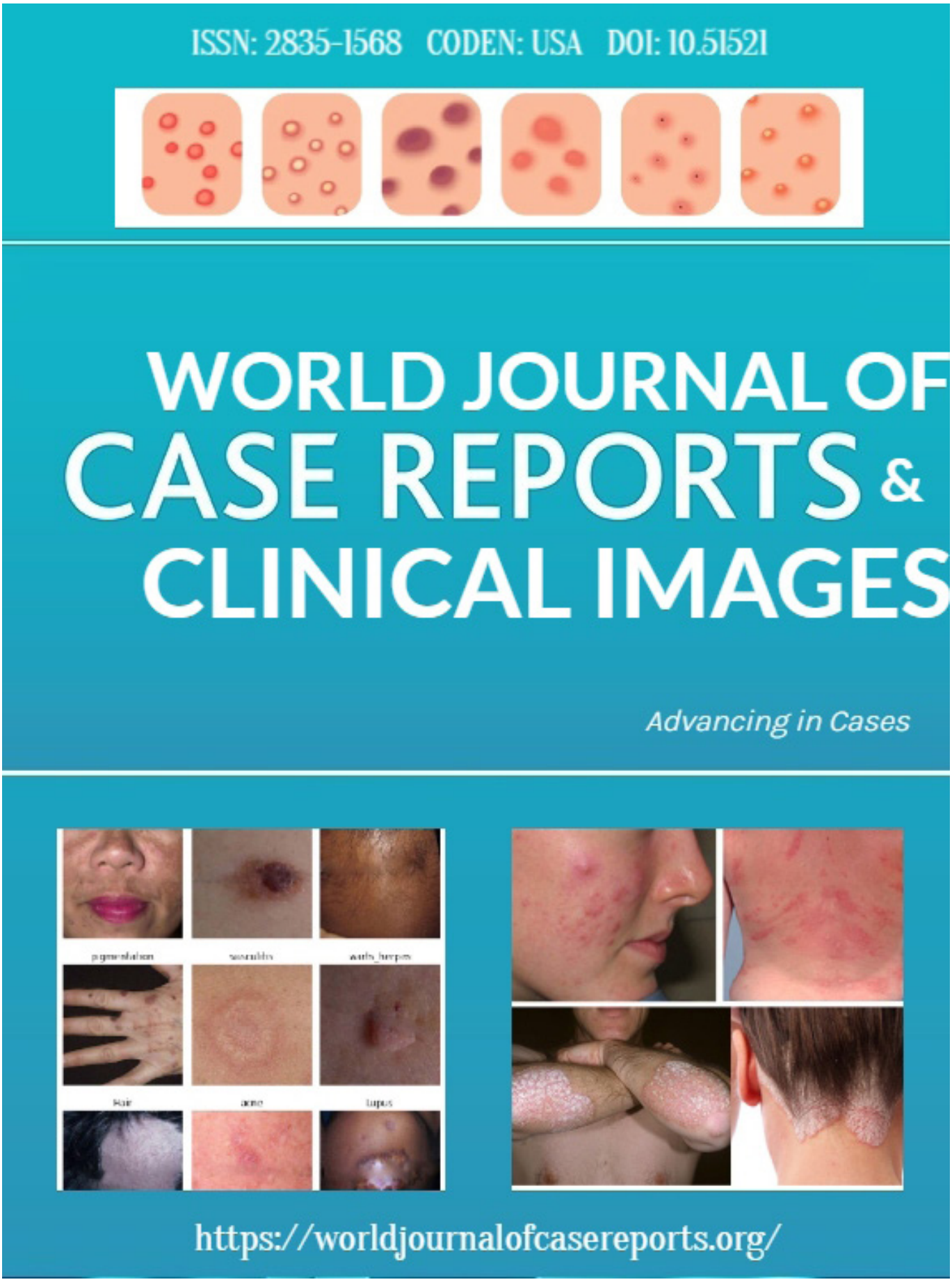

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CASE REPORT

Beyond the STEMI; Unmasking A Coronary Artery Aneurysm

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ABSTRACT

Background

Coronary artery aneurysms (CAAs) are relatively rare defects that range significantly in clinical presentation, most often discovered in adults with concomitant coronary artery disease. There remains no general consensus on the optimal management of CAAs. A vast majority of cases reported in literature are managed operatively or with percutaneous stenting. We however demonstrate a case of a CAA discovered upon angiography for a STEMI managed entirely with intracoronary thrombolytics and intravenous heparin, followed by a repeat coronary angiography that confirmed re-opening of the occluded vessel now with distal coronary blood flow.

Case Summary

In this case we present a 46-year-old male with a past medical history of hypertension (HTN), hyperlipidemia (HLD), type 2 diabetes mellitus (T2DM), and obesity who was brought to the emergency department for crushing, substernal chest pain. Electrocardiogram (EKG) demonstrated ST segment elevations in leads V3-V6 and leads II, III, and AVF. Coronary angiography revealed severe ectasia, with an aneurysm of the middle LAD containing a 100% thrombotic occlusion. A repeat angiography 24-hours later demonstrated diffuse coronary ectasia, with a now patent LAD, and the presence of a focal 10 mm aneurysm of the mid left anterior descending (LAD) artery. There was poor distal flow of the LAD with complete occlusion in the apical region. The patient was not a candidate for percutaneous coronary intervention (PCI) due to the large thrombotic burden, and risk of propagation of the clot, thus the decision was made to manage medically. This case highlights a unique presentation of CAA with a superimposed thrombus, causing a complete total occlusion of the mid LAD, resulting in the initial hospital presentation as an ST-segment elevation myocardial infarction (STEMI).

KEYWORDS:

Coronary Artery Aneurysm (CAA); Coronary Artery Disease (CAD); Coronary Artery Ectasia (CAE)

Abbreviations:

ACS: Acute coronary syndrome
CAA: Coronary artery aneurysm
CAD: Coronary artery disease
CAE: Coronary artery ectasia
DAPT: Dual antiplatelet therapy
EKG: Electrocardiogram
LCX: Left circumflex artery
MACE: Major adverse cardiovascular event
NSTEMI: Non ST-segment elevation myocardial infarction
PCI: Percutaneous coronary intervention
PDA: Posterior descending artery
RCA: Right Coronary Artery
STEMI: ST-segment elevation myocardial infarction
Transthoracic echocardiogram (TTE)

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Introduction

Coronary artery aneurysms are rare defects of the coronary arteries seen at a relatively low incidence of 0.3-4.9%, and have an estimated 5-year survival rate of only 71%. This phenomenon occurs when a localized portion of a coronary artery is non-physiologically dilated. Clinical symptoms vary vastly ranging from an asymptomatic presentation or incidental discovery, to presenting as a major cardiovascular event such a non-ST segment elevation myocardial infarction (NSTEMI), an ST segment elevation myocardial infarction (STEMI), or more catastrophically as an episode of sudden cardiac death (SCD). Differential diagnoses of a CAA could include an ecstatic segment or a pseudoaneurysm that mimics the appearance of a true aneurysm. Here we present the case of a STEMI with a true CAA discovered during urgent coronary angiography performed for revascularization. We perform a concise review of the multiple modalities often employed in the treatment of CAAs, focusing primarily on the medical management technique utilized in this case; through the use of intracoronary thrombolytics.

Case Presentation

A 46-year-old Asian male with a past medical history of hypertension, hyperlipidemia, Type 2 Diabetes Mellitus, and obesity presented to the emergency department as a STEMI pre-notification for progressively worsening, crushing, substernal chest pain for a 2-hour duration. A preliminary EKG performed by emergency medical services demonstrated ST segment elevations in leads V3-V6 & leads II, III, AVF (Figure 1). The patient was diagnosed with inferior-lateral STEMI, loaded with Aspirin 325 mg, Brilinta 180 mg, and Atorvastatin 80 mg, and planned for emergent coronary angiography. Initial Troponin level was 27.3 ng/L. Coronary angiography revealed severe diffuse ectasia and a focal aneurysm of the middle LAD with a superimposed 100% thrombotic occlusion (Figures 2 & 3). No collaterals to the distal segment were present.

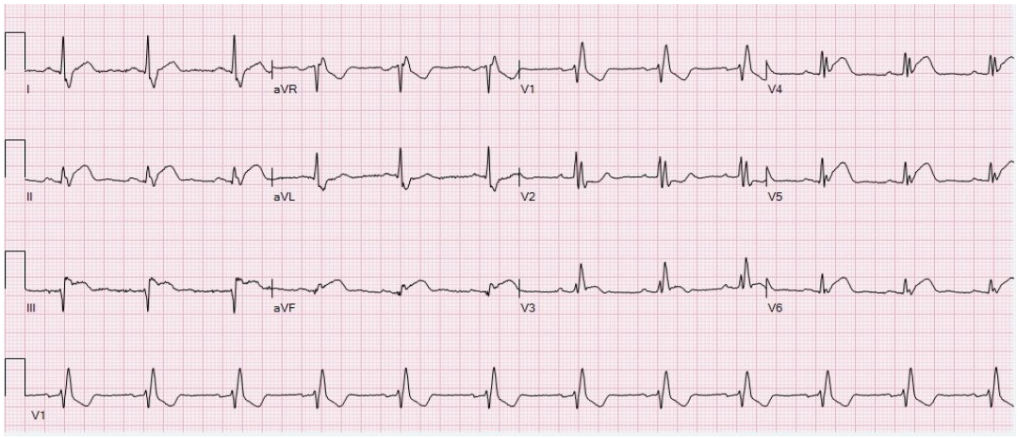

Figure 1 This EKG demonstrates ST-elevations in leads V3-V6 & leads II, III, AVF and reciprocal ST depressions in leads I & AVL.

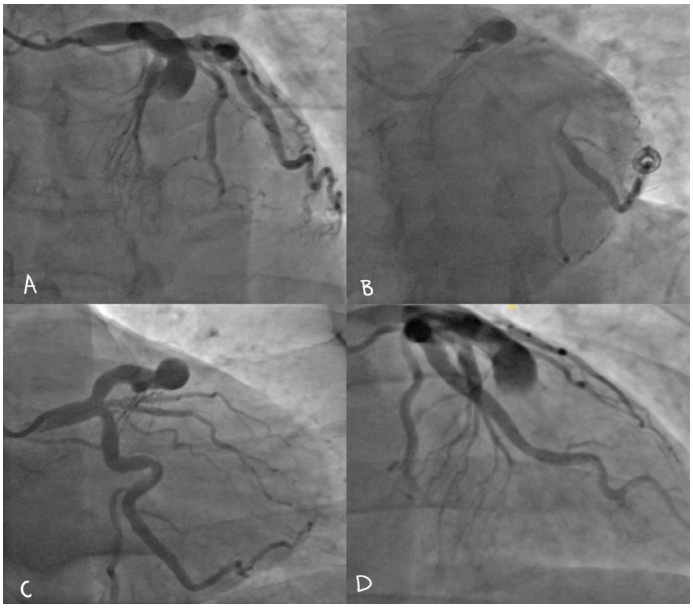

Figure 2 This image depicts images obtained from the first angiography performed. (A) LAO cranial view, B) LAO caudal view, C) LAO caudal view, D) LAO cranial view. All images depict an aneurysm measuring approximately 10 mm in diameter, located in the mid segment of the LAD trapping contrast, and no distal perfusion. The left main appears normal, while the left circumflex artery appears tortuous.

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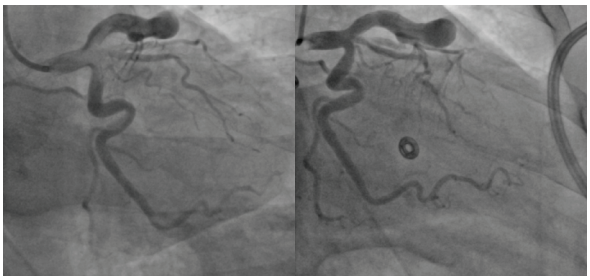

Figure 3 These images depict an RAO caudal view, depicting the large aneurysmal segment in the mLAD.

The patient was not eligible for PCI or thrombotic aspiration due to the large thrombotic burden and risk of clot propagation or stroke. The decision was made to proceed with intracoronary Eptifibatide and heparin boluses, followed by twelve hours of intravenous Eptifibatide and intravenous heparin infusions. A repeat coronary angiography one day later demonstrated diffuse coronary ectasia, with a now patent LAD, confirming the presence of a focal 10 mm aneurysm of the mid LAD, and poor distal flow with complete occlusion in the apical region (Figures 4, 5 & 6). A trans-thoracic echocardiogram (TTE) showed an estimated left-ventricular ejection fraction (LVEF) of 55-60% with Grade 1 Diastolic Dysfunction, and mild aortic insufficiency.

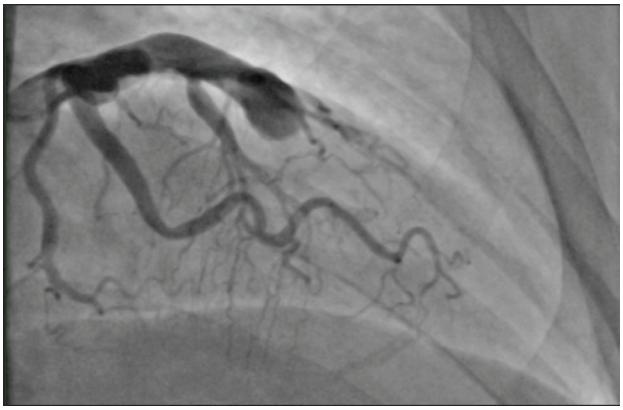

Figure 4 This image shows an RAO cranial view, depicting trapping of contrast in the aneurysmal sac.

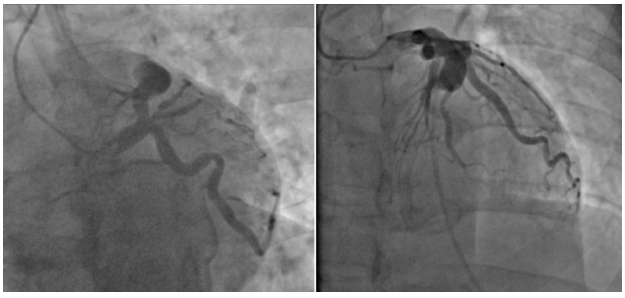

Figure 5 This image depicts an LAO caudal (left) & LAO cranial (right) view of the second angiography performed following thrombolytic administration. Contrast is visualized filling the aneurysmal segment, however now with distal flow present.

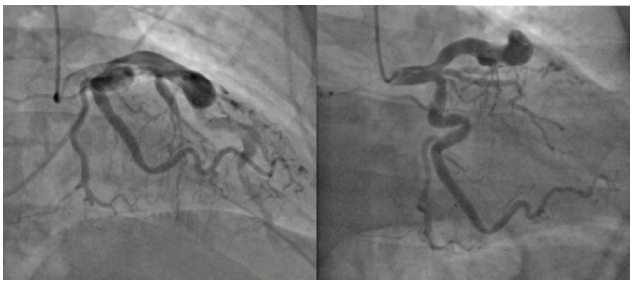

Figure 6 This image depicts an RAO cranial (left) & RAO caudal (right) view of the second angiography performed. Similarly, distal flow is now present.

The patient remained vitally stable and denied any repeat episodes of chest pain or discomfort. Repeated blood work did not demonstrate a further uptrend in his troponin levels, nor were there any new EKG changes depicting ongoing cardiac ischemia. The patient was discharged with Aspirin 81 mg oral once daily, Brillinta 90 mg oral twice daily, Atorvastatin 80 mg oral once daily, and Metoprolol Succinate 50 mg oral once daily. He was also instructed to make appropriate dietary and activity changes, and was referred for outpatient follow-up with cardiology for further treatment, and monitoring of his recovery. The patient was also referred to an outpatient cardiac rehabilitation program.

Discussion

CAAs are an uncommonly encountered phenomenon defined as the abnormal dilatation of a coronary artery exceeding 1.5 times the original vessel diameter [1], involving a localized segment, or diffuse portion of all three layers of the arterial wall [4,5]. Aneurysms can be saccular, involving a localized out-pouching of the vessel, or fusiform, which involves a diffuse widening of a coronary segment [2,18]. This can be further subdivided into the category of a 'giant' CAA, defined as a dilatation of more than 4 times the normal vessel size [4]. Coronary artery ectasia (CAE), considered a variant of coronary artery disease (CAD), is differentiated from an isolated aneurysmal lesion due to its segment exceeding 1/3rd of the total coronary artery length [41,42].

CAAs are most commonly isolated to a single major coronary vessel, with the highest incidence known to occur in the right coronary artery (RCA), less frequently in the LAD, followed by the left circumflex artery (LCX) [3,8]. CAAs of the left main-stem coronary artery are of an even greater rarity, seen in a minute 0.1% of the population [4]. CAAs discovered on angiography are at a relatively low incidence of 0.3-4.9%, with a higher preference in the male population [3,11,40]. CAAs are generally less commonly encountered in Asia when compared to North America or Europe [40]. The 5-year survival rate is estimated to be approximately 71%, which places emphasis on the necessity of adequate monitoring post-diagnosis and treatment [39].

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Up to 33% of all cases of CAAs are associated with underlying CAD [6]. Though the true pathogenic mechanism remains unknown, aneurysm formation due to underlying atherosclerosis is thought to be the most common cause in adults as it is observed in up to 90% of discovered CAA cases. In these instances, the aneurysm is usually isolated to a single coronary vessel, however can involve multiple focal areas due to the diffuse nature of coronary atherosclerosis [6]. Atherosclerotic plaque buildup leads to a localized inflammatory state within the vessel, causing subsequent arterial weakening and potential for wall degeneration [2,40].

Complications from Kawasaki's disease, an acute inflammatory syndrome that results in a systemic vasculitis, is the prevailing cause of CAA formation in adolescents, or a delayed presentation later in adult life [4,7,10]. Presentations in Kawasaki's disease can range anywhere from coronary ectasia, to giant CAA development, with a hypothesis that later treatment time directly correlates with a larger aneurysmal size [43,44]. Pathogenesis of CAA formation in this instance remains poorly understood, but is thought to be a consequence of increased systemic levels of matrix metalloproteinase (MMP) enzymes, as well as localized inflammatory cell infiltrates that directly wear away at the coronary vessels [43,45]. When aneurysms are simultaneously found in the RCA and LAD, it is likely of a Kawasakis' pathology [13].

The current standard treatment for Kawasaki's Disease, primarily a disease of adolescence, is a single high dose of intravenous immunoglobulin (IVIG) and aspirin therapy if detected in a timely manner [43,45]. Irregardless of therapy or lack thereof, a study by Takahashi et al demonstrated that approximately 66% of all patients with Kawasaki's related CAA formation underwent spontaneous regression, while the treated population had an almost 71% regression rate [43,46]. Predominantly a disease in children, coronary artery complications such as rapid expansion and rupture are most likely to occur within the first 2-months of illness acquisition [43]. Long-term complications include persistence of the aneurysm, stenosis, or superimposed thrombosis [43].

Persons afflicted with genetic disorders including, but not limited to, Ehlers-Danlos Syndrome, Marfan's Syndrome, Systemic Lupus Erythematosus (SLE), or preferentially vasculitic disorders are at an increased risk for the development of CAAs when compared to the general population. This is likely due to inherent structural defects in the vessels, therefore increasing their susceptibility to localized weakening and dilatation [11,12]. Similarly, several studies have also detailed the occurrence of CAAs following systemic bacterial, fungal, or Lyme infections, likely late complications of inflammmation [2,41].

It is also important to acknowledge the incidence of post saphenous vein graft aneurysms, seen in persons at an average of thirteen years following coronary artery bypass grafting (CABG) [14]. This is a rare complication of surgery, thought to be secondary to wall degeneration and weakening [14]. Similarly in an iatrogenic sense, angioplasty with and without coronary stenting have been associated with the development of CAAs [15]. While not an established cause, CAAs have been identified in persons undergoing repeat angiography following placement of both bare-metal and drug-eluting stents, likely a result of poor vessel healing [15-17].

Although often asymptomatic and undetected, CAAs when symptomatic present frequently as a consequence of alterations in coronary arterial blood flow [10], subsequently presenting as an acute coronary syndrome (ACS). Clinical presentations vary vastly from stable or unstable angina to an NSTEMI [9,11,20]. In more emergent and less frequently encountered situations, a person may present as a STEMI, as exemplified by our case, or even fatally as an episode of sudden cardiac death [11]. Symptoms that may cause alarm for an ACS, such as dyspnea, angina, or palpitations, may aid in narrowing in on the pathological source of a CAA. Chest pain is thought to be a direct consequence of altered blood flow along the abnormal aneurysmal surface, leading to regional myocardial ischemia and typical chest pain [9].

Also likely due to turbulence and stasis of blood flow, superimposed thrombi are often found to be contained within the aneurysmal segment. Patients often remain symptom free until there are thromboembolic complications [1,9,19]. When there is a luminal occlusion and alterations in tissue perfusion, persons may present with ischemic chest pain. Less typically, vague presentations, such as with isolated neck pain, superior vena cava syndrome, or even an incidentally discovered 'cardiac mass' have been reported in the instance of giant coronary aneurysms [9,25]. Considering the wide range in which CAAs may present, a large degree of clinical suspicion is pertinent to avoid a missed diagnosis.

Coronary angiography remains the ideal invasive method in the diagnosis of CAAs, as it allows for the most accurate means of assessing aneurysm location, size, and morphology [8]. Angiography would also allow for the evaluation of concomitant coronary artery disease [8]. This is often combined with the use of intravascular ultrasound (IVUS) for more accurate intra-luminal sizing, stent sizing if indicated, and for overall treatment planning [26]. While an individual presenting with symptoms of an ACS is likely to swiftly receive cardiac catheterization, persons presenting with less emergent symptoms, or in an outpatient setting are likely to benefit from an alternative and less invasive means of coronary imaging.

Coronary computed tomography (CCTA) has been utilized for the evaluation of CAAs in these settings, often during the diagnostic workup of chest pain. Coronary CT has proven to be highly efficacious in identifying CAAs, with a reported sensitivity of 100%, similar to that seen in angiography

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[1,20,23,40]. Similar to CCTA is the use of cardiac magnetic resonance (CMR) imaging, however is less often employed due to its inferior ability to distinctly delineate coronary pathology and higher overall costs [44].

Despite the various documented CAA cases, there remains no definitive treatment in the presentations of such. Management is often situational and depends heavily on the presenting complaint of the individual. In the instance of an acute myocardial infarction (MI) presentation, the highest priority would be to restore coronary blood flow. Percutaneous coronary intervention (PCI) with stenting is occasionally performed to 'exclude' the aneurysm, such is to successfully prevent further blood flow into the weakened segment, thus reducing the risk of expansion and rupture [20,30].

While it is highly circumstantial and operator dependent, stenting is more likely to be successful in aneurysms between 16 to 26 mm in diameter as the stent may not successfully cover the entire surface of a larger ballooned segment [29,30]. If PCI is performed without stenting, as was in our case, it is often with the addition of intracoronary glycoprotein IIb/IIIa (GP2B3A) inhibitors, or other intracoronary thrombolytics [27]. Direct intracoronary delivery of the thrombolytic agent is done so for thrombus dissolution, thereby increasing TIMI flow [32].

The decision not to perform stenting in our patient was multiple-fold. Data has shown that in the setting of an ACS, stenting a CAA is associated with a higher rate of adverse events [2]. Stenting in the presence of a superimposed thrombus comes with the risk of inaccurate stent sizing, or risk of propagating the clot resulting in distal flow obstruction [2]. Additionally, the metal hardware of the stent itself poses as a nidus for thrombus formation [30].

Similarly, to stenting, thrombus aspiration is not performed during PCI for STEMI as multiple randomized trials have failed to show an improvement in clinical outcomes, and have instead trended toward increased cardiovascular death, transient ischemic attacks (TIA), or cerebrovascular accidents (CVAs) [47,48]. As of present literature, this is referred to as a Class III indication consistent with current ACA and AHA guidelines [47,48].

Eptifibatide, a GP2B3A inhibitor, acts by disaggregating platelet clumps, thereby improving microvascular flow in the epicardial arteries [31]. It is especially useful as an adjunct in PCI when stenting is insufficient, or not possible, such as when there is a particularly large thrombus burden [32]. The use of intracoronary Eptifibatide has effectively shown to restore TIMI flow and reduce the risk of major adverse cardiovascular events (MACE) [31].

Whether or not stenting is performed by the operator, dual antiplatelet platelet therapy (DAPT) has shown a modest reduction in MACE when compared to a no-treatment population [20,21]. This should especially be considered in persons with an ACS presentation, as the thrombus alone denotes a significant future thrombi-embolic risk [20,29]. Aspirin, in combination with an additional anti-platelet agent is prescribed as common practice [4,20]. Careful consideration should be taken with the use of DAPT, specifically in persons with a high bleeding risk [4].

Other considerations to make in the treatment of CAAs include the control of angiotensin converting enzyme (ACE). Data has shown that the over-expression of ACE and the subsequent production of Angiotensin II may promote the development of coronary artery ectasia [33]. With the additional cardioprotective benefits, it is common in practice to prescribe an ACE inhibitor, or alternatively an angiotensin receptor blocker (ARB) after the diagnosis of a CAA is made [1,33]. In patients with CAD and/or have presented as an ACS, treatment should resemble guidelines following revascularization. Statins reduce low dense lipoprotein (LDL) levels in the blood, and are thus essential in reducing atherosclerotic plaque formation [34,35].

Statins also have anti-thrombotic properties, scavenge harmful reactive oxygen species (ROS), and have an overall stabilizing effect on arterial plaque buildup [35]. These effects indicate that statins reduce patient susceptibility to ischemic events, and independently reduce the risk of a MACE [34,35]. Beta-blockers have multiple beneficial effects, such as with reducing myocardial contractility and workload, thus reducing the oxygen demand of cardiac tissue [37]. Similarly, to Statins, studies have demonstrated that beta-blockers independently reduce the risk of progression of carotid intima-media thickness (IMT), a marker of atherosclerosis [34,36].

The combination therapy of a statin and a beta-blocker exerts an even more potent reduction in MACE [34]. Strict control of these factors with the use of the aforementioned drugs may prevent the formation, or limit the expansion of already existing coronary aneurysms [27,34]. Of clinical importance, nitroglycerin (NTG) offers no clinical or symptomatic benefit in the treatment of CAAs [28]. A study illustrated that NTG has a paradoxical effect in that it worsens cardiac ischemia when administered in the presence of a CAA, which is in stark contrast with the proven benefit of NTG in typical CAD [28].

Surgical management in the treatment of CAAs are indicated in multiple circumstances; if there is severe concomitant CAD, CAAs with complications such as fistula formation or compressive features, rapidly expanding aneurysms with a high risk of rupture, and aneurysms that are secondary to iatrogenic causes [6]. There are various surgical techniques that may be employed, including bypass grafting, ligation, or resection of the aneurysm [22,38]. CAAs left to progress undergo a likelihood of calcification, thrombosis, or progressive expansion; all of which increase the risk of spontaneous aneurysmal rupture [24]. Patients

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with underlying atherosclerosis that predispose to thrombus formation, such as in our patient, are at risk of experiencing an ACS.

Questions:

Question 1: Which imaging modality is the primary technique of choice in evaluating coronary artery aneurysms?

A. Transthoracic echocardiogram
B. Cardiac MRI
C. Coronary Angiography
D. Transesophageal echocardiogram
E. Coronary CT Angiography

Explanation for Question 1: C) Coronary angiography remains the modality of choice in evaluating coronary artery aneurysms despite its invasive nature due to its ability to clearly visualize the aneurysm size, location, and morphology.

Question 2: What is the most common proposed etiology of CAA formation in adults?

A. Kawasaki's Disease
B. Consequence of concomitant CAD
C. Systemic Lupus Erythematosus
D. Ehlers Danlos Syndrome
E. Post-coronary arterial graft bypass

Explanation for Question 2: B) CAA formation is most frequently seen in adults with concomitant coronary artery disease (CAD). It is proposed that localized vessel inflammation and alterations in coronary flow along the vessel wall may predispose to CAA formation.

Question 3: Which of the following is a Class III indication as per ACC/AHA guidelines that relates to intervention regarding a CAA?

A. Aspirating a superimposed thrombus contained within a CAA
B. Injecting intracoronary glycoprotein IIb/IIIa inhibitors
C. The use of intravenous heparin
D. Undergoing CABG for revascularization
E. Aspirin monotherapy

Explanation for Question 3: A) A Class III indication is a condition that limits performing a procedure in an instance where the intervention is unlikely to be helpful and may possibly produce harm. Aspiration of a thrombus in this instance may cause clot propagation and increase the risk of stroke or MACE.

Question 4: What benefits can dual-antiplatelet therapy (DAPT) provide in terms of preventing complications associated with a CAA?

A. Reduces myocardial contractility
B. Scavenging of reactive oxygen species (ROS)
C. Reduction of low dense lipoprotein (LDL) levels in the blood
D. Reduction of risk of thrombus formation
E. Reduces the expression of Angiotensin converting enzyme (ACE)

Explanation for Question 4: D) DAPT has been shown to reduce risk of thrombus formation and therefore reduce the risk of thrombo-embolic complications, with an overall reduction in the risk of MACE.

Question 5: What imaging modality can be considered as an alternative to coronary angiography in the evaluation of CAAs with a similar sensitivity in detection rates?

A. Cardiac MRI
B. Coronary CT angiography
C. Transthoracic echocardiography
D. Transesophageal echocardiography
E. Nuclear scintigraphy

Explanation for Question 5: B) Coronary CT angiography can provide detailed imaging of the coronary vessels with a similar sensitivity to coronary angiography. This can be considered if the presenting complaint is less emergent and does not require urgent cardiac catheterization.

Conclusion

Coronary artery aneurysm formation in adults is often seen concomitantly with existing coronary artery disease, or as a late discovery in patients who were previously afflicted with Kawasaki's disease in adolescence. Management techniques remain controversial due to the distinct nature in which every case presents, as well as a lack of parameters to follow. Commonly employed methods however include medical management, PCI, or surgical intervention. We describe a case of a STEMI who was taken for immediate coronary angiography, during which a CAA with a superimposed thrombotic occlusion was discovered. The lesion was not amenable to thrombotic aspiration or stenting with PCI due to a significant risk of clot propagation or embolic complications, resulting in the utilization of intracoronary thrombolytics. The approach in the management of CAAs remains a challenge due to a lack of cohesive guidelines, and further prospective studies are likely to aid cardiac interventionalists in decision making in such predicaments.

Author Contributions:

RG: Conceptualization, writing of original draft and editing of manuscript

SS: Image acquisition and review

AG: Review and editing

GU: Supervision and final approval of manuscript

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FR: Supervision and final approval of manuscript

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Consent: Obtained

Ethics Approval: Not applicable.

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