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EC CARDIOLOGY
Case Report

# Piranha in a Goldfish Suit

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## Abstract

We present an interesting case of an atypical presentation in a 63-year-old male with sudden onset chest and jaw pain, and electrocardiographic findings suggestive of pericarditis. Following a positive exercise stress test, and ongoing intermittent symptoms not relieved by nitrate administration, he unfortunately succumbed to cardiac arrest with pulseless electrical activity secondary to hypovolemic shock. The preliminary autopsy report revealed an aortic dissection (AoD).

***Keywords:*** *Chest Pain; Pericarditis; ST Elevation; Aortic Dissection*

## Learning Objective

Aortic dissection though rare (3 per 100, 000) is an important condition requiring rapid diagnosis as it can quickly prove to be fatal. Whilst most cases (> 95%) have the traditional 'knife-like tearing pain', on occasion, patients can present with rather symptoms suggestive of an alternative diagnosis which can prove to be challenging to unify the diagnosis.

## Introduction

We present a case report of a 63-year-old male who presented with sudden onset chest and jaw pain, and subsequently succumbed to cardiac arrest secondary to hypovolemic shock from AoD.

This case report further re-enforces the difficulties in identifying AoD as a cause of chest pain in clinical practice. AoD is a diagnosis delayed or frequently missed mostly due to its rarity and large range of non-specific clinical manifestations.

## Case Description

A 63-year-old male was brought in by ambulance to the emergency department following the sudden onset of severe, burning central chest and jaw pain. The patient described the pain as similar in character to which he commonly experienced secondary to gastro-oesophageal reflux disease (GORD), but worse in severity. His medical background was only significant for GORD and osteoarthritis, for which he took no regular medications. He was a lifelong non-smoker and maintained excellent exercise tolerance.

His pain had subsided following 2.5 mg of morphine and 25mcg of fentanyl received in the ambulance on route to the emergency department. At the time of review, his heart rate dropped to 30 and was pre-syncopal, which resolved following the administration of 600mcg of atropine. Chest X-Ray revealed no possible cause for the pain, with normal lung fields and no widening of the mediastinum.

His ECG (Figure 1) was transmitted for review by the tertiary Cardiology service and was deemed to be consistent with pericarditis with widespread ST elevation and no reciprocal changes. He was admitted to the Coronary Care Unit, for work up of high risk acute coronary syndrome pain. Of note, his high-sensitivity troponin-I values remained low with delta values of 6 and 10.

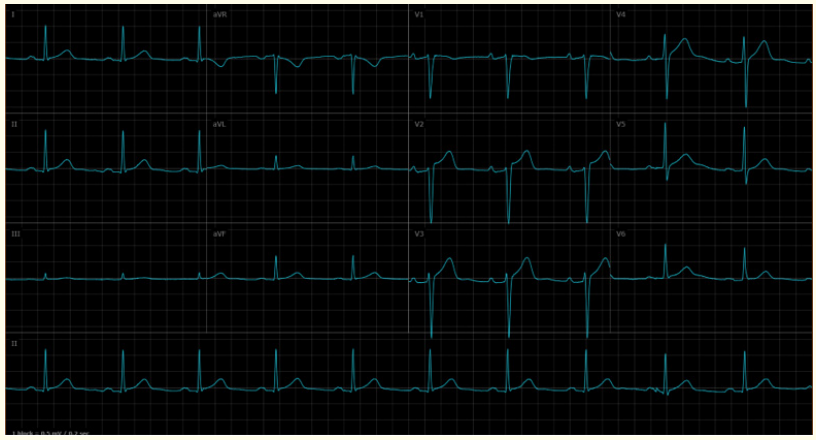

***Figure 1:*** *Initial ECG on presentation.*

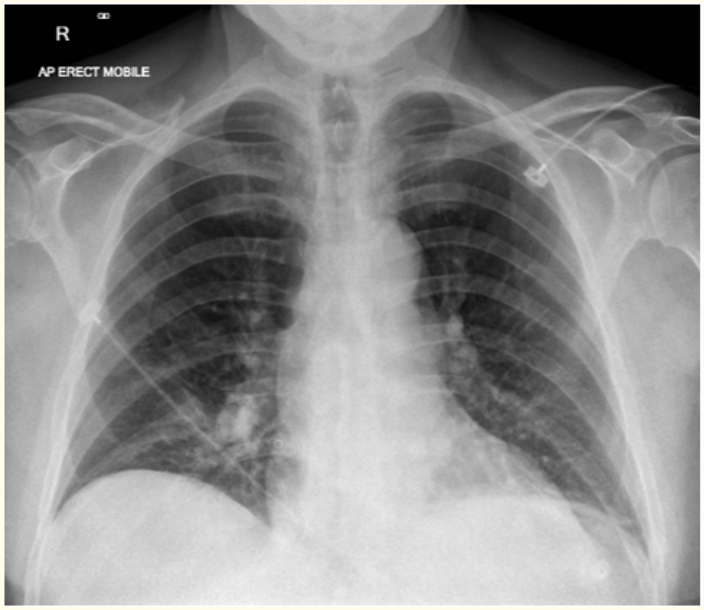


***Figure 2:*** *Chest X-ray showing no mediastinal widening, or other possible causes of chest pain.*

ECGs from five years prior were obtained from his general practitioner which showed long-standing ST elevation over the antero-lateral leads. A stress test was attempted as a non-invasive assessment of possible coronary vessel disease. The test was stopped after 5 minutes on the BRUCE protocol with a score of 7 METS due to re-occurrence of his jaw pain.

The next morning his ECG showed markedly increased ST elevation across all regions and subsequent PR depression (Figure 3). Clinically there was no pericardial rub or other signs of pericardial effusion, and he remained mostly asymptomatic except for two episodes of jaw pain overnight which were unresponsive to sublingual nitroglycerin but responded well to low-dose intravenous morphine. A computed-topography coronary angiogram (CTCA) was ordered, but unable to be performed due to concerns of poor image quality as his heart rate remained ~75 beats per minute in spite of beta-blockade.

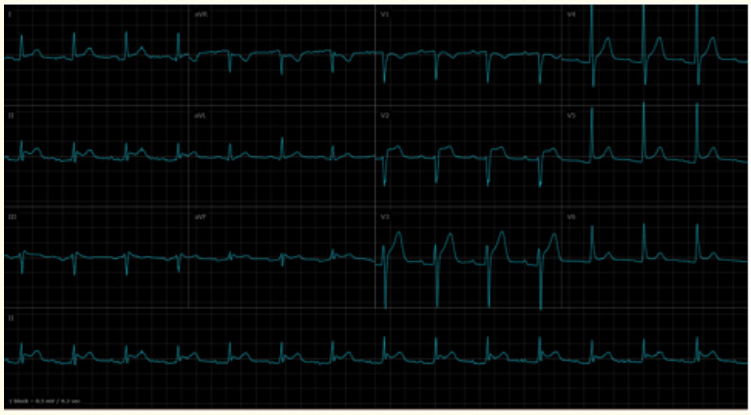

***Figure 3:*** *Progression of ECG on Day 3. ECG taken routinely, patient asymptomatic.*

He remained asymptomatic overnight, and with no changes in routine ECGs. The following afternoon he collapsed and was found to have pulseless electrical activity (PEA) with an unrecordably low blood pressure. Cardio-pulmonary resuscitation (CPR) was commenced, and he was intubated and started on inotropic infusions.

An urgent bedside transthoracic echocardiogram revealed a mild to moderate pericardial effusion, and a poorly contracting heart across all chambers. Pericardiocentesis was performed, with a small improvement in cardiac output following the removal of 20 ml of blood-stained fluid. The patient was moved to the intensive care unit for ongoing support and management, but subsequently suffered another cardiac arrest with poor cardiac output, and the decision for palliation was made following discussion with his family. Autopsy was performed, and preliminary verbal reports revealed a Type A aortic dissection to be the cause of death.

## Discussion

Aortic dissection (AoD) is a rare, life-threatening disease with high associated morbidity and mortality [1,2]. Despite this, it is potentially treatable if diagnosed and managed early and appropriately [1]. Untreated, mortality is recorded as 33% within the first 24 hours and up to 50% within the first 48 hours [2]. The most common pre-disposing factors include hypertension and atherosclerotic disease, with other important conditions such as connective tissue disorders and bicuspid aortic valves [3].

AoD has slightly varying pathophysiology for each presentation due to several predisposing factors which will influence the site of the tear in the intima, leading to blood flow along the aortic media and the formation of a false lumen [2]. Classification systems for AoD are commonly based on anatomical location, with the Stanford classification describing type A dissections as those involving the ascending aorta and type B dissection as all others [1,2].

Clinical manifestations of AoD are varied in nature, and thus routine clinical examination is insufficient in ruling out the disease [2]. A review of twenty years of data from the International Registry of Acute Aortic Dissection (first established in 1996) including 7300 cases from 12 countries attempted to elicit a diagnostic framework for assessing thoracic pain [4].

Abnormal ECG findings are commonly present and often non-specific or suggestive of coronary artery disease (42% of patients described by the International Registry of Acute Aortic Dissections). This often delays diagnosis and treatment due to the indication of a much more common primary differential [4].

In addition to the lack of classical symptomatology or disease course, this patient did not develop any biochemical or radiological features of aortic dissection. Cardiac troponin-I is elevated frequently in patients with Stanford Type A AoD and is associated with higher mortality [5]. A standard practice in patients presenting with chest pain, chest x-ray is thought to reveal AoD through the presence of widening of the mediastinum [3,4]. Our patient re-inforced that this is not always the case, with over 20% of patients with confirmed AoD lacking mediastinal or aortic contour abnormalities on chest x-ray [3].

Unfortunately, in this patient, the diagnosis was not made until autopsy. This case highlights some of the various challenges present in diagnosing AoD with atypical clinical and biochemical manifestations as those described in the literature. Given the variability in its clinical manifestations, AoD should be considered as a differential diagnosis in all patients presenting with chest, back or abdominal pain, syncope, or symptoms of perfusion deficit. There are several proposed strategies to make a diagnosis of AoD. We believe that these patients should be risk stratified by the presence of high-risk features, predisposing factors and help order the appropriate imaging to help make the diagnosis. We propose a simplified strategy (Figure 4).

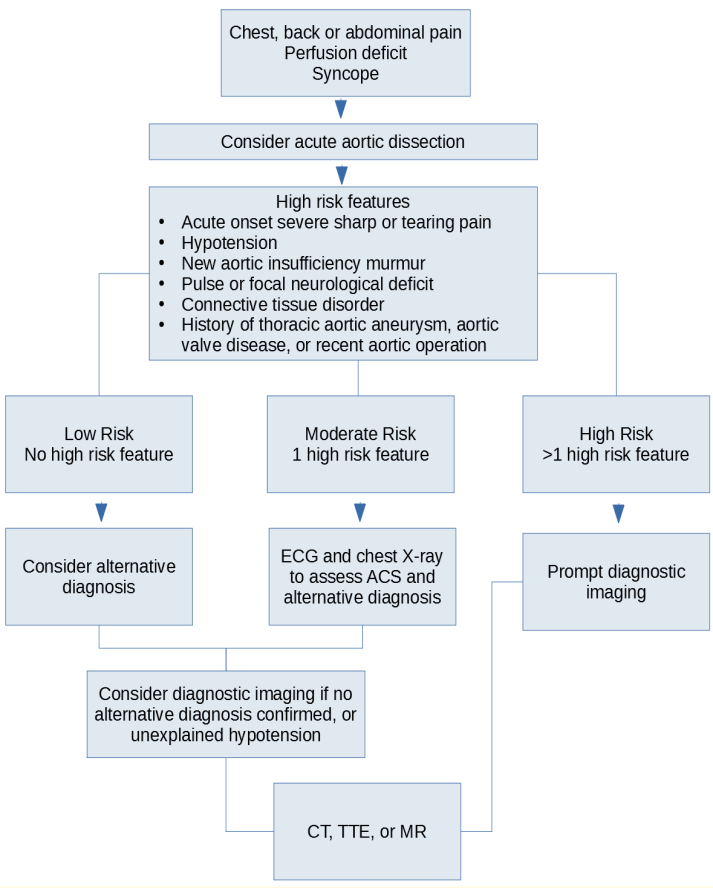


***Figure 4:*** *Proposed diagnostic pathway of patients presenting with potential AoD.*

## Conclusion

AoD is a rare cause of thoracic pain with increasing amounts of literature attempting to provide diagnostic tools to assist in its difficult diagnosis. This case effectively highlights one of many atypical presentations of AoD, suggesting that diagnostic tools must be used with care and in conjunction with clinical judgement and further definitive investigations.

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