In September 2011, a 55 year old active man, ex-smoker with previous history of squamous cell cancer of the lung, presented to the hospital with frontal headache of > 12 hours duration and low grade fever. Headache was severe and not accompanied by nausea, vomiting or sensitivity to light. His lung cancer was treated with surgery and radiation therapy, which was completed in 2010. His treatment was complicated by post radiation pneumonitis. He was very active, bicycling regularly up to 10 miles, three times per week. His main reason for hospital visit was headache, but on arrival he also reported atypical right sided pleuritic chest pain, which was attributed to his radiation pneumonitis.

A computerised tomographic (CT) scan of the chest showed new right middle lobe opacity, consistent with inflammation or an infectious process. A 12 lead electrocardiogram (ECG) showed a small Q wave in lead I11 and AVF. Peak troponin I level was 3.5 ng/ml (normal value = 0.00 to 0.03 ng/ml, with a cut off value of 0.5ng/ml for diagnosis of MI). A 2D echocardiogram showed normal left and right ventricular systolic function with no regional wall motion abnormalities and a CT brain did not reveal any pathology. Review of previous CT chest showed coronary artery calcification. Patient was treated with aspirin, beta-blockers and statin. In view of coronary calcification, elevated troponin and previous chest radiation, he underwent an emergent coronary angiogram which showed a significant lesion with thrombus in his left anterior descending (LAD) artery for which he underwent percutaneous coronary angioplasty and stenting. (Figure 1 and 2). Patient was not exactly sure as to when his headache completely resolved; however, he recalls waking up after the coronary intervention with full resolution of his headache. Since his coronary intervention he has had no further headache.

ABSTRACT:
We present a case of 55 year old man, with myocardial infarction and coronary thrombosis, whose initial presentation was with severe headache and review the literature.

INTRODUCTION:
Coronary ischemia typically presents with retrosternal pain that radiates to left arm (1). It may present atypically in various forms like indigestion (2), otalgia (3), facial pain (4) and syncope (5). Headache as the sole presentation of myocardial infarction (MI) is rare; however it has been reported previously. In a study of 150 patients with angina, about 6% were found to have concomitant headache (6). In this article, we present a patient with myocardial infarction, whose main presentation was with headache and review other cases published in the literature.

CASE REPORT
In September 2011, a 55 year old active man, ex-smoker with previous history of squamous cell cancer of the lung, presented to the hospital with frontal headache of > 12 hours duration and low grade fever. Headache was severe and not accompanied by nausea, vomiting or sensitivity to light. His lung cancer was treated with surgery and radiation therapy, which was completed in 2010. His treatment was complicated by post radiation pneumonitis. He was very active, bicycling regularly up to 10 miles, three times per week. His main reason for hospital visit was headache, but on arrival he also reported atypical right sided pleuritic chest pain, which was attributed to his radiation pneumonitis.

Pain was pleuritic with no features of angina. Patient had no previous history of known coronary artery disease or intake of nitrates. He had no previous history of headaches and no family history of migraine. On examination his temperature was 37.4C, with oxygen saturation of 95% on room air. Clinical examination was otherwise unremarkable. Investigation included a chest x-ray which showed right middle lobe opacity, suggestive of pneumonitis. A computerised tomographic (CT) scan of the chest showed new right middle lobe opacity, consistent with inflammation or an infectious process. A 12 lead electrocardiogram (ECG) showed a small Q wave in lead I11 and AVF. Peak troponin I level was 3.5 ng/ml (normal value = 0.00 to 0.03 ng/ml, with a cut off value of 0.5ng/ml for diagnosis of MI). A 2D echocardiogram showed normal left and right ventricular systolic function with no regional wall motion abnormalities and a CT brain did not reveal any pathology. Review of previous CT chest showed coronary artery calcification. Patient was treated with aspirin, beta-blockers and statin. In view of coronary calcification, elevated troponin and previous chest radiation, he underwent an emergent coronary angiogram which showed a significant lesion with thrombus in his left anterior descending (LAD) artery for which he underwent percutaneous coronary angioplasty and stenting. (Figure 1 and 2). Patient was not exactly sure as to when his headache completely resolved; however, he recalls waking up after the coronary intervention with full resolution of his headache. Since his coronary intervention he has had no further headache.

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However after 3-4 months patient had episodes of chest heaviness, sometimes with exertion and occasionally with rest. In March 2012, he underwent coronary angiogram in his hometown, which showed diffuse disease in the area or previous LAD stent and first diagonal, for which he underwent coronary artery bypass grafting (left internal mammary artery to LAD and saphenous vein graft to first diagonal). He has remained well since than with no angina or headache.

**DISCUSSION**

According to the International classification of headache 2004, the diagnostic criteria for cardiac cephalgia includes: Headache, aggravated by exertion, accompanied by nausea & associated with myocardial ischemia and resolution with effective therapy for myocardial ischemia (7). Review of previous cases, as listed in Table 1 indicates that not all patients with cardiac cephalgia have exertional headache and it may be induced at rest. Exertional headache, in appropriate clinical setting may alert physicians to consider myocardial ischemia.

Cardiac cephalgia has no specific clinical features. It may be unilateral or bilateral, localised or radiated, present in different sites and may or may not be associated with chest pain. In reported cases (Table 1) (8-18), all patients had at least one cardiovascular risk factor and there was complete resolution with treatment of coronary ischemia (either pharmacological or interventional) with no recurrence of headache, except in cases of reocclusion. Migrainous headache would give similar clinical scenario. So it is difficult to assume whether an ischemic cardiac pain is the cause of such a headache or cardiac event had precipitated an attack of migraine. Our patient presented with low grade fever due to pneumonitis and fever can also precipitate migrainous headache in susceptible individual.

The mechanism behind cardiac cephalgia is still unclear. However a number of theories are thought to contribute to this rare presentation. The first theory is based on the belief that any afferent pathway conveying visceral pain from the heart must converge on cells in central nervous system that receive information from varied somatic regions (19,20,21). A study of cardiac afferents showed that angina pain is mediated by sympathetic fibres in 50-60% of cases, by vagal fibres in 10-20% and through both of them in 30-40% of cases (19). According to this we can give possible explanation for different anginal pain (19).

Typical pain is mediated by cardiac autonomic afferents from dorsal roots of T1-T5 which converge with somatic inputs from chest and left arm in posterior horns of spinal cord or in thalamus (20, 21). Occipital headache, pain in neck and jaw area is thought to be mediated by convergence of cardiac afferents mainly vagal with somatic afferents from C1-C3 in upper spinothalamic tract (20, 21). The spinal nucleus of trigeminal has extension right down to C2 which is responsible for neck pain and migraine. The trigemino-vascular system through the trigeminal nucleus caudalis may as well receive innervations from cardiac afferents. The trigeminal nerve, distributes pain sensations to nasal mucosa, oral cavities, nasal cavities facial skin and portions of dura matter (22). Coronary ischemia may present as headache through the convergence of vagal afferents from heart with trigeminal neuron in the spinal trigeminal nucleus (20, 21, 23). Rarity of this manifestation might be attributed to small percentage of cases regulated by vagal fibres only (10-20%), (19).

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### Table 1: Review of previous cases of coronary ischemia, presenting as headache

<table>
<thead>
<tr>
<th>Reference</th>
<th>Age/sex</th>
<th>Site</th>
<th>Intensity</th>
<th>Chest Pain</th>
<th>Trigger</th>
<th>Risk Factors</th>
<th>ECG Findings</th>
<th>Coronary Angiogram</th>
<th>Therapy</th>
<th>Outcome</th>
<th>Discharge Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>[8]</td>
<td>78/F</td>
<td>Bitemporal</td>
<td>Severe</td>
<td>No</td>
<td>NA</td>
<td>H,L</td>
<td>ST Elevation</td>
<td>Triple vessel disease</td>
<td>CABG</td>
<td>Resolved</td>
<td>Alive</td>
</tr>
<tr>
<td>[8]</td>
<td>77/F</td>
<td>Right frontal &amp; maxillary</td>
<td>Severe</td>
<td>No</td>
<td>NA</td>
<td>NA</td>
<td>Non specific ST changes T wave inversion</td>
<td>Patent coronaries</td>
<td>NA</td>
<td>NA</td>
<td>Alive</td>
</tr>
<tr>
<td>[9]</td>
<td>42/M</td>
<td>Frontal &amp; bitemporal</td>
<td>Severe</td>
<td>No</td>
<td>NA</td>
<td>H,L,S,F</td>
<td>ST elevation Q wave T inversion</td>
<td>LAD 100% occlusion</td>
<td>PCI</td>
<td>Resolved</td>
<td>Alive</td>
</tr>
<tr>
<td>[10]</td>
<td>61/F</td>
<td>Forehead radiated to head, neck, jaw &amp; teeth</td>
<td>Severe</td>
<td>No</td>
<td>Exertion</td>
<td>H,L,F</td>
<td>ST elevation in lateral walls ST depression in V1-V6</td>
<td>LAD 70%, RCA 80%, CXA 95%, PDA 90%</td>
<td>PCI</td>
<td>Resolved</td>
<td>Alive</td>
</tr>
<tr>
<td>[11]</td>
<td>62/M</td>
<td>Right frontal</td>
<td>Severe</td>
<td>Yes</td>
<td>Exertion</td>
<td>S,L</td>
<td>ST depression</td>
<td>LAD 100%, RCA 100%</td>
<td>CABG</td>
<td>Resolved</td>
<td>Alive</td>
</tr>
<tr>
<td>[12]</td>
<td>52/F</td>
<td>Bilateral</td>
<td>Severe</td>
<td>Yes</td>
<td>Rest</td>
<td>NR</td>
<td>NA</td>
<td>Patent coronaries</td>
<td>Nitrates</td>
<td>Resolved</td>
<td>Alive</td>
</tr>
<tr>
<td>[12]</td>
<td>67/F</td>
<td>Bilateral tempoparietal &amp; mandibular</td>
<td>Severe</td>
<td>No</td>
<td>Exertion</td>
<td>D,L,H</td>
<td>Normal</td>
<td>Double vessel disease</td>
<td>PCI</td>
<td>Resolved</td>
<td>Alive</td>
</tr>
<tr>
<td>[14]</td>
<td>55/M</td>
<td>Bilateral Parietal radiated to jaw</td>
<td>Severe</td>
<td>No</td>
<td>Exertion</td>
<td>L,S,H,O</td>
<td>Normal</td>
<td>LAD 60% RCA 100%</td>
<td>PCI</td>
<td>Resolved</td>
<td>Alive</td>
</tr>
<tr>
<td>[16]</td>
<td>74/F</td>
<td>Bitemporal</td>
<td>Severe</td>
<td>Yes</td>
<td>Rest</td>
<td>O,D, stroke</td>
<td>ST depression</td>
<td>NA</td>
<td>Nitrates</td>
<td>Resolved</td>
<td>Alive</td>
</tr>
<tr>
<td>[16]</td>
<td>64/F</td>
<td>Unilateral radiated to jaw</td>
<td>Severe</td>
<td>No</td>
<td>Exertion &amp; Rest</td>
<td>D,H,L</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>Death</td>
</tr>
<tr>
<td>[17]</td>
<td>36/M</td>
<td>Bilateral occipital, vertex</td>
<td>Severe</td>
<td>No</td>
<td>Rest</td>
<td>Stroke</td>
<td>ST elevation</td>
<td>LAD 90%</td>
<td>PCI</td>
<td>Resolved</td>
<td>Alive</td>
</tr>
<tr>
<td>[17]</td>
<td>85/F</td>
<td>Eye ball</td>
<td>Severe</td>
<td>Yes</td>
<td>Rest</td>
<td>No</td>
<td>Normal</td>
<td>Patent coronaries</td>
<td>Nitrates</td>
<td>Resolved</td>
<td>Alive</td>
</tr>
</tbody>
</table>

H = hypertension; L = hyperlipidemia; S = smoker, D = diabetes; F = family history; PCI = percutaneous coronary intervention; TIA = transient ischemic attack; LAD = left anterior descending artery; RCA = right coronary artery; RCX = right circumflex artery; PDA = posterior descending artery
Second theory claims that reduction of cardiac output causes decreased venous return from brain resulting in increased intracranial pressure $^{19, 20}$. Though it is not proved that myocardial infarction is accompanied by increased intracranial pressure, this theory might explain cardiac headache triggered by exertion, cough and sexual activity.

Finally the third theory attributes the headache to the pro-inflammatory mediators released during ischemic process (bradykinin, serotonin, histamine, adenosine) causing cerebral vasodilatation $^{19, 20}$. This theory is not completely understood as headache in these patients is resolved with nitrates which are supposed to cause headache and not relieve it.

**CONCLUSION**

Headache as main manifestation of acute myocardial infarction is a rare but a well recognised entity. In patients with cardiovascular risk factor and no previous history of migraine or headache, and in whom other causes for headache has been excluded, a possibility of a coronary event must be considered. A high degree of clinical suspicion is needed to diagnose this condition, which can be fatal if left untreated. Initial ECG may be normal; hence serial ECGs are helpful in arriving at a correct diagnosis $^{14}$. 

**Figure 2** illustrates the pathways of referred pain for coronary ischemia.

**Figure 2**: Diagram illustrating pathways of referred pain for coronary ischemia. NTS: nucleus tractus solitary (the nucleus of the solitary tract).
REFERENCES

22. Spinal nucleus & tract of the trigeminal Available from: http://www.neuroanatomy.wisc.edu/virtualbrain/BrainStem/03CNV.html