

An Unusual Presentation of Inferioposterior Wall Acute Myocardial Infarction as Severe Headache

Kaul Mohit*, Gupta Ayush*, Datta Kishalay**, Das Indranil***

Abstract

Despite the progress in the laboratory detection of acute myocardial infarction (AMI), medical history remains the most important diagnostic step in order to establish the diagnosis. Most patients with AMI describe a severe, pressure-type pain in the mid-sternum, often radiating to the left arm, neck or jaw. The pain resembles angina, but it can be distinguished from it by its intensity, duration (>30 min), and failure to resolve with nitroglycerin administration. The pain may also be accompanied by nausea, vomiting and diarrhea, especially when the infarction is located in the inferior wall. Other symptoms include dyspnea, diaphoresis, dizziness, palpitations, cold perspiration, profound weakness and syncope. The diagnosis of AMI may be difficult when atypical symptoms occur, such as indigestion, unusual localization of the pain, agitation and altered mental status. Furthermore, AMI may be silent in more than 25% of cases, in which the infarction is not recognized by the patient and evidence of the infarction is provided by the electrocardiogram or post-mortem examination. Asymptomatic infarction occurs more frequently in elderly patients with hypertension, angina pectoris, and, mostly, diabetes mellitus, as a result of the polyneuropathy that accompanies long-standing and uncontrolled diabetes. We describe here a case which presented to ED with severe headache accompanied with nausea, vertigo. During routine investigation, ECG was done which showed ST elevation in lead II, III, AVF and showed marked ST-segment depression in leads V₁ to V₅ and slight ST-segment depression in leads I and aVL. So headache was the sole symptom of AMI which was very rare.

Keywords: Myocardial Infarction; Headache; Vertigo; Chest Pain; Nausea.

Author's Affiliation:

*MEM PGY 1, **Attending Consultant, ***HOD, Senior Consultant, Department of Emergency Medicine, Max Hospital, Shalimarbagh, New Delhi.

Corresponding Author:

Mohit Kaul, MEM PGY 1, Department of Emergency Medicine, Max Hospital, Shalimarbagh, New Delhi., Delhi 110088.
E-mail: dr.mohitkaul@gmail.com

Introduction

Despite the progress in the laboratory detection of acute myocardial infarction (AMI), medical history remains the most important diagnostic step in order to establish the diagnosis. Most patients with AMI describe a severe, pressure-type pain in the mid-sternum, often radiating to the left arm, neck or jaw. The pain resembles angina, but it can be distinguished from it by its intensity, duration (>30 min), and failure to resolve with nitroglycerin administration. The pain may also be accompanied by nausea, vomiting and diarrhea, especially when the infarction is located in the inferior wall. Other symptoms include dyspnea, diaphoresis, dizziness, palpitations, cold

perspiration, profound weakness and syncope. The diagnosis of AMI may be difficult when atypical symptoms occur, such as indigestion, unusual localization of the pain, agitation and altered mental status. Furthermore, AMI may be silent in more than 25% of cases, in which the infarction is not recognized by the patient and evidence of the infarction is provided by the electrocardiogram or post-mortem examination. Asymptomatic infarction occurs more frequently in elderly patients with hypertension, angina pectoris, and, mostly, diabetes mellitus, as a result of the polyneuropathy that accompanies long-standing and uncontrolled diabetes.

We describe a case presented with headache as the leading symptom of AMI.

Case History

49 yr old female patient presented to the ED with complains of sudden onset of severe headache accompanied with nausea, vertigo and difficulty in walking since 1day. She had no complain of Chest pain, breathlessness or sweating. She had no previous history of headache or vertigo. No history of any trauma or fall in past. No history of CAD. She was recently diagnosed for Hypertension and Diabetes Mellitus.

During routine investigation, ECG was done which showed ST elevation in lead II, III, AVF and showed marked ST-segment depression in leads V₁ to V₅ and slight ST-segment depression in leads I and aVL. There was ST-segment elevation in the posterior leads V₇ to V₉, s/o Acute Inferioposterior wall MI. NCCT Head was done in view of complain of headache which didn't reveal any bleed, Echo showed LVEF 48%. After informed consent, she underwent CAG which revealed single vessel disease, PTCA+ STENT to LCX was done.

On Examination the Vitals were

Pulse - 120/min

BP- 160/90 mmHg

RR-18/min

Spo2-100% in Room air

RBS- 340mg/dl.

Temp- Afebrile.

Cardiac monitor - Normal Sinus rhythm

HEENT- WNL and no JVD and no Carotid bruit.

PUPIL- Right- NSNR

Left- NSNR

Chest- B/L Air Entry +, Crepitations+

CVS- S1 and S2 audible and no murmur heard.

P/A- Soft and no guarding and no rigidity, no tenderness and normal bowel sounds and Bowel Sounds present. No pulsatile mass and no bruit or thrill felt in the abdomen.

CNS- Conscious and oriented, GCS 15/15.

Memory: Short & Long term memory- Normal

Orientation: To time, place and person - Normal

Speech :Normal.

Cranial nerve :I to XII intact.

Sensation: Superficial sensation intact, Joint sense intact.

Motor: Rt UL & LL WNL and no deficit

Lt. UL & LL WNL and no neurodeficit.

Power: Right upper & Lower limb 5/5.

Left Upper & Lower Limb 5/5.

Plantar: B/L Flexor

Sensation- Intact (B/L UL & LL).

Extremity and Exposure: WNL and no pedal edema and no rashes.

Past History: Recently Diagnosed case of HTN and DM.

AMPLE history: No allergy, No past medical or surgical history, Ate her breakfast, Sudden onset of Headache, nausea and headache.

Impression:- Cad, Inferioposterior Wall MI

Investigations

ECHO -

LA-3.6

AORTA-2.9

LVID-4.4

IVS-1.3

PW-1.3

PULM-96 CM/SEC

AORTIC-122 CM/SEC

E-62 CM/SEC

A-69 CM/SEC

Conc. Lvh

Hypokinetic lcx territory

Lvef-48%

Mild mr

No clot/veg/pe

Ncct head was done in view of c/o headache- which didn't reveal any bleed.

Cag was done through rt radial route

Lmca-normal

Lad-normal

Lcx-proximal 100% occlusion

Rca-dominant; normal

IMP-SVD

ADV-P+S TO LCX

P+S TO LCX

Rt radial 6f

Lmca was hooked with ebu 3.5 6f
 Bmw wire
 Predilated with 2.0 x 10 and 2.5 a 10 mm balloon @ 10 atm
 Bvs absorb 3.0 x 28 mm was deployed @ 14 atm
 Post dilated with 3.25 x 9 mm @ 18 atm and 3.5 x 9 mm nc balloon @ 15-18 atm

TIMI-3 FLOW

No Complication; Good End Result

Laboratory Investigations:

TLC	14.3
RBC	4.49
HAEMOGLOBIN	9
PACKED CELL VOLUME	29.7
MCV	66.1
PLATELET COUNT	150
NEUTROPHILS	82%
LYMPHOCYTES	8%
MONOCYTES	6%
EOSINOPHILS	4%
SERUM CREATININE	0.65
SODIUM	132.9 L
POTASSIUM	4.6
CHLORIDE	94.5
TOTAL PROTEIN	7.5g
ALBUMIN	4g/dL
BILIRUBIN,TOTAL	0.4mg/dL
BILIRUBIN,DIRECT	0.1mg/dL
SGOT (AST)	18 LIU/L
SGPT (ALT)	20 IU/L
A.G. RATIO	1.1 L
GLOBULIN	3.5g/dL
BICARBONATE	24mmol/L
CHOLESTEROL SERUM	167mg/dL
TRIGLYCERIDE	136mg/dL
CHOLESTEROL HDL	47.5mg/dL
LDL CHOLESTEROL	118mg/dL
VLDL CHOLESTEROL	27.2mg/dL
TSH	2.18uIU/mL
TROP I	0.69ng/mL
HbA1c	8.5%

Prothrombin	14.7
Partial Thromboplastin Time	20
INR	1.15
APTT Control Plasma	28

Treatment Given

1. Acetylsalicylic acid sr ud 75mg tab,ec 75mg po qdaily(10hrs)
2. Alprazolam 0.5mg ud tab 0.5mg po qhs(daily 10:00pm)
3. Augmentin 1.2gm vial inj, susp 1.2 gm iv q8h (6,14 &22 hrs)
4. Cholecalciferol ud 60000 pouch 1 packet po prn twice weekly
5. Dexorange plus cap,oral 1 capsule po qam(06hrs)
6. Folic acid ud 5mg tab 5mg po qam(06hrs)
7. Furosemide 10mg/ml 2ml ud amp inj,soln 20 mg iv q12h(6,18hrs)
8. Huminsulin r inj 100iu vial inj 100unt/1ml sq Qac(7,12&18 hrs) 6 units bbf 6 units bl 6 units bd
9. Lantus 100iu 3ml cartridge inj 100unt/1ml sq Qhs(daily 10:00pm) 18 units hs after dinner
10. Metoprolol xr ud 25mg cap,oral 1 capsule po bid(10&22hrs)
11. Nervz b cap,oral 1 capsule po qam(06hrs)
12. Pantoprazole ud 40mg tab,ec 40mg po qam(06hrs)
13. Rosuvastatin ud 40mg tab 40mg po qhs(daily 10:00pm)
14. Telma am h 40 tab 1tablet po qam(06hrs)
15. Ticagrelor ud 90mg tab,oral 1 tablet po bid(10 &22hrs).

Conclusion

AMI with typical chest pain is relatively easy to diagnose. However, the diagnostic procedure becomes difficult and can be missed out when the patient complains for atypical symptoms. Headache may co-exist with other typical symptoms. However, headache as an only leading symptom of an AMI is extremely rare. Such cases have been rarely documented.

In a patient with a history of diseases, such as arterial hypertension, diabetes mellitus, hyperlipidemia, smoking, and positive family history

which are a risk factor for cardiovascular diseases, increase the possibility of AMI, and therefore AMI should always be included in the differential diagnosis when evaluating such patients

In this case the fact that the patient was relieved from his severe headache immediately after the PCI suggests that the headache and cardiac ischemia may be closely correlated. Although the underlying pathophysiologic mechanism of headache is not well understood, one could presume that an acute reduction of the cardiac output as a result of an AMI, could result to the onset of neurological manifestations, such as headache. Alternatively, the headache in the setting of an acute cardiac event may be related to a generalized catecholaminemia-induced vasospastic disorder and subsequent increase of intracranial pressure. Another explanation of the headache in case of AMI involves the perception of cardiac ischemic pain as headache due to the convergence - most likely at a thalamic level - of cardiac autonomic nerve fibers with somatic nerves originating from the head. Finally, it has been suggested that headache-eliciting mediators released during cardiac ischemia may also have a role in the manifestation of headache; however this issue is still under investigation

In conclusion, we present an extremely rare case of an AMI presenting with headache as the only leading symptom. Despite the progress in the laboratory detection of acute myocardial infarction (AMI), medical history remains the most important diagnostic step in order to establish the diagnosis. Physicians should always consider headache as a presenting symptom of an acute coronary event, especially in patients at risk for cardiovascular disease. Physicians should consider the possibility of ischemic heart disease at every patient with recurrent headache on exertion, after the exclusion of intracranial pathology, especially in the presence of cardiovascular risk factors.

Key Points

- MI can present as headache.
- MI can present as only arm pain.
- MI can present as pain abdomen only.

References

1. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. *Diabetes Care*. 2004; 27: 1047-1053.
2. Backer G, Ambrosioni E, Boch-Johnsen K, Brotons C, Cifkova R, Dallongeville J. European guidelines on cardiovascular disease prevention in clinical practice. Third Joint Task Force of European and other Societies on Cardiovascular Disease Prevention in Clinical Practice (consulted by representatives of eight societies and by invited experts) *Eur J Cardiovasc Prev Rehabil*. 2003; 10 Suppl: S1-78.
3. Estadísticas Sanitarias Mundiales (Internet) Geneva: World Health Organization; 2012 (cited 2012 Aug 5). 178 p.
4. Kannel WB, McGee DL. Diabetes and glucose tolerance as risk factors for cardiovascular disease: the Framingham study. *Diabetes Care*. 1979; 2: 120-126.
5. Kannel WB, McGee DL. Diabetes and cardiovascular disease. The Framingham study. *JAMA*. 1979; 241: 2035-2038.
6. Stamler J, Vaccaro O, Neaton JD, Wentworth D. Diabetes, other risk factors, and 12-yr cardiovascular mortality for men screened in the Multiple Risk Factor Intervention Trial. *Diabetes Care*. 1993; 16: 434-444.
7. Kang X, Berman DS, Lewin HC, Cohen I, Friedman JD, Germano G, Hachamovitch R, Shaw LJ. Incremental prognostic value of myocardial perfusion single photon emission computed tomography in patients with diabetes mellitus. *Am Heart J*. 1999; 138: 1025-1032.