Relation of Nausea and Vomiting in Acute Myocardial Infarction to Location of the Infarct

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To determine whether the incidence of nausea and vomiting in patients with acute myocardial infarction (AMI) varies with infarct location, we studied 180 patients who had been admitted to our hospital for ST-segment elevation AMI or AMI associated with left bundle branch block. The presenting symptoms (chest pain, nausea, and vomiting), initial electrocardiographic findings, and additional demographic, clinical, laboratory, and outcome data were extracted from the medical records and correlated with the infarct location. Of the 180 patients with AMI, 108 (60%) had inferior and 72 (40%) had anterior infarcts. Nausea was reported in almost 2/3 of all patients, and vomiting in nearly 1/3. Both nausea and vomiting showed a trend toward a greater incidence in patients with inferior than with anterior infarcts (69% vs 56% and 33% vs 26%, respectively). However, the differences were not statistically significant. In conclusion, nausea and vomiting are common presenting symptoms in patients with either inferior or anterior wall AMI, but their frequency is unrelated to the infarct location. **(Am J Cardiol 2009;104:1638–1640)**

Nausea and vomiting occur frequently in patients with acute myocardial ischemia and acute myocardial infarction (AMI), although the stimuli that evoke the nausea and vomiting remain uncertain. Some studies have reported that nausea and vomiting are much more common in patients presenting with AMI involving the inferior portion of the left ventricle than in patients with anterior wall AMI.[1,2] Other studies have found just the opposite.[3,4] To re-examine the relation between infarct location and both nausea and vomiting, we performed a cohort study of 180 patients who had been admitted to our community-based teaching hospital for either ST-segment elevation AMI (STEMI) or AMI associated with left bundle branch block (LBBB).

Methods

Our hospital's institutional review board approved the study on October 24, 2006. The candidates included 194 patients who had been admitted to our hospital from July 1, 2001 to October 24, 2006 with STEMI or AMI associated with LBBB. Of the 194 patients, 14 were excluded, 9 because their records were unavailable, 4 because their AMI was both anterior and inferior, and 1 because the history regarding nausea and vomiting was unobtainable owing to an out-of-hospital cardiac arrest without regain of consciousness. The remaining 180 patients, 176 with STEMI and 4 with AMI and LBBB, were included. The infarct location (i.e., inferior vs anterior) in the patients with STEMI was determined using the established World Health Organization electrocardiographic criteria[5] and was confirmed after review of each electrocardiogram by an experienced cardiologist (JFH) unaware of the clinical presentation. The infarct location in the 4 patients with AMI and LBBB (which was new in 3 patients) was assigned according to the electrocardiographic criteria[6] and also by review of the cardiac catheterization findings by the cardiologist. The anterolateral, lateral, and anteroseptal infarcts were classified as anterior, and the inferoposterior and inferolateral infarcts were classified as inferior.

The notes from all healthcare providers were reviewed by one of the authors (EEF). The presence or absence of nausea and vomiting at presentation was clearly documented in these notes for 179 of the 180 patients. In 1 patient, no comment was found anywhere in the extensive medical record regarding nausea or vomiting, and we assumed that the patient had had neither. The primary outcome of the present study was a clinical presentation with spontaneous nausea or vomiting, or both. The patients were considered to have spontaneous nausea or vomiting at presentation only if either symptom (or both) occurred acutely and before the administration of morphine for chest pain or clopidogrel loading. Retching (dry heaves) was considered equivalent to vomiting. Every patient with vomiting also had nausea.

Our null hypothesis was that the proportion of patients with nausea or vomiting would be similar in the patients presenting with inferior or anterior AMI. We tested this hypothesis using Fisher's exact tests.[7] To compare the categorical variables for patients with inferior versus anterior AMI, we also used Fisher's exact tests. To compare the continuous variables, we used group *t* tests when the data were normally distributed[7] or Mann-Whitney *U* tests when

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Table 1
Baseline characteristics

Characteristic	AMI Location		p Value
	Inferior (n = 108)	Anterior (n = 72)	
Age (years)	61.3 ± 14.0	62.1 ± 14.9	0.72
Men	75 (69%)	53 (74%)	0.62
White	86 (80%)	55 (76%)	0.71
Smokers			
Current	46 (43%)	24 (33%)	0.28
Former	26 (24%)	12 (17%)	0.27
Body mass index (kg/m^2)	27.9 ± 5.0	28.5 ± 5.9	0.46
History of			
Hypertension	58 (54%)	45 (63%)	0.28
Hyperlipidemia	66 (61%)	37 (51%)	0.22
Diabetes mellitus	23 (21%)	18 (25%)	0.59
Previous AMI or coronary disease	26 (24%)	21 (29%)	0.49
Peripheral arterial disease	10 (9%)	6 (8%)	1.00
Cerebrovascular disease	10 (9%)	7 (10%)	1.00
Chronic kidney disease	7 (6%)	5 (7%)	1.00

Data are expressed as mean ± SD or number (%).

Table 2
Outcomes in the study population by AMI location

Outcome	AMI Location		p Value
	Inferior (n = 108)	Anterior (n = 72)	
Troponin I (ng/ml)			
Initial	0.3 (0–4.2)	0.8 (0.2–12.7)*	0.08
Peak	91.8 (10.2–269.8)	251.0 (43.0–>500)	0.02
Creatine kinase (IU/L)			
Initial	133 (82–234)†	143 (85–326)	0.65
Peak	1,004 (299–2,297)	1,820 (463–3,616)	0.02
Creatine kinase-MB (ng/ml)			
Initial	4.2 (2.1–13.1)†	3.6 (2.0–19.1)	0.80
Peak	105.8 (28.3–222.5)†	137.4 (51.6–376.7)	0.07
Hospital stay (days)	3 (2–5)	4 (3–5)	0.15
Treatment received			
Percutaneous intervention	105 (97.2%)	70 (97.2%)	1.00
Coronary bypass	3 (2.8%)	2 (2.8%)	1.00
Death	2 (1.9%)	4 (5.6%)	0.22

Data are expressed as median (interquartile range) or number (%).
* n = 71.
† n = 107.

the data were not normally distributed.[8] Two-tailed p values <0.05 were considered statistically significant.

Results

Of the 180 patients with AMI, 108 (60%) were diagnosed with inferior wall AMI and 72 (40%) with anterior wall AMI. The baseline characteristics of the patients with inferior and anterior wall AMI were similar (Table 1).

Of the patients in the inferior AMI group, 94% presented with chest pain compared to 90% in the anterior AMI group (p = 0.38). As listed in Table 2, the peak serum troponin I and creatine kinase concentrations were significantly greater in those with anterior AMI than in those with inferior AMI.

The vast majority of patients in each infarct group were treated using an early percutaneous coronary intervention strategy (Table 2). The median length of hospitalization and overall hospital mortality rates were numerically greater for patients with anterior AMI than for those with inferior AMI, but these differences were not statistically significant (Table 2).

Nausea was reported at clinical presentation in 64% of all patients with AMI (95% confidence interval 57% to 71%). Of the patients with inferior AMI, 69% presented with nausea compared to 57% of the patients with anterior AMI (Table 3). The difference in the incidence of nausea in those with inferior compared to those with anterior AMI was not statistically significant (p = 0.11).

Vomiting was reported at presentation in 31% of all patients with AMI (95% confidence interval 24% to 38%). Of the patients with inferior AMI, 33% presented with vomiting compared to 26% of those with anterior AMI (Table 3). The difference in the incidence of vomiting in those with inferior versus anterior AMI was not statistically significant (p = 0.41)

Table 3
Nausea and vomiting on presentation stratified by AMI location

Symptom	AMI Location		p Value
	Inferior (n = 108)	Anterior (n = 72)	
Nausea	75 (69%)	41 (57%)	0.11
Vomiting*†	36 (33%)	19 (26%)	0.41

* All patients who reported vomiting also reported nausea.
† Retching (dry heaves) was classified as vomiting.

Discussion

Two previous studies of the relation of nausea or vomiting to infarct location evaluated not only STEMI, but also those with non–STEMI.[3,4] In both studies, nausea or vomiting occurred much more commonly in patients with STEMI than in those with non–STEMI. These observations led to the speculation that a stimulus for nausea and vomiting might be located in the epicardial (subpericardial) portion of the left ventricle and not in than the subendocardial portion.[4]

Nausea and vomiting can be prominent symptoms in patients experiencing a "silent" AMI (i.e., AMI not accompanied by precordial pain). It has been reported that ≤1/3 of patients presenting with AMI do not report chest pain and that this group has a greater mortality rate than those reporting chest pain.[9] Because >90% of patients in our study reported chest pain, it was difficult to reach any conclusions regarding "silent" presentations.

Nausea was reported in nearly 2 of every 3 patients admitted to our community hospital with AMI associated with either ST-segment elevation or LBBB, and vomiting was reported in nearly 1 of every 3 patients. Although a slightly greater numeric incidence of nausea and vomiting was recorded for the inferior than for the anterior AMI

Table 4
Comparison of previous studies[1–4] and present study for incidence of nausea and/or vomiting in patients with inferior versus anterior acute myocardial infarction (AMI)

Year	Country	Population	Patients (n)	Outcome	Inferior AMI	Anterior AMI	p Value
1978	United States[1]	AMI	62	Nausea and/or vomiting*	69%	27%	<0.001
1980	England[3]	Q wave, AMI	58	Vomiting only	31%	58%	<0.05
1987	United States[4]	Q wave, AMI	94	Nausea and/or vomiting*	51%	66%	0.14
2001	Croatia[2]	First STEMI	1,646	Nausea	59%	41%	NS†
				Vomiting	28%	19%	NS‡
2009	United States (present study)	STEMI or AMI with LBBB	180	Nausea	69%	57%	0.11
				Vomiting	33%	26%	0.41

* Reported as composite of nausea and vomiting and not as separate variables.
† Odds ratio, inferior vs noninferior, 2.01 (95% confidence interval 1.64–2.46).
‡ Odds ratio, inferior vs noninferior, 1.55 (95% confidence interval 1.22–1.97).
AMI = acute myocardial infarction; LBBB = left bundle branch block; NS = not stated; STEMI = ST-segment elevation AMI.

group, the differences were not statistically significant and were probably of little clinical relevance. Table 4 lists our findings and those from the 4 previous studies on this topic,[1–4] although the previous studies differed somewhat in study design and method from our study.

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