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Research paper

Gender difference in the use of coronary interventions for patients with acute coronary syndrome: Experience from a major metropolitan hospital in Melbourne, Australia

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At the conclusion of this article a Continuing Professional Development activity is attached

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ABSTRACT

Background: Literature suggests an ongoing gender disparity in the use of coronary angiography and subsequent interventions among patients with acute coronary syndrome (ACS).
Objectives: The study aimed to examine gender differences in the use of coronary interventions amongst patients with acute coronary syndrome (ACS) admitted to a major metropolitan hospital in Melbourne during the period 2009–2012.
Methods: We undertook a retrospective analysis of a hospital database of 2096 ACS patients. ACS included unstable angina (UA), ST-segment-elevation myocardial infarction (STEMI) and non-STEMI (NSTEMI).
Results: The mean age of the patients was 64.3 years and 624 (30%) were women. Half of them were diagnosed as NSTEMI, 23% as STEMI and 25% as UA. Compared to men, women were older at admission, less likely to be diagnosed with STEMI and less likely to smoke. No gender difference was observed for severe co-morbidities or use of coronary angiography. Women diagnosed with STEMI were 39% less likely to receive an angioplasty stent (adjusted odds ratio 0.61, 95% confidence intervals 0.39–0.96) and 66% less likely to receive grafts (adjusted OR 0.34, 95% CIs 0.13–0.93). Women diagnosed with NSTEMI were 44% less likely to receive grafts (adjusted OR 0.56, 95% CIs 0.37–0.83). Younger women aged 35–49 years were less likely to receive an angioplasty stent, and older women >50 years were less likely to receive grafts.
Conclusions: Adherence to guideline based treatment will help to ensure knowledge translation from guideline to practice. Further research investigating symptom presentation, use of non-invasive tests and medical management of ACS by gender may further explain gender difference for coronary interventions.
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1. Introduction

Coronary angiography is often referred to as the 'gold standard' for the investigation of acute coronary syndrome (ACS).[1] Coronary angiography provides an opportunity to evaluate coronary plaque present in coronary arteries and determine the degree of stenosis. If appropriate, reperfusion using percutaneous coronary intervention (PCI), usually followed by the insertion of a coronary stent, or

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coronary artery bypass graft (CABG) can be performed, thus reducing the risk of subsequent cardiac events.[2,3]

According to the Australian guidelines, all patients diagnosed with ST-segment-elevation myocardial infarction (STEMI) should have a reperfusion strategy implemented immediately if their onset of symptoms is within 12 h and the facility to perform invasive procedure is available; coronary angiography is the diagnostic choice and depending on the extent of the coronary plaque invasive reperfusion is the treatment of choice.[4] In situations in which patients with STEMI present after 12 h of symptom onset or the facility is not readily available specifically in rural or remote areas, reperfusion with fibrinolytics would be indicated.[4] The management of patients with non-STEMI (NSTEMI) and unstable angina (UA), involves stratifying risk using a paradigm which relies largely on the presence of pain combined with changes on ECG or elevated troponin.[4] Amongst the high-risk NSTEMI and UA patients, all except those with severe co-morbidities should be managed like STEMI patients; they should receive invasive coronary interventions as well as aggressive medical management.[4] Treatment of low-risk NSTEMI and UA, however, may employ either an early invasive approach or a conservative strategy, which involves only medical therapy.[5] Several major studies (FRISC II; RITA 3; TACTICS-TIMI 18) have sought to investigate the advantages of an early invasive versus conservative approach.[6–10] Findings from a meta-analysis of these and other studies indicated that an invasive strategy did not appear to benefit women substantially in the absence of troponin elevation.[11] Consequently, the American Heart Association (AHA) guidelines currently recommend a conservative approach for low-risk women,[2] whereas Australian[4] and European guidelines[1] remain non-gender specific.

Whilst the use of coronary interventions for males and females of all ages has markedly increased in the last twenty years, knowledge translation from guideline to clinical practice remains suboptimal. Several studies continue to report lower rates of coronary angiography in women than men, and this has been substantiated by a systematic review.[12] A recent study involving 39 Australian hospitals found a disparity in the use of coronary angiography by gender, concluding that Australian women diagnosed with high-risk NTSEMI and/or UA were 26% less likely than men in the same high-risk category to receive a coronary angiography and that represented an under-investigation amongst women.[13] The underuse of coronary angiography in women may be directly related to the gender difference in the use of secondary prevention medications in women, and this disparity amounts to a form of sexual discrimination.[12,14] Others have suggested that biological 'sex-based' differences in the pathophysiology and aetiology of heart disease in women may be at the root of the gender bias in the treatment of ACS.[15,16] It is also possible that guideline adherence could result in less frequent use of coronary angiography in women. For example, if women with NSTEMI or UA are more likely than men to fall into the low-risk category or to have severe co-morbidities; this would result in less frequent use of coronary angiography in women compared to men.[17,18]

We, therefore, aimed to investigate whether gender differences existed in the use of coronary angiography and revascularization (PCI with stent and CABG) for patients admitted to a major metropolitan hospital in Melbourne with a primary diagnosis of ACS during the period 2009–2012 and, if so, to investigate factors related to such differences.

2. Methods

We performed retrospective analyses of a database of patients with a primary diagnosis of ACS who were treated at a major metropolitan hospital in Melbourne between January 2009 and December 2012. The institution is a large tertiary public hospital with 848 beds. The hospital provides inpatient and outpatient services as well as research and training for health staff. Apart from those needing to attend to the Emergency Department for any initial presentation, cardiac patients attend to cardiac outpatients, inpatients or day-procedure clinics for any planned diagnostic or therapeutic interventions.

Patients admitted to the study hospital for the first time during the selected time period with a primary diagnosis of ACS ($n = 2096$) were included as the study population. No age exclusions were applied. From the database, we extracted information on demographics (date of birth, gender, postcode, catchment area), admission (first admission to the hospital, age on admission, admission department, admission date and time, diagnoses including primary diagnosis, length of stay), discharge (discharge date and time, discharge destination), coronary interventions (first or subsequent coronary angiography, PCI with stent, total number of CABG), co-morbidities (including Charlson and Elixhauser score) and risk factors of ACS (smoking including current and ex-smokers, hypertension, dyslipidaemia, obesity, and diabetes). The following data definitions were used:

2.1. ACS

ACS included primary diagnosis of unstable angina (UA), ST-segment elevation myocardial infarction (STEMI) or non-ST-segment elevation myocardial infarction (NSTEMI) using the following ICD-10 codes: UA (I20.0), STEMI (I21.0, I21.1, I21.2, I21.3), NSTEMI (I21.4).

2.2. Risk factors of ACS

For risk factors of ACS, we used the following ICD-10 codes: current smoking (Z72.0), ex-smoking (Z86.43), hypertension (401.0, 401.1, 401.9, 405.01, 405.12, 405.91, 405.99, 416.0, 572.3, 642.23, 642.24, 642.72, 642.93, I10, I15.0, I15.1, I15.2, I15.8, I15.9, I27.0, I27.2, K76.6, O16), dyslipidaemia (272.2, 272.4, E78.2, E78.4, E78.5, T46.6, Y52.6), obesity (278.0, 278.00, 278.01, E66.0, E66.1, E66.2, E66.8, E66.9), diabetes (E10-, E11-, E12-, E13-, E14-).

2.3. Charlson co-morbidities

Charlson co-morbidity scores[19] were initially calculated by allocating one point for each of: myocardial infarction (MI), congestive heart failure (CHF), peripheral vascular disease (PVD), cerebrovascular disease, dementia, chronic obstructive pulmonary disease (COPD), connective tissue disease, peptic ulcer disease, diabetes mellitus (1 point uncomplicated, 2 points if end-organ damage); two points for each of: moderate to severe chronic kidney disease, hemiplegia, leukaemia, malignant lymphoma; and, solid tumour (2 points, 6 points if metastatic), liver disease (1 point mild, 3 points if moderate to severe) and AIDS (6 points). Subsequently, MI was our primary diagnosis, and to be consistent with another recent study, MI was excluded from the index score.[20] Co-morbidity scores were then categorised into normal (scoring 0), moderate (scoring 1), severe (scoring 2) or very severe (scoring ≥ 3).

2.4. Elixhauser co-morbidities

A series of 28 co-morbidities were included for Elixhauser co-morbidities.[21] We calculated degree of sickness by denoting 1 point for each of Elixhauser co-morbidities and then combining the scores. We categorised the degree of sickness by Elixhauser co-morbidities into normal (scoring 0), moderate (scoring 1), severe (scoring 2) and very severe (scoring ≥ 3).

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