Oxygen therapy for acute ST-segment-elevation myocardial infarction

A randomised controlled trial in Australia found that administering inhaled oxygen did not limit damage to heart muscle and could be associated with an increase in muscle damage in people with acute ST-segment-elevation myocardial infarction.

Overview:

- Supplemental oxygen therapy did not result in less heart damage in people with ST-segment-elevation myocardial infarction (STEMI), as measured by cardiac troponin I levels.
- Creatinine kinase results suggest that oxygen therapy could be associated with an increase in muscle damage during STEMI.
- This new evidence supports NICE guidance that oxygen should not be routinely administered to people with suspected STEMI.

Background: ST-segment-elevation myocardial infarction (STEMI) occurs when a coronary artery becomes blocked by a blood clot. Such a blockage can cause the heart muscle supplied by the artery to die from lack of oxygen and nutrients (ischaemia).

Some studies suggest that oxygen therapy could be beneficial as initial treatment in people with STEMI (Stavitsky et al. 1998). Other analyses, such as a recent Cochrane review (Cabello et al. 2013), do not support the use of oxygen therapy in people with STEMI and suggest that it may even be harmful.

Current advice: The NICE guideline on chest pain of recent onset (currently being updated) recommends that people with chest pain should be referred to hospital as an emergency if an acute coronary syndrome, such as STEMI, is suspected.

The guideline advises that oxygen should not be routinely administered to people with suspected acute coronary syndrome. Oxygen saturation should be monitored using pulse oximetry as soon as possible, ideally before hospital admission. Supplemental oxygen should only be offered to:

- people with oxygen saturation of less than 94% who are not at risk of hypercapnic respiratory failure, aiming for oxygen saturation of 94–98%

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people with chronic obstructive pulmonary disease who are at risk of hypercapnic respiratory failure, to achieve a target oxygen saturation of 88–92% until blood gas analysis is available.

The NICE pathway on acute coronary syndromes brings together all related NICE guidance and associated products on the conditions in a set of interactive topic-based diagrams.

**New evidence:** A randomised controlled trial in Australia by Stub et al. (2015) compared supplementary oxygen therapy with no oxygen therapy in people with STEMI. The Air Versus Oxygen in Myocardial Infarction (AVOID) study recruited people with chest pain who had signs of STEMI according to electrocardiogram by a paramedic.

People randomised to the oxygen therapy group received supplemental oxygen therapy at 8 litres/min by face mask in the ambulance until after transfer from the cardiac catheterisation laboratory to the cardiac unit. People randomised to the no oxygen therapy group did not receive oxygen unless oxygen saturation fell below 94%. These people received oxygen by nasal cannula (4 litres/min) or face mask (8 litres/min) until saturation reached 94%. All patients received 300 mg oral aspirin from paramedics.

STEMI was assessed by a doctor on arrival at the hospital and confirmed using angiography. Additional antiplatelet therapy and choice of anticoagulation and intervention strategy were at the discretion of the treating cardiologist.

Blood samples were taken from participants at baseline and then every 6 hours for the first 24 hours and every 12 hours up to 72 hours. The primary outcome was extent of heart muscle damage (size of myocardial infarct), assessed using peak concentration of cardiac troponin I and creatinine kinase (cardiac biomarkers released as a result of heart damage).

A total of 638 people were randomised by paramedics to oxygen therapy (n=318) or no oxygen therapy (n=320). The primary outcome was assessed in 441 patients in whom STEMI was confirmed (n=218 in the oxygen group and n=223 in the no oxygen group).

Peak concentration of cardiac troponin I was similar in the oxygen group (57.4 microgram/litre) and the no oxygen group (48.0 microgram/litre; ratio of means=1.20, 95% confidence interval [CI] 0.92 to 1.55, p=0.18). Peak creatinine kinase concentration was higher in the oxygen group (1948 U/litre) than in the no oxygen group (1543 U/litre; ratio of means=1.26, 95% CI 1.05 to 1.52, p=0.01).

At hospital discharge, the mortality rate was not statistically different in the oxygen group (1.8%) and the no oxygen group (4.5%; p=0.11). However, people in the oxygen group had a higher rate of in-hospital recurrent myocardial infarctions (5.5% versus 0.9% in the no oxygen group; p=0.006) and cardiac arrhythmias (40.4% versus 31.4%; p=0.05).

Limitations of this study include that it did not have enough statistical power to produce robust results for the clinical outcomes of mortality, recurrent myocardial infarction and cardiac arrhythmia. In addition, paramedics, patients and hospital cardiology teams were not blinded to treatment allocation.

Commentary by Professor Tom Quinn, Professor of Nursing, Faculty of Health, Social Care and Education, Kingston University London and St George’s, University of London:

“Giving supplementary oxygen to people who have myocardial infarction has been commonplace for decades, despite a randomised trial by Rawles et al. in 1976 suggesting harm. A Cochrane review published in 2010 (Cabello et al.) also suggested harm and led to changes in professional society guidelines in the USA, Europe and Australasia to reflect existing guidance by the British Thoracic Society.

“This new trial from Australia adds to the suspicion that oxygen may do more harm than good in patients with STEMI. The study found that supplemental oxygen therapy did not result in less heart damage according to cardiac troponin I levels. Instead, the creatinine kinase results suggest oxygen therapy could be associated with an increase in muscle damage during STEMI. No significant difference in major adverse cardiac events (a composite of all-cause mortality, recurrent ischaemia,
repeat revascularisation and stroke) was observed between those randomised to oxygen or air.

“There are several limitations to this study, including lack of reporting of mortality in 14 patients who declined consent following enrolment by paramedics, missing data on peak cardiac troponin I levels in 18, and loss to follow up of 22. In addition, this study was not powered for mortality and focused instead on the surrogate end point of infarct size.

“Undertaking randomised trials in the prehospital ambulance setting is challenging, and very different from recruiting in the ward or clinic. A pragmatic approach, as taken for this study, is appropriate. Exposure of an individual paramedic to a patient with STEMI, the condition of interest in this trial, is infrequent – perhaps once or twice a year. Misdiagnosis is not uncommon – around a quarter of patients enrolled in this trial did not have STEMI confirmed at subsequent cardiological review. And of course blinding is nigh on impossible with this intervention in this clinical setting (although blind assessment of the primary end point was achieved).

“Practice probably should not change on the basis of this study. Current guidance seems appropriate until the results of the much larger DETO2X-AMI study are published. This study currently recruiting in Sweden and is powered for mortality. The findings will hopefully answer the important question of supplemental oxygen therapy for myocardial infarction for patients and clinicians alike.”

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