

Teachable Moment | LESS IS MORE

Chest Pain and Supplemental Oxygen
Too Much of a Good Thing?

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Story From the Frontline

A man in his 60s with hypertension, dyslipidemia, and coronary artery disease presented with intractable retrosternal chest pain. The patient's vital signs were normal and his oxygen saturation on room air was above 95%. The paramedics started him on oxygen by non-



Invited Commentary

breathing face mask and he was transferred to our emergency department where oxygenation was continued. A 12-lead electrocardiogram revealed anterior ST-elevations and a chest radiograph showed no intrathoracic pathology. The patient underwent urgent cardiac catheterization with 2 drug-eluting stents placed in the left anterior descending artery achieving good postprocedure flow. Oxygen was stopped 2 days later. His postprocedure course was complicated by recurrent episodes of nonsustained ventricular tachycardia and atrial fibrillation requiring an amiodarone infusion. A transthoracic echocardiogram performed 24 hours after presentation revealed a left ventricular ejection fraction of 30% to 35%. The ventricular function remained decreased 6 weeks later.

Teachable Moment

The use of supplemental oxygen in acute coronary syndromes was introduced more than a century ago when it was found that oxygen inhalation relieved symptoms of angina pectoris.¹ Indeed, the idea that supplemental oxygen is beneficial in cardiac ischemia makes sense if one assumes it improves oxygen delivery to ischemic myocardial tissue. However, as early as the 1960s, hemodynamic studies suggested that hyperoxia may have deleterious effects on cardiac function, including worsening ischemia, decreased cardiac output, and increased systemic vascular resistance.² Although the mechanism remains unclear, reactive oxygen species-induced coronary vasoconstriction and reperfusion injury from overexpression of oxygen free radicals are believed to contribute.²

Prior to 2015, only 4 trials had evaluated the effects of supplemental oxygen in patients presenting with acute myocardial infarction. Meta-analysis of these 4 trials showed a nonstatistically significant 2-fold higher risk of death in patients with confirmed acute myocardial infarction who received oxygen compared with those who did not.² A recent large trial randomized 638 patients with ST-elevation myocardial infarction to receive supplemental oxygen or no oxygen. Of these patients, 441 were included in the statistical analysis. The primary outcome

of the trial was myocardial injury assessed by serum cardiac troponin I and creatine kinase levels. Patients in the oxygen group had higher in-hospital rates of both recurrent myocardial infarction (5.5% vs 0.9%, $P = .006$) and major cardiac arrhythmias (40.4% vs 31.4%, $P = .05$).³ They also had greater infarct size at 6 months measured by cardiac magnetic resonance imaging (20.3 g vs 13.1 g, $P = .04$).³ These findings imply a number needed to harm from oxygen of 22 patients for recurrent in-hospital myocardial infarction and 11 patients for major cardiac arrhythmias.

The use of oxygen has been shown to be deleterious in many clinical conditions other than acute cardiac emergencies. In chronic obstructive pulmonary disease (COPD), the harms of supplemental oxygen, particularly hypercapnia, are widely known and appreciated. In 2010, a trial that randomized 405 patients with shortness of breath to receive high-concentration vs titrated oxygen. Mortality in the group receiving titrated oxygen was significantly lower than in the high-concentration oxygen group, especially in the subgroup of patients with confirmed COPD (relative risk, 0.22, 95% CI, 0.05-0.91).⁴ Although not explored in this vignette, other clinical conditions where hyperoxia was shown to be harmful include stroke, cardiopulmonary resuscitation, and prematurity.^{1,3,4}

While the most recent American Heart Association guidelines on the management of ST-elevation myocardial infarction only recommend using supplemental oxygen in hypoxemic patients, supplemental oxygen remains widely used "for comfort" or to treat dyspnea in patients with normal oxygen saturations.⁵ Given the frequency of oxygen overuse, it is possible that thousands of patients are harmed by excess use of oxygen. Despite having normal oxygenation, our patient was given supplemental oxygen by multiple practitioners. It may be reasonable for first responders to empirically administer supplemental oxygen to a patient presenting with undifferentiated cardiorespiratory complaints; however, once the diagnosis is established and normal oxygen levels are confirmed, the common practice of keeping oxygen "for comfort" in nonhypoxic patients is questionable. For our patient, it is possible that some of his episodes of nonsustained ventricular tachycardia and atrial fibrillation were, in part, related to the unnecessary oxygen that he received. Recognizing the potential harms of oxygen therapy, we echo the British Thoracic Society's call to better utilize oxygen as a prescription drug with emphasis placed on its specific indications and contraindications.⁵

ARTICLE INFORMATION

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