Letter to the Editor

Should All Patients with an Acute Coronary Syndrome Be Treated with Oxygen? Time to Reconsider the Evidence and Current Practice

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Oxygen (O_2) has been used in the treatment of myocardial infarction (MI) for over 100 years, in the hope that increased O_2 delivery to the ischaemic myocardium might counterbalance the effects of reduced blood flow and thus improve patient symptoms, reduce the size of the MI, and improve clinical outcomes. Evidence in support of this approach was derived primarily from animal models and uncontrolled human studies. There are also data that O_2 administration may reduce the incidence of unsuspected hypoxaemia in patients with acute coronary syndromes (ACS).[1,2]

This line of reasoning, however, has been greatly weakened by studies showing possible untoward effects of hyperoxaemia on both the systemic and the coronary circulation. In patients with MI, hyperoxaemia causes vasoconstriction and increases systemic vascular resistance, thus reducing cardiac output. Therefore, high flow O_2 administration may not increase tissue oxygen delivery in non-hypoxaemic patients, as the increase in O_2 content is offset by the reduction of cardiac output.[3]

In patients with stable coronary artery disease, inhalation of 100% O_2 for 10-15 min is associated with a decrease in coronary blood flow by 20-30% through constriction of the microvascular resistance vessels. It is possible that coronary vasoconstriction occurs because oxidative degradation of coronary endothelium-derived nitric oxide is accelerated by reactive oxygen species.[4] Additionally, several other mechanisms for hyperoxic coronary vasoconstriction have been reported, including closure of adenosine triphosphate-sensitive potassium channels, direct action on oxygen-sensitive L-type calcium channels in vascular smooth muscle cells, and induction of increased levels of the potent vasoconstrictors endothelin-1 and 20-HETE.[5]

Another concern with O_2 therapy is that hyperoxaemia resulting from the administration of high concentration O_2 may exacerbate reperfusion injury to the heart, owing to the increased production of free oxygen radicals.[6] This may be particularly relevant to the current therapeutic goal in patients with ST-elevation MI of achieving urgent reperfusion of the ischaemic myocardium by restoration of coronary blood flow via thrombolysis or percutaneous coronary intervention.

Moreover, there is evidence from small, randomised clinical trials that the routine use of high-flow O_2 in uncomplicated MI may, instead of improving clinical outcomes, increase infarct size and the risk of mortality.[7,8] Recent overviews of the existing literature underline the urgent need for randomised controlled trials that

would be sufficiently powered to evaluate the effect of O_2 therapy on the risk of mortality in patients with MI.[5,9]

To allow for these concerns, the current guidelines suggest some major changes in the way O_2 is administered in acute disease. Routine administration of high flow O_2 for all acutely ill patients should clearly be abandoned and be replaced by judicious O_2 administration guided by pulse oximetry.[10-12] Therefore, O_2 should be administered only to hypoxaemic patients with oxygen saturation below 90%, and O_2 flow should be targeted to achieve an O_2 saturation of 94-98% (or 88-92% if the patient is at risk of hypercapnic respiratory failure).

In the era of evidence-based medicine it is not uncommon for long standing perceptions and practices to be reconsidered or abandoned under the pressure of newer evidence. It is important for healthcare practitioners to be aware of these changes in the management of ACS.

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