
We thank Dr Esquinas and colleagues for their interest in our study and for their important remarks.

First, our criteria for noninvasive ventilation (NIV) weaning were: disappearance of dyspnea; FiO_2 ≤0.4, continuous positive air pressure (CPAP) ≤4 cmH_2O, and PaO_2 ≥100 mmHg for CPAP mode; or FiO_2 ≤0.4, pressure support level ≤4 cmH_2O, positive end-expiratory pressure ≤4 cmH_2O, and PaO_2 ≥100 mmHg for bilevel-positive airway pressure (bilevel-PAP) mode; plus improvement in clinical condition. Still?

Second, bilevel-PAP mode in the acute myocardial infarction (AMI) group was performed as pressure support only at 3.5 cmH_2O plus expiratory PAP. As noted, our findings could mean that the increase in intrathoracic pressure, but not the mode of ventilation (CPAP or bilevel PAP), is important in patients with cardiogenic pulmonary edema. However, we did not describe this hypothesis because few patients used bilevel-PAP mode (11.3% of the AMI group).

Third, reperfusion treatment with percutaneous coronary intervention (PCI), as possible as early, is mandatory in complicated AMI patients with pulmonary edema. NIV is preferable in this clinical setting because it can be initiated faster than mechanical ventilation with endotracheal intubation. We must be aware of the possibility of the development of more distress or shock, but that is unlikely and reperfusion therapy should not be delayed because of it. Early reperfusion and oxygenation might prevent the development of shock. We argue that NIV in AMI patients with pulmonary edema is a good indication, even during PCI.

Fourth, the CCU mortality rate tended to be higher in the AMI group than in the non-AMI group. Assessing mortality as an endpoint in patients with acute cardiogenic pulmonary edema treated with NIV may lead to many misunderstandings. Patients presenting with pulmonary edema with or without MI may experience serious adverse events related to the disease itself. Among patients with acute decompensated heart failure, short-term mortality is generally higher with AMI than with non-AMI etiologies. Treatment with NIV may affect short-term mortality, but not CCU or overall hospital mortality rates.

Fifth, late requirement for endotracheal intubation after weaning from NIV was significantly more frequent in the AMI group. However, in both groups, for all the patients requiring intubation, more than 30 h had elapsed after weaning from NIV. So we do not consider the requirement for intubation as a failure of NIV. However, as noted by Dr Esquinas and colleagues, there were too few events to infer this data.

Finally, patients with low blood pressure in acute decompensated heart failure are generally complicated by dominancy of low cardiac output rather than pulmonary edema. The efficacy of NIV might be limited in that setting and we would therefore use NIV cautiously in cardiogenic pulmonary edema patients with low blood pressure.

Disclosures

None.

References


Takeshi Yamamoto, MD
Division of Intensive and Cardiovascular Care Unit, Department of Cardiovascular Medicine, Nippon Medical School Hospital, Tokyo, Japan

Shinhiro Takeda, MD
Division of Intensive and Cardiovascular Care Unit, Department of Anesthesiology and Intensive Care, Nippon Medical School Hospital, Tokyo, Japan

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