Anaesthesiology Intensive Therapy
2016, vol. 48, no 1, 61–62
ISSN 1642–5758
10.5603/AIT.2016.0010
www.ait.viamedica.pl

Cut-off point for switching from non-invasive ventilation to intubation in severe ARDS. Still a spectrum of greys and whites

Szymon Skoczyński[1], Antonio M. Esquinas[2]

[1]Department of Pneumology, School of Medicine in Katowice, Poland
[2]Intensive Care Unit, Hospital General Universitario Morales Meseguer, Murcia, Spain

Dear Editor,

Acute respiratory failure (ARF) and aspiration pneumonia were the most frequently observed respiratory complications after acute heroin overdose requiring endotracheal intubation (ETI) and intensive care unit (ICU) admission [1]. In some selected cases of drug overdose, noninvasive ventilation (NIV) could have avoided associated complications [2].

We were fortunate to have had a chance to read an article entitled "Swift recovery of acute hypoxemic respiratory failure under non-invasive ventilation" by Pichot *et al.* [3]. The authors present interesting evidence regarding the possible implementation of NIV in a patient presenting acute respiratory failure (ARDS) with extremely impaired PaO_2/FiO_2 ratio. It is important to underline that the report provides new potential indication in highly selected patients which, in special circumstances, it is possible to treat ARDS with the use of NIV with high positive end expiratory pressure (PEEP) and FiO_2.

However, although the dynamic changes of clinical status during treatment were explained in the article, we consider that there are some factors to take into account in order to consolidate this observation in unconscious patients, those suspected of cocaine intake and with severe impairment of arterial blood gases (pH = 7.19, $PaCO_2$ = 69 mm Hg, PaO_2 = 57 mm Hg, SaO_2 = 84%), precise more precisely the precautions that should have been considered by the authors.

Firstly, regarding equipment and interface, the patient was treated with the Drager Evita XL Ventilator, meaning an ICU-dedicated ventilator. From a clinical point of view, in this case it would have been more clinically valuable for readers if the authors had provided data on the interface used. Such features are of particular importance as differences in dead space between oro-nasal, full-face masks and a helmet could influence optimal synchronization and leakage in a patient with severe tachypnea of 30 breathes per minute as reported [4, 5].

Secondly, the authors have concluded that swift recovery was influenced by high PEEP-NIV strategy. However, this strategy has well known risks, namely: a) high PEEP levels induce leaks and gastric leaks and gastric distention and risk of aspiration, b) large tidal volumes not reported by the authors (800–1200 mL), associated with high airway resistance, low respiratory system compliance, and short inspiratory time, all increasing airway pressure and air entering the stomach [6] and c) the stability of oesophageal sphincter pressure (~20––25 cm H_2O in adults) which, in turn, could vary by due to some pathways leading to gastric content aspiration possibly influenced by opiate toxicity. There is still controversy as to whether heroin-opioids may increase the risk of pulmonary aspiration by decreasing the pressure of oesophageal sphincter-intragastric pressure and hemodynamic compromise (supraventricular arrhythmia).

Thirdly, it is important to underline that the necessary high levels of FiO_2 could also worsen alveolar damage and surfactant production. In this case, the FiO_2 level was lower than 0.60, this figure being the lung toxicity cut off point after 08:45 hours of treatment [7].

Fourthly, the definition of "swift time period of 10 hours is still not broadly accepted. We know from previous studies, that an inability to improve PaO_2/FiO_2 after 1 hour of NIV was a predictor of treatment failure [8, 9]. We consider that for an appropriate extrapolation, it could be necessary to take into account other non-pulmonary factors as neurologic conditions and precise drugs.

Lastly, amiodarone infusion in a patient without severe cardiac arrhythmia with shock seems to be controversial. On the basis of the presented case history, we can assume that the patient was free of dangerous cardiac arrhythmia and the increased heart rate was probably caused by severe hypoxemia, dyspnoea, agitation and/or opiates reversed by naloxone treatment. In these circumstances, a heart rate (HR) of 171 and blood pressure (BP) of 141/71 mm Hg should have been diagnosed as physiological sinus tachycardia, indicating that the improvement of oxygenation should have been effective first line treatment [10]. Moreover, amiodarone with its alveolar toxicity may cause further lung damage and a poorer prognosis [11].

In conclusion, it is necessary to emphasize that although this case report gives one important data on ARDS treatment under strict supervision in the ICU, NIV is contraindicated

in severe ARDS patients who are unable to protect their airways' from aspiration. Finally, the swift time period is still controversial.

ACKNOWLEDGEMENTS

1. The authors declare no financial disclosure.
2. The authors declare no conflict of interest.

References:

1. *Grigorakos L, Sakagianni K, Tsigou E, Apostolakos G, Nikolopoulos G, Veldekis D:* Outcome of acute heroin overdose requiring intensive care unit admission. J Opioid Manag 2010; 6: 227–231.
2. *Ridgway ZA, Pountney AJ:* Acute respiratory distress syndrome induced by oral methadone managed with non-invasive ventilation. Emerg Med J 2007; 24: 681.
3. *Pichot C, Petitjeans F, Ghignone M, Quintin L:* Swift recovery of severe acute hypoxemic respiratory failure under non-invasive ventilation. Anaesthesiol Intensive Ther 2015; 47: 138–142. doi: 10.5603/AIT.a2014.0053.
4. *Nava S, Navalesi P, Gregoretti C:* Interfaces and humidification for noninvasive mechanical ventilation. Respir Care 2009; 54: 71–84.
5. *Storre JH, Bohm P, Dreher M, Windisch W:* Clinical impact of leak compensation during non-invasive ventilation. Respir Med 2009; 103: 1477–1483.
6. *De Keulenaer BL, De Backer A, Schepens DR, Daelemans R, Wilmer A, Malbrain ML:* Abdominal compartment syndrome related to noninvasive ventilation. Intensive Care Med 2003; 29: 1177–1181.
7. *Aggarwal NR, Brower RG:* Targeting normoxemia in Acute Respiratory Distress Syndrome may cause worse short-term outcomes because of oxygen toxicity. Ann Am Thorac Soc 2014; 11: 1449–1453. doi: 10.1513/AnnalsATS.201407-297PS.
8. *Antonelli M, Conti G, Moro ML et al.:* Predictors of failure of noninvasive positive pressure ventilation in patients with acute hypoxemic respiratory failure: a multi-center study. Intensive Care Med 2001; 27: 1718–1728.
9. *Antonelli M, Conti G, Esquinas A et al.:* A multiple-center survey on the use in clinical practice of noninvasive ventilation as a first-line intervention for acute respiratory distress syndrome. Crit Care Med 2007; 35: 18–25.
10. *Blomström-Lundqvist C, Scheinman MM, Aliot EM et al.:* American College of Cardiology; American Heart Association Task Force on Practice Guidelines; European Society of Cardiology Committee for Practice Guidelines. Writing Committee to Develop Guidelines for the Management of Patients with Supraventricular Arrhythmias. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias--executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients with Supraventricular Arrhythmias). Circulation 2003; 108: 1871–1909.
11. *Dhokarh R, Li G, Schmickl CN et al.:* Drug-associated acute lung injury: a population-based cohort study. Chest 2012; 142: 845–850.

Corresponding author:
Szymon Skoczyński
Department of Pneumology, School of Medicine in Katowice, Medical University of Silesia,
Medyków 14, 40-752 Katowice, Poland
e-mail: simon.mds@poczta.fm

Anaesthesiology Intensive Therapy
2016, vol. 48, no 1, 62–64
ISSN 0209–1712
10.5603/AIT.2016.0011
www.ait.viamedica.pl

Cut-off point for switching from non--invasive ventilation to intubation in severe ARDS. Fifty shades of grey?

Luc Quintin

Department of Physiology, University of Lyon, Lyon, France

Key words: acute respiratory distress syndrome, severe ARDS, non-invasive ventilation

Sir, I would like to thank Drs Skoczyński and Esquinas for their comments. Firstly, let us turn to their secondary points:

1. The initial intention was to pre-oxygenate a severely hypoxic patient before tracheal intubation [1] with a Respironics ventilator in the emergency department (ED), not to manage the whole case under non-invasive ventilation (NIV) with an Evita 4 XL ventilator in the Critical Care Unit (CCU). Nevertheless, the ventilatory discoordination disappeared almost immediately following the initiation of NIV, calling for an *iterative* re-assessment of preconceived strategy.
2. This case was *not* acute respiratory distress syndrome (ARDS), but acute hypoxemic non-hypercapnic respiratory failure: the opacities required by the Berlin definition could not be seen on the chest x-ray taken minutes after admission to the ED.
3. Although the patient was conscious, cooperative and drowsy (Glasgow 14), he was fully able to answer questions, and denied repeatedly having inhaled heroin. Esquinas [2] reported intubation with Glasgow ≤ 11. Thus, unconsciousness is irrelevant.
4. The arrhythmia was not sinus tachycardia, but supraventricular arrhythmia: no P waves were observed on the oscilloscope using a *high*-speed display. Nevertheless, arrhythmia was, presumably, a consequence of hypoxia, a trivial issue not further discussed in the report [3]. Magnesium followed by amiodarone was aimed at isolating, as early as possible upon presentation, a «pure» ventilatory distress *vs*. a combined ventilatory and