Case report: Clinical dilemma — acute on chronic respiratory failure complicated by acute lobar atelectasis

JUSTINE NAYLOR Whitlam Joint Replacement Centre, Sydney, Australia

INTRODUCTION

The adequacy of ventilation is indicated by the partial pressure of arterial carbon dioxide (CO$_2$) (Nunn, 1999). Ventilatory failure, therefore, is signalled by hypercapnia (CO$_2$ > 50 mmHg). Chronic obstructive pulmonary disease (COPD) is the most common cause of chronic hypercapnia (Nunn, 1999); varying degrees of ventilation–perfusion mismatch; increased airway resistance, diffusion limitation, chemoreceptor abnormality and respiratory muscle fatigue conspire to produce this outcome (Nunn, 1999; West, 2003). Exacerbation of COPD poses a significant therapeutic challenge to physiotherapists: the increased respiratory muscle work and gas exchange impairment intuitively undermines tolerance to chest physiotherapy as the latter is associated with increased oxygen (O$_2$) consumption and carbon dioxide (CO$_2$) production (Horiuchi et al., 1997); expiratory flow limitation undermines mucus clearance; depressed chemosensitivity to arterial CO$_2$ and O$_2$ impairs cognition and renders dyspnoea unreliable for indicating deterioration; finally, dependence on hypoxic drive to maintain ventilation requires that the levels of supplemental O$_2$ administered must be conservative. With the clinical minutiae of COPD in mind, this account depicts a familiar clinical scenario which is unlikely to be explored through randomized controlled trials: acute on chronic respiratory failure complicated by acute lobar atelectasis and confusion. The clinical dilemma therefore is compelling: how can the therapist deliver a comprehensive treatment when efficacy is not guaranteed, the risks uncertain and patient consent untenable?

CLINICAL SCENARIO

GK presented to hospital with an exacerbation of COPD with signs and symptoms typical of severe hypercapnic respiratory failure: intractable breathlessness; intolerance to physical stress; constrained expiratory flow; suspected sputum retention; and transient lucidity. On admission he demonstrated a respiratory acidosis (Table 1). He was admitted to the high dependency unit with acute respiratory failure secondary to a chest infection. The infection was on a background of chronic respiratory failure secondary to COPD for which he required continuous home O$_2$. Two weeks earlier, routine spirometry (FEV$_1$/FVC) obtained by his respiratory physician was 0.55/0.83 L
Complicated acute on chronic respiratory failure

(66%). Relevant medical history included myocardial infarction, arterial hypertension, type 2 diabetes, truncal obesity and excessive alcohol consumption. It was established soon after admission that invasive ventilatory measures would not be undertaken.

The medical management Day 0 to Day 4 comprised masked continuous positive airway pressure (CPAP), non-invasive ventilation (BiPAP), antibiotics, intravenous salbutamol and high-dose corticosteroids. Alcohol withdrawal was also managed with medication. Physiotherapy comprised breathing exercises in sitting; the comparatively conservative physiotherapeutic approach possibly explained by the patient’s presentation and apparent unco-operativeness, and by the fact that the treating therapist was a recent graduate.

By Day 5, the patient’s deterioration was evident to all attending the morning ward round (Table 1). The chest X-ray indicated worsening bi-basal atelectasis/consolidation. The intensivist reasoned that intubation alone could be fatal; thus, intensive physiotherapy was considered the last option. The ward round was privy to a discussion about the potential risks and benefits of aggressive physiotherapy in view of this patient’s co-morbidities (AARC, 1991). It was apprehensively agreed that intensive physiotherapy would be tried once the patient and family were informed of the apparent urgency to do so.

Physiotherapy comprising vibrations, percussion and naso-pharyngeal suction was performed in side-lying, initially horizontal, then at 15° head-down. BiPAP remained in situ, but was removed intermittently to permit suction. Cardiovascular indices were monitored throughout. Large volumes of mucus were obtained over a 20-minute period. Similar treatments were performed across two days; improvements in arterial blood gas profile between treatments were evident from Day 5 to Day 7 (Table 1). Notably, each treatment episode was resisted by the patient. On Day 6, arrhythmias were observed during treatment. Plasma electrolytes revealed slight

### TABLE 1: Trends in arterial gas exchange across time

<table>
<thead>
<tr>
<th></th>
<th>Admission (HDU)</th>
<th>Day 0 (Pre-CP)</th>
<th>Day 1 (Post-CP session 1)</th>
<th>Day 5 (Post-CP session 2)</th>
<th>Day 5 (pm)</th>
<th>Day 7 (am)</th>
<th>Day 7 (pm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.09</td>
<td>7.15</td>
<td>7.29</td>
<td>—</td>
<td>7.32</td>
<td>7.4</td>
<td></td>
</tr>
<tr>
<td>PaO₂</td>
<td>73</td>
<td>62</td>
<td>88</td>
<td>67</td>
<td>—</td>
<td>88</td>
<td>75</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>151</td>
<td>123</td>
<td>70</td>
<td>101</td>
<td>—</td>
<td>79</td>
<td>76</td>
</tr>
<tr>
<td>HCO₃</td>
<td>41</td>
<td>34.4</td>
<td>39.8</td>
<td>—</td>
<td>35</td>
<td>46</td>
<td></td>
</tr>
<tr>
<td>BE</td>
<td>—</td>
<td>15</td>
<td>15</td>
<td>95%</td>
<td>95%</td>
<td>94%</td>
<td>93%</td>
</tr>
<tr>
<td>SaO₂/SpO₂</td>
<td>—</td>
<td>81%</td>
<td>90%</td>
<td>86%</td>
<td>95%</td>
<td>95%</td>
<td>94%</td>
</tr>
<tr>
<td>Supplemental O₂ (l/min)</td>
<td>50%</td>
<td>10L</td>
<td>10L</td>
<td>10L</td>
<td>10L</td>
<td>4L (nasal prongs)</td>
<td>6L (mask)</td>
</tr>
<tr>
<td>Ventilatory assistance</td>
<td>CPAP 7.5</td>
<td>BiPAP 6–20</td>
<td>BiPAP 6–20</td>
<td>BiPAP 6–20</td>
<td>BiPAP</td>
<td>Spontaneous</td>
<td>Spontaneous</td>
</tr>
</tbody>
</table>

HDU = high dependency unit; pH = negative log of hydrogen ion; PaO₂ = partial pressure oxygen (arterial blood), mmHg; PaCO₂ = partial pressure carbon dioxide (arterial blood), mmHg; HCO₃ = bicarbonate ion, mmol/L; BE = base excess; SaO₂/SpO₂ = arterial or transcutaneous arterial oxygen saturation; L = litres; CPAP = continuous positive airway pressure, cm H₂O; BiPAP = bi-level positive airway pressure, cm H₂O.
hyponatraemia, hypercalcaemia and hyperkalaemia. Acknowledging the greater propensity for arrhythmias in the presence of abnormal electrolytes (Holt, 2003), treatment time was reduced as was the angle of tilt. By Day 7 the patient tolerated assisted mobilization and could use the Flutter™ to assist mucus removal. The afternoon pulse oximetry reading (SpO₂, 93%) indicated he had maintained his improvement in gas exchange with this more conservative routine. Of note, the improvements in gas exchange across this entire period preceded any radiographic improvements. GK was discharged to a medical ward on Day 10 and to a rehabilitation unit on Day 15.

**CONCLUSION**

The complex pathophysiology of COPD renders it difficult to treat and difficult to predict in terms of its response to treatment. Confounding co-morbidities add to the uncertainty. Physiotherapy is not considered to be effective during acute exacerbations (Hall et al., 2003; Soto and Varkey, 2003). However, the presence of specific lobar involvement renders physiotherapy necessary. Evidence indicating best physiotherapeutic practice in acute COPD with specific lobar involvement is not available. For all these reasons, the dilemma for, and the apparent hesitancy of, the new graduate in this instance is understandable. What is promoted here, therefore, is the importance of a team approach when the correct clinical path is uncertain. A planned yet flexible approach based on interventions known to resolve atelectasis (Berney et al., 2004) initiated the patient’s recovery, but team support and consent by proxy were also critical to the success of the collaboration.

**REFERENCES**


Address correspondence to: Justine M Naylor, Senior Research Fellow, Whitlam Joint Replacement Centre, Fairfield Hospital, Cnr Polding St and Prairievale Rd, Prairievale, 2176 Sydney, NSW, Australia (E-mail: Justine.Naylor@sswhhs.nsw.gov.au).

(Submitted December 2004; accepted July 2005)