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Imipenem/cilastin dosage during acute renal failure and hemofiltration

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Building on data published by MC Vos and colleagues in 1992 on imipenem/cilastatin and CAVHD [1] we investigated imipenem/cilastatin in 16 patients with (n = 10, group 1) and without (n = 6, group 2) renal insufficiency; both groups received continuous, volume-constant hemofiltration (CVHF). The indications for hemofiltration were oliguric renal dysfunction and increases in urea and creatinine of 8 mmol/day and more. The main indications for hémofiltration were oliguric renal failure (CVHF). The indications for hémofiltration were oliguric renal dysfunction and increases in urea and creatinine of 8 mmol/day and more. The main indications for hémofiltration were oliguric renal failure.

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Pharmacokinetics were calculated as a postdilution technique with a polysulfone high-flux filter and an blood flow rate of 200 ml/min and was adjusted to keep PTT at about 40 s. Arterial blood gases were normal and the patient could be extubated. Stridor immediately subsided, however, on fiberoptic bronchoscopy, the vocal cords appeared to be almost completely adducted without edema or signs of inflammation. A minitracheostomy was performed and treatment with continuous-flow CPAP connected to the minitracheostomy was started. This resulted in a marked reduction of the stridor, with adequate ventilation. The patient was successfully weaned from this CPAP arrangement without recurrent laryngospasms and was transferred to the ward after 5 days with minitracheostomy in situ. Additional investigation with body-plethysmography breathing showed a marked increase in inspiratory resistance, with almost normal expiratory resistance, pattern in accordance with extrathoracic, variable airflow obstruction, e.g. vocal cord dysfunction. Episodes of severe stridor recurred after 2 weeks. On direct laryngoscopy, the vocal cords were still adducted. A permanent tracheostomy was performed to avoid further ICU admission and interventions.

In Parkinson's disease, dopamine depletion leads to diminished inhibition of the extrapyramidal motor system. This may lead to severe laryngospasm. Respiratory problems are well known, and aspiration pneumonia is one of the most common causes of death among such patients [1].

References


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Primary laryngospasm in a patient with Parkinson's disease: treatment with CPAP via minitracheostomy following intubation

Received: 4 December 1994  
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Sir: We would like to report on a primary laryngospasm in a patient with Parkinson's disease and its treatment with CPAP (continuous positive airway pressure) via minitracheostomy.

A 60-year-old man with about a 10-year history of Parkinson's disease treated with Sinemet (carbidopa and levodopa) was admitted to the hospital with a life-threatening upper airway obstruction. He was intubated, and mechanical ventilation was initiated. Chest X-ray revealed bilateral basal consolidations. After 3 days, the condition had improved, blood gases were normal and the patient could be extubated. Stridor immediately subsided, however, on fiberoptic bronchoscopy, the vocal cords appeared to be almost completely adducted without edema or signs of inflammation. A minitracheostomy was performed and treatment with continuous-flow CPAP connected to the minitracheostomy was started. This resulted in a marked reduction of the stridor, with adequate ventilation. The patient was successfully weaned from this CPAP arrangement without recurrent laryngospasms and was transferred to the ward after 5 days with minitracheostomy in situ. Additional investigation with body-plethysmography breathing showed a marked increase in inspiratory resistance, with almost normal expiratory resistance, pattern in accordance with extrathoracic, variable airflow obstruction, e.g. vocal cord dysfunction. Episodes of severe stridor recurred after 2 weeks. On direct laryngoscopy, the vocal cords were still adducted. A permanent tracheostomy was performed to avoid further ICU admission and interventions.

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In Parkinson's disease, dopamine depletion leads to diminished inhibition of the extrapyramidal motor system. This may lead to severe laryngospasm. Respiratory problems are well known, and aspiration pneumonia is one of the most common causes of death among such patients [1].
Dysfunction of the upper airways has only recently been recognized in patients with Parkinson's disease [2]. In most cases, this leads to impairment of static and dynamic pulmonary function. In some patients, laryngeal involvement was the main reason for airway obstruction [2]. In our patient, the most likely diagnosis is primary laryngospasm associated with Parkinson's disease.

Treatment by minitracheostomy connected to a continuous flow CPAP resulted in a clinically relevant relief of stridor. The mechanism of this effect might be the slight positive airway pressure of 2–4 cmH₂O in combination with a 4-mm free artificial airway. However, the latter cannot be the only explanation for the clinical relief of stridor, since the stridor increased while the patient was breathing through an open minitracheostomy without CPAP connection. Although minitracheostomy is most frequently used in the treatment of sputum retention, it is used most frequently in patients with Parkinson's disease.

In conclusion, laryngospasm caused by dysfunction of recurrent laryngeal nerves may be associated with Parkinson's disease. CPAP via minitracheostomy proved to be temporarily successful in the management of this problem. Tracheostomy may be inevitable in cases of persistent relapses.

References

5. B. Levy P.E. Bollaert A. Larcan

Inhaled nitric oxide is often efficient in severe ARDS

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Sir: In a recent issue of Intensive Care Medicine, Mira et al. [1] presented a study concerning the lack of efficiency of inhaled nitric oxide in ARDS. They reported a series of six patients with severe ARDS who did not respond to NO, three of whom responded to a subsequent trial of NO. It was suggested that soluble guanylate cyclase of pulmonary vasculature smooth muscle could be unresponsive to NO.

We used inhaled NO in 30 patients with severe ARDS (PaO₂/FiO₂ = 81±8 on FiO₂ 1 PEEP 11±1; LIS = 3.45) [2]. NO (5 ppm) was administered early (mechanical ventilation for 7±2 days). Twenty-eight patients were considered to be NO responders (+20% PaO₂/FiO₂). We did not find any correlation between the improvement in arterial oxygenation and the decrease in mean pulmonary arterial pressure [PAPm = 29±3 at TO and PAPm = 28±2 at T1 hour (NS)]. The 2 NO non-responders had acute hemorrhagic pulmonary edema with refractory septic shock (SAP under 70 mmHg with high doses of epinephrine and norepinephrine). Owing to hemodynamic instability, PEEP levels were low and did not allow for alveolar recruitment. In such a situation, it is not surprising that an inhaled agent might be inefficient.

According to published studies, 30–50% of patients are considered to be responders to inhaled NO [3, 4]. In ARDS, pulmonary hypertension is secondary not only to hypoxic pulmonary vasoconstriction, but also to increased Va/Q abnormality, atelectasis, loss of vascular bed and small vessel obstruction. Given this heterogeneous vascular insult, it does not seem surprising that pulmonary artery pressure was not dramatically changed. Several additional factors may interfere with the efficacy of NO in patients with severe ARDS: reduction or loss of the hypoxic pulmonary vasoconstriction (pulmonary infection, lung trauma, lung hyperinflation) and replacement of actively constricted small pulmonary vessels by fibrotic and irreversibly narrowed pulmonary vessels [5]. The last explanation may be of value for the patients presented by Mira et al. Indeed, as a reference center for LFPPV-ECCO₂R, they may have recruited a higher proportion of end-stage ARDS than we did.

Modifications of pulmonary vasculature in late ARDS associated with large areas of non-ventilated, non-recruitable parenchyma may be responsible for a decreased response to inhaled NO.

Finally, the wide variation in responders to NO inhalation could be related to the patient's pulmonary vascular levels of guanylate cyclase, and also to the degree of pulmonary parenchyma and vascular fibrosis.

References


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