

Treatment of Cardiogenic Pulmonary Oedema by Helmet-delivered Non-invasive Pressure Support Ventilation in Children With Scorpion Sting Envenomation

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Abstract

Introduction: The aim of this study was to evaluate the feasibility of non-invasive positive pressure ventilation through a new interface helmet in the treatment of cardiogenic pulmonary oedema due to scorpion sting envenomation in children. **Clinical Picture:** Three patients presented with fever, and respiratory distress following scorpion sting. Their cardiac enzymes were abnormal. Electrocardiogram (ECG) of 3 patients showed features of myocardial strain with ST elevation. Bedside chest X-ray taken in emergency showed marked bilateral infiltrates suggestive of pulmonary oedema. M-mode, two-dimensional colour-flow Doppler echocardiogram showed left ventricular dysfunction. **Treatment and Outcome:** At paediatric intensive care unit admission, they were treated with antivenom, prazosin (0.03 mg/kg/dose), dopamine (15 mcg/kg/min), dobutamine (10 mcg/kg/min) and nitroprussid (1 mcg/kg/min). Epinephrine (0.1 mcg/kg/min) were added later. They were hypoxic and dyspnoeic. A slight sedation was induced with ketamine and/or midazolam. Non-invasive pressure support ventilation (NPSV) was delivered via the helmet by means of an intensive care unit ventilator. We evaluated the effect of NPSV delivered by helmet on oxygenation, respiratory rate, haemodynamics, complications and outcome. An improvement of oxygenation was observed within 2 hours of treatment. The helmet was well tolerated by all the children. No complications occurred in the 3 patients. **Conclusion:** This new approach of delivering NPSV through a helmet allows the successful treatment of cardiogenic pulmonary oedema in children with scorpion sting envenomation, assuring a good tolerance without complications. Future studies are needed before recommending the extensive application of this technique in all cases of cardiogenic pulmonary oedema due to scorpion sting envenomation.

Ann Acad Med Singapore 2008;37:230-3

Key words: Acute respiratory failure, Child, Helmet interface, Non-invasive ventilation

Introduction

Scorpion stings represent an important and serious public health problem worldwide due to their high incidence and potentially severe and often fatal clinical manifestations, especially among children.^{1,2} The severity of the envenomation is related to haemodynamic and cardiorespiratory alterations, with cardiac failure and cardiogenic pulmonary oedema being the major causes of death.³

Non-invasive positive pressure ventilation (NPPV) via facial mask has been used as an effective early treatment in cooperative patients in whom respiratory failure develops, increasing functional residual capacity.⁴ NPPV should be considered first, given the possibility of avoiding the direct complications of tracheal intubation and conventional ventilation (bleeding, barotrauma, infection), thereby

possibly reducing the high mortality rate related to the use of invasive ventilation.^{5,6} The new helmet interface offers important advantages: an improved tolerability compared to other NPPV interfaces, a fixation system with a lower risk of cutaneous injury and the possibility of fitting it to any patient, regardless of his or her face contour.⁷

To our knowledge, this study is the first report of helmet application in paediatric cardiogenic pulmonary oedema due to scorpion sting envenomation.

Helmet

The helmet (CaStar, Starmed, Italy) is made of transparent latex-free polyvinylchloride; it is secured by 2 armpit braces at 2 hooks (anterior/posterior) on the metallic ring joining the helmet with a soft collar. This collar permits neck adhesion, allowing a sealed connection. The pressure

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increase during ventilation makes the soft collar seal comfortable to the neck and the shoulders, avoiding air leakage. The helmet is available in 3 adult and 1 paediatric size to ensure comfort and a proper seal. The whole apparatus is connected to the ventilator by a conventional respiratory circuit. The 2 ports of the helmet act as inlet (upper) and outlet (lower) of the gas flows. For patients with a nasogastric tube, a specific seal connector placed in the metabolic ring was used to allow the passage of the tube and to avoid air leakage. When a proper size helmet is chosen, the occurrence of air leaks is rare. However, if there are air leaks, the following options are available: i) readjustment of the helmet; ii) changing the size to a smaller one; iii) if clinically compatible, reduction of the level of pressure support and/or positive end-expiratory pressure (PEEP). The transparency of the device allows the children to see and interact with their parents, nurses and the environment. The head of the bed is kept at a 30° to 45° angle. After helmet positioning, pressure support can be increased in increments of 2 to 3 cmH₂O to improve ventilatory performance, as evidenced by oxygen need and respiratory rate decrease, and the disappearance of accessory muscle activity. PEEP is increased from 5 to 12 cmH₂O to assure a peripheral oxygen saturation (SpO₂) of at least 92% with the lowest FiO₂ possible.

Criteria for Intubation

The criteria for endotracheal intubation were the inability to maintain PaO₂/FiO₂ ratio >100 during helmet-delivered non-invasive pressure support ventilation (h-NPSV), deterioration of mental status, haemodynamic instability, intolerance of the technique, or the inability to manage copious secretions, or to correct dyspnoea.

Case Reports

Patient 1

An 18-month-old girl presented with fever, unconsciousness and respiratory distress following a scorpion sting. Examination showed Glasgow Coma Score (GCS) 10, tachypnoea and tachycardia. She had nasal flaring and crepitation rales were heard bilaterally on auscultation.

Laboratory findings revealed: white blood cell 25,000/mm³ with a predominance of polymorph nuclear leukocytes, haemoglobin: 10 g/L and thrombocytes: 300,000/mm³. Analyses of blood urea nitrogen, serum creatinine, electrolytes, calcium, phosphorus, glucose, aspartate aminotransferase, alanine aminotransferase, bilirubin and urine gave normal results. Blood gas analysis revealed pH 7.30, pO₂ 45 mmHg, pCO₂ 35 mmHg, BE-13, HCO₃ 15 mmol/L, and a lactate level of 10 mmol/L. Her cardiac enzymes were abnormal. CPKMB was 115 ng/mL and troponin t (cTnI) was 10 ng/mL. An electrocardiogram (ECG) of the patient showed features of myocardial strain

with ST elevation. Bedside chest X-ray taken in the emergency room showed marked bilateral infiltrates suggestive of pulmonary oedema. M-mode, two-dimensional colour-flow Doppler echocardiogram showed left ventricular dysfunction. Ejection fraction was 0.39 and left ventricular fractional shortening was 20.

We monitored intra-arterial blood pressure and central venous pressure, pulse oximetry and the arterial blood gases. On admission to the Pediatric Intensive Care Unit (PICU), the patient was treated with antivenom, prazosin (0.03 mg/kg/dose), dopamine (10 mcg/kg/min), dobutamine (10 mcg/kg/min), and nitroprusside (1 mcg/kg/min). We subsequently added epinephrine (0.1 mcg/kg/min). She was hypoxic (with PaO₂:FiO₂ of 145-FiO₂ 0.50 on a venture oxygen mask) and dyspnoeic. SpO₂ was 80% to 82%, respiratory rate 55/min, heart rate 175, and blood pressure 70/30 mmHg. A slight sedation was induced with ketamine and h-NPSV (paediatric size) was started on pressure support (initial parameters: PS 10, PEEP 7, FiO₂ 0.90, flow trigger) using a Evita-2 ventilator (Drager, Germany). On h-NPSV, SpO₂ increased and oxygen supplementation was decreased to 0.55, PaO₂:FiO₂ increased from 145 to 225 (2 hours after h-NPSV initiation), respiratory rate decreased from 55 to 39/min and heart rate decreased from 175 to 135 /min. Control blood gas analysis revealed pH 7.45, pO₂ 120 mmHg, pCO₂ 42 mmHg, BE-1, HCO₃ 21 mmol/L, and the lactate level decreased from 10 to 2 mmol/L. Clinical and respiratory improvements were noted after a few days. The patient was weaned off inotropic treatment, and h-NPSV was applied intermittently. Four days after admission, she was taken off the ventilator. Seven days after admission, control ECHO was normal and inotropic drugs were stopped. She was discharged 12 days after admission.

Patient 2

A 7-year-old boy presented with fever, convulsions and respiratory distress following a scorpion sting. Examination showed GCS 12, dyspnoea, tachypnoea and tachycardia. He had nasal flaring and crepitation rales were heard bilaterally on auscultation.

Laboratory findings revealed: white blood cell 35,000/mm³ with a predominance of polymorph nuclear leukocytes, haemoglobin: 9 g/L and thrombocytes: 150,000/mm³. The results of analyses performed on blood urea nitrogen, serum creatinine, electrolytes, calcium, phosphorus, glucose, aspartate aminotransferase, alanine aminotransferase, bilirubin and urine were normal. Blood gas analysis revealed pH 7.23, pO₂ 40 mmHg, pCO₂ 40 mmHg, BE-13, HCO₃ 15 mmol/L, and a lactate level of 10 mmol/L. His cardiac enzymes were abnormal. CPKMB was 103 ng/mL and cTnI was 6 ng/mL. An ECG of the patient showed features of myocardial strain with ST elevation. Bedside chest X-ray taken in the emergency room showed marked bilateral infiltrates suggestive of pulmonary oedema.

M-mode, two-dimensional colour-flow Doppler echocardiogram showed left ventricular dysfunction. Ejection fraction was 0.42 and left ventricular fractional shortening was 23.

We monitored intra-arterial blood pressure and central venous pressure, pulse oximetry and the arterial blood gases. On admission to the PICU, we treated the patient with antivenom, prazosin (0.03 mg/kg/dose), dopamine (15 mcg/kg/min), dobutamine (10 mcg/kg/min), and nitroprusside (1 mcg/kg/min). We subsequently added epinephrine (0.1 mcg/kg/min). He was hypoxic (with PaO₂:FiO₂ of 130-FiO₂ 0.50 on a venture oxygen mask), dyspnoea and pale. SpO₂ was 80% to 84%, respiratory rate 45/min, heart rate 155, and blood pressure 80/40 mmHg. A slight sedation was induced with midazolam and ketamine. h-NPSV (adult small size) was started on pressure support (initial parameters: PS 14, PEEP 9, FiO₂ 0.80, flow trigger) using a Evita-2 ventilator (Dräger, Germany). On NPSV, SpO₂ increased and oxygen supplementation was decreased to 0.50, PaO₂:FiO₂ increased from 130 to 240 (1 hour after h-NPSV initiation), respiratory rate decreased from 45 to 30/min and heart rate decreased from 155 to 108/min. Control blood gas analysis revealed pH 7.43, pO₂ 120 mmHg, pCO₂ 40 mmHg, BE-2, HCO₃ 22 mmol/L, and the lactate level decreased from 10 to 2 mmol/L. Clinical and respiratory improvements were noted after a few days. h-NPSV was continued for 48 hours. The patient was weaned of inotropic treatment. Ten days after admission, control ECHO was normal. He was discharged 12 days after admission.

Patient 3

A 5-year-old girl presented with fever and respiratory distress following a scorpion sting. Examination showed that the patient had dyspnoea, tachypnoea, and tachycardia. She had nasal flaring and crepitation rales were heard bilaterally on auscultation.

Laboratory findings revealed: white blood cell 18,500/mm³ with a predominance of polymorph nuclear leukocytes, haemoglobin: 8 g/L, thrombocytes: 550,000/mm³. The results of analyses on blood urea nitrogen, serum creatinine, electrolytes, calcium, phosphorus, glucose, aspartate aminotransferase, alanine aminotransferase bilirubin and urine were normal. Blood gas analysis revealed pH 7.27, pO₂ 48 mmHg, pCO₂ 23 mmHg, BE-13, HCO₃ 15 mmol/L, and a lactate level of 7 mmol/L. Her cardiac enzymes were abnormal. CPKMB was 153 ng/mL and cTnI was 7.8 ng/mL. An ECG of the patient showed features of myocardial strain with ST elevation. Bedside chest X-ray taken in the emergency room showed marked bilateral infiltrates suggestive of pulmonary oedema. M-mode, 2-dimensional colour-flow Doppler echocardiogram showed left ventricular dysfunction. Ejection fraction was 0.35 and left ventricular fractional shortening was 20.

We monitored intra-arterial blood pressure and central venous pressure, pulse oximetry and the arterial blood gases. On admission to the PICU, we treated the patient with antivenom, prazosin (0.03 mg/kg/dose), dopamine (10 mcg/kg/min), dobutamine (10 mcg/kg/min), and nitroprusside (1 mcg/kg/min). We subsequently added epinephrine (0.1 mcg/kg/min). She was hypoxic (with PaO₂:FiO₂ of 160-FiO₂ 0.50 on a venture oxygen mask), dyspnoea and pale. SpO₂ was 83% to 86%, respiratory rate 48/min, heart rate 158, and blood pressure 75/40 mmHg. A slight sedation was induced with ketamine. h-NPSV (adult small size) was started on pressure support (initial parameters: PS 13, PEEP 8, FiO₂ 0.90, flow trigger) using a Evita-2 ventilator (Dräger, Germany). On NPSV, the SpO₂ increased and oxygen supplementation was decreased to 0.60, PaO₂:FiO₂ increased from 160 to 220 (1 hour after h-NPSV initiation), respiratory rate decreased from 48 to 33/min and heart rate decreased from 158 to 110/min. Control blood gas analysis revealed pH 7.46, pO₂ 135 mmHg, pCO₂ 42 mmHg, BE-1, HCO₃ 22 mmol/L, and the lactate level decreased from 7 to 2 mmol/L. Clinical and respiratory improvements were noted after a few days. We stopped inotropic treatment, and h-NPSV was applied intermittently. Four days after admission she was taken off the ventilator. Nine days after admission, control ECHO was normal. She was discharged 12 days after admission.

Discussion

Scorpion stings represent an important and serious public health problem worldwide due to their high incidence and their potentially severe and often fatal clinical manifestations, especially among children. The life-threatening complications of myocarditis and pulmonary oedema is known in scorpion sting envenomation. The occurrence of cardiac injury in severe cases of scorpion envenomation has been demonstrated for several years in both experimental studies and cases of human envenomation. Its physiopathology, however, has not yet been completely established. The life-threatening complications of myocarditis and pulmonary oedema require urgent attention and intensive care for a period of time, which may range from a few hours to days.⁸ Our patients presented to our paediatric intensive care unit with respiratory distress, hypoxaemia and pulmonary oedema. All our patients reached us 6 to 15 hours after being sting, when their condition had deteriorated at a peripheral hospital.

Invasive mechanical ventilation through an endotracheal tube is a life-saving procedure for acute respiratory failure, but endotracheal intubation increases patients' discomfort and stress, often requires sedation, may cause injuries of the tracheal mucosa and tracheal stenosis,⁵ and represents one of the most important predisposing factors for developing nosocomial pneumonia.⁶ Invasive mechanical

ventilation has been associated with increased morbidity and mortality in patients admitted to the intensive care unit.^{9,10}

NPPV may provide effective ventilatory support in selected patients with acute respiratory failure.¹¹ NPPV can be delivered as continuous positive airway pressure, pressure support ventilation, or volume and pressure cycled systems by means of a nasal or face mask.¹² Both types of masks can reduce the work of breathing in patients with respiratory failure.¹³ However, dyspnoeic patients often breathe through their mouths, causing air leakage and lowering the efficacy of NPPV when nasal mask is used.¹⁴ Therefore, the face mask is preferable in this type of patient because it greatly improves alveolar ventilation.¹² NPPV may fail because of technical problems, such as gas leaks around the mask, skin lesions and mask discomfort.^{14,15} The NPPV helmet is a relatively new interface, developed in Italy.⁷ The new helmet interface permits the delivery of non-invasive ventilatory support from the onset of respiratory failure. Its main advantage over face masks is better patient compliance, fewer complications, and lower nurse workload.^{7,16} The helmet is made of transparent latex-free PVC; it is secured by 2 hooks (anterior/posterior) on the metallic ring joining the helmet with a soft collar. This collar permits neck adhesion allowing a sealed connection. The apparatus is connected to the ventilator by a conventional respiratory circuit. Like other NPPV interfaces, the NPPV helmet can be used for almost every mode of ventilation, provided that the respirator has the appropriate specifications (e.g., leak compensation). Recent studies demonstrated favourable outcomes with the helmet for hypoxaemic and hypercapnic respiratory failure.^{7,17} These authors compared the efficacy of NPPV using the helmet with NPPV through standard facial mask, and showed that this new device allowed the successful treatment of acute respiratory failure, had a higher patient tolerance compared to facial mask, with fewer complications. The helmet was used with continuous positive airway pressure (CPAP) to treat 26 outpatients with cardiogenic pulmonary oedema. CPAP was administered through the helmet connected to a flow oxygen source with mechanical PEEP and improved oxygen saturation, heart rate, mean arterial pressure, respiratory rate, and wet rales score in all patients.¹⁸ We confirmed these results in 3 paediatric patients of scorpion sting envenomation with cardiogenic pulmonary oedema. To our knowledge, this is the first report of helmet application in paediatric cardiogenic pulmonary oedema due to scorpion sting envenomation.

NPPV complications such as aspiration, pain, gastric dilatation and poor patient compliance are common, but no such complications occurred in our patients. One of the disadvantages of the helmet is that respiratory cycle volumes

and pressures cannot be measured routinely. However, we had no difficulty estimating the efficiency of the ventilatory support or progress of the weaning process by monitoring clinical and laboratory variables.

In conclusion, this new approach to delivering NPPV through a helmet allows the successful treatment of cardiogenic pulmonary oedema in children with scorpion sting envenomation, assuring a good tolerance without complications. However, future studies are needed before recommending the extensive application of this technique in all cases of unstable cardiogenic pulmonary oedema due to scorpion sting envenomation.

REFERENCES

- Ghalim N, El-Hafny B, Sebti F, Heikel J, Lazar N, Moustansir R, et al. Scorpion envenomation and serotherapy in Morocco. *Am J Trop Med Hyg* 2000;62:277-83.
- Goyffon M, Vachon M, Broglio N. Epidemiological and clinical characteristics of the scorpion envenomation in Tunisia. *Toxicon* 1982;20:337-44.
- Cupo P, Hering SE. Cardiac troponin I release after severe scorpion envenoming by *Tityus serrulatus*. *Toxicon* 2002;40:823-30.
- Katz JA, Mark JD. Inspiratory work with and without continuous airway pressure in patient with acute respiratory failure. *Anesthesiology* 1985;63:598-607.
- Burns HP, Dayal VS, Scott A, Van Nostrand AW, Bryce PP. Laryngotracheal trauma: observations on its pathogenesis and its prevention following prolonged orotracheal intubation in the adult. *Laryngoscope* 1979;89:1316-25.
- Meduri GU. Non-invasive ventilation. In: Marini J, Slutsky AS, editors. *Physiological Basis of Ventilatory Support: Lung Biology in Health and Disease*. New York: Marcel Dekker, 1998:921-98.
- Antonelli M, Conti G, Pelosi P, Gregoretti C, Pennisi MA, Costa R, et al. New treatment of acute hypoxemic respiratory failure: non-invasive pressure support ventilation delivered by helmet – a pilot controlled trial. *Crit Care Med* 2002;30:602-8.
- Bhadani UK, Tripathi M, Sharma S, Pandey R. Scorpion sting envenomation presenting with pulmonary oedema in adults: a report of seven cases from Nepal. *Indian J Med Sci* 2006;60:19-23.
- Pingleton SK. Complications of acute respiratory failure. *Am Rev Respir Dis* 1988;137:1463-93.
- Chatila WM, Criner GJ. Complication of long-term mechanical ventilation. *Respir Care Clin N Am* 2002;8:631-47.
- International Consensus Conferences in Intensive Care Medicine: non-invasive positive pressure ventilation in acute respiratory failure. *Am J Respir Crit Care Med* 2001;163:283-91.
- Mehta S, Hill NS. Non-invasive ventilation. State of the art. *Am J Respir Crit Care Med* 2001;163:540-77.
- Carrey Z, Gottfried SB, Levy RD. Ventilatory muscle support in respiratory failure with nasal positive pressure ventilation. *Chest* 1990;97:150-8.
- Antonelli M, Conti G. Non-invasive ventilation in intensive care unit patients. *Curr Opin Crit Care* 2000;6:11-6.
- Gregoretti C, Confalonieri M, Navalesi P, Squadrone V, Frigerio P, Beltrame F, et al. Evaluation of patient skin breakdown and comfort with a new face mask for non-invasive ventilation: a multi-center study. *Intensive Care Med* 2002;28:278-84.
- Scandroglio M, Piccolo U, Mazzone E, Agrati P, Aspesi M, Gamberoni C, et al. Use and nursing of the helmet in delivering non-invasive ventilation. *Minerva Anestesiol* 2002;68:475-80.
- Rocco M, Dell'Utri D, Morelli A, Spadetta G, Conti G, Antonelli M, et al. Non-invasive ventilation by helmet or face mask in immunocompromised patients: a case-control study. *Chest* 2004;126:1508-15.
- Foti G, Cazzaniga M, Villa F, Vale E, Sabato M, Apicella F, et al. Out of hospital treatment of acute pulmonary oedema by non-invasive continuous positive airway pressure: feasibility and efficacy. *Intensive Care Med* 1999;S112:A431.