

# Curative effect of pleural lavage on open chest trauma caused by seawater immersion

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**BACKGROUND AND OBJECTIVES:** Open chest trauma with seawater immersion can lead systematic inflammatory response and multiple organ dysfunction syndromes (MODS). Early intervention of seawater immersion significantly decreases mortality. This study aims to explore the curative effect of pleural lavage in the treatment of open chest trauma caused by seawater immersion on dogs.

**DESIGN AND SETTINGS:** An in vivo experimental study was performed in healthy cross-breeding adult dogs (n=20).

**SUBJECTS AND METHODS:** A dog model of open chest trauma caused by seawater immersion was established. All experimental dogs were divided into control group and pleural lavage group, with 10 dogs in each group. In the control group, dogs were performed ventilator-assisted breathing, and thoracic tube was kept open for adequate chest water drainage; in the pleural lavage group, dogs were further injected with 0.9% sodium chloride (35 mL/kg) immediately into the right side of the chest after the pleural effusion was drained off. The internal environment, oxygen partial pressure, and pathological changes of the lung tissue were observed and recorded.

**RESULTS:** Following open chest trauma caused by seawater immersion, both groups showed obviously increased serum sodium and plasma osmolality and sharply decreased oxygen partial pressure. After treatment, the serum sodium and plasma osmolality decreased, whereas oxygen partial pressure increased in both groups. The pleural lavage group showed better improvement than the conventional treatment group. The pathological changes in the pleural group were lighter than in the conventional treatment group.

**CONCLUSION:** Compared with conventional treatment, repeated pleural lavage shows improved treatment in the correction of blood hypertonic state and hypoxemia in seawater-immersed open chest trauma.

Open chest trauma with seawater immersion has become more and more frequent due to the increase of marine production or military activities. Because of physicochemical features of seawater, chest open wound and seawater immersion can not only lead to lung injury directly, but also cause high sodium blood symptom, hyper tonicity dehydration, and other complex and special internal environmental changes, leading to systematic inflammatory response, serious lung injury, and multiple organ dysfunction syndromes (MODS).<sup>1</sup> Therefore, an early treatment of seawater immersion wound becomes the hot spot of current research.<sup>2</sup> Pleural lavage is the washing of the thoracic cavity and is widely used in open chest trauma to minimize the incidence of inflammation, infection, and empyema.<sup>3</sup> The study aims to observe the treatment effect of pleural lavage on dogs with open chest trauma caused by sea-

water immersion, and to explore its protective action on lung function.

## SUBJECTS AND METHODS

### *Artificial seawater preparation*

Experimental artificial seawater is batched with the main ingredients of seawater from southeastern area supplied by National Ocean Bureau 2nd Ocean Research Institute. The osmotic pressure of artificial seawater was maintained at 1250 to 1350 mmol/L, and seawater proportion maintained at 1.020 to 0.125, with pH value of 7.9 to 8.1. The average temperature of the lab and artificial water was maintained at 25°C and 22°C, respectively.

### *Preparation of animal models*

The guidelines for animal ethics were followed in all the

care and handling of the animals, and protocols were approved by Ethics Committee of The PLA 117 Hospital. A total of 20 healthy cross-breeding adult dogs, weighing 12 to 15 kg, were used in this study, with their gender evenly distributed. Intraperitoneal injection of Ketamine (20 mg/kg) was carried out to anesthetize animals, followed by endotracheal intubation. The left carotid and jugular vein were separated to connect infusion tube and pressure sensor, respectively. A rubber tube (0.5 cm) was embedded at the fourth intercostal space in the right thoracic cavity to form open pneumothorax, and the drainage tube was embedded at the sixth intercostal space in the right thoracic cavity, which was clamped for further experiment. The experimental animals were immersed into artificial water, ensuring the rubber tube sank into the water. After 15 minutes, the experimental animals were taken out of the seawater, and thus animal models with open chest trauma caused by seawater immersion were produced.

#### *Experimental animals grouping and treatment*

Experimental animals were randomly divided into 2 groups: control group and pleural lavage group, with 10 animals in each group. The control group was performed conventional treatment as follows: ventilator-assisted breathing was maintained (tidal volume: 8 mL/kg; respiratory rate: 30 beats/min; oxygen concentration: 100%); then the rubber tube was occluded and the thoracic tube was kept open for adequate chest water drainage. Ketamine, diazepam, and vecuronium were applied in animals to maintain sedation. The 5% glucose was infused intravenously, and the amount of fluid was calculated according to the following formula: ([serum sodium measured value – serum sodium normal value) × body weight × 4]. Half of the calculated amount of fluid was given within the first 4 hours, and the remaining fluid volume was decided by the measured serum sodium. The pleural lavage group was treated conventionally, in which case 0.9% sodium chloride (35 mL/kg) was immediately injected into the right side of the chest after the pleural effusion was drained off, and the fluid temperature was maintained at 35°C for 30 minutes. The lavage and drainage were repeated for 3 times.

#### *Monitoring index*

(1) The arterial blood of experimental animals was collected for blood gas analysis to determine the arterial partial pressure of oxygen (pO<sub>2</sub>) and carbon dioxide (pCO<sub>2</sub>) before injury, before treatment (0 hour), and 0.5 hour, 1 hour, 2 hours, 4 hours, and 8 hours after treatment. (2) Venous blood of experimental animals was also collected to determine serum electrolyte indicators and biochemi-

cal indicators (3) Plasma crystal osmotic pressure was calculated according to the following formula: plasma crystal osmotic pressure = 2 × (Na<sup>+</sup> + K<sup>+</sup>) + glucose + blood urea nitrogen, with all units as mmol/L.<sup>4</sup> After 8 hours, the right lower lung tissue was taken off for pathological examination.

#### *Statistical analysis*

The statistical analysis was performed using SPSS software, version 14.0 (SPSS, Chicago, Illinois). Data were expressed as mean (SEM). Student t test (unpaired, 2 tailed) was performed to compare the means between 2 groups. *P* < .05 was considered as statistically different.

## RESULTS

#### *Serum electrolyte indicators*

All experimental animals were alive for more than 8 hours. In the control group, the peak of the measured serum sodium value was 157.8 (4.5) mmol/L at 1 hour after injury. The serum sodium value at 8 hours after injury was close to the values before injury. In the pleural lavage group, the peak of the measured serum sodium value was 153.0 (7.3) mmol/L, and this appeared before the treatment (0 hour). After the treatment, the serum sodium level rapidly declined to the level before injury. Animals in the pleural lavage group had significantly lower serum sodium values than in the control group at 1 hour, 2 hours, and 4 hours (*P* < .05) (Figure 1A). No significant differences were found in the serum potassium level between the 2 groups at any given time (*P* > .05) (Figure 1B).

#### *Biochemical indicator*

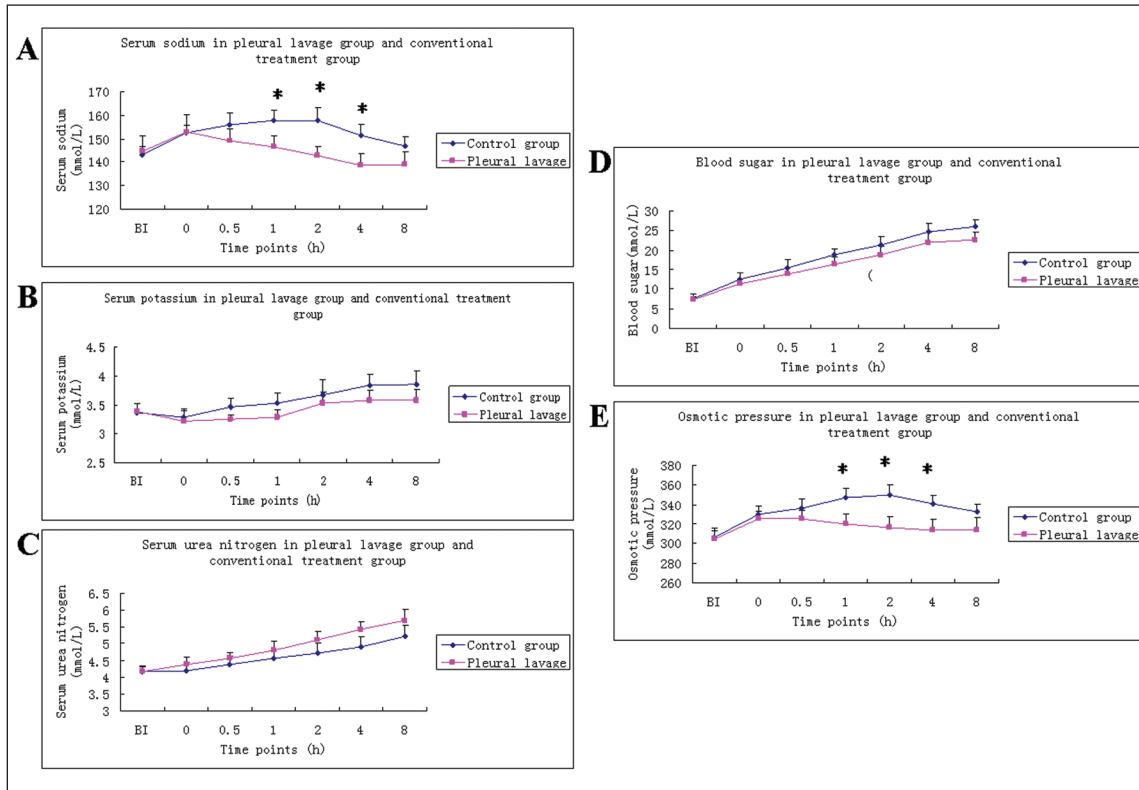
There were no significant differences in urea nitrogen and blood sugar between the 2 groups at any given time (*P* > .05) (Figure 1C, 1D).

#### *Plasma crystal osmotic pressure*

The peak value of the plasma crystal osmotic pressure in conventional group was 346.32 (3.65) mmol/L at 2 hours after injury. Then the values gradually decreased. In the pleural lavage group, the peak value of the crystal osmotic pressure was 325.15 (8.01) mmol/L before the treatment (0 hour), followed by a gradual decrease. Animals in the pleural lavage group had significantly lower plasma crystal osmotic pressure values than in the control group at 1 hour, 2 hours, and 4 hours (*P* < .05) (Figure 1E).

#### *Arterial blood gas analysis*

The arterial partial pressure of oxygen (pO<sub>2</sub>) decreased



**Figure 1.** Serum electrolytes levels and plasma crystal osmotic pressure values before and after the injury in the control group and the pleural lavage group (mmol/L). A) Animals in the pleural lavage group had significant lower serum sodium values than in the control group at 1 h, 2 h, and 4 h. B) There was no significant difference in serum potassium between two groups. C) There were no significant differences in urea nitrogen between two groups at any time points. D) No significant differences were found in blood sugar between two groups. E) Animals in the pleural lavage group had significant lower plasma crystal osmotic pressure values than in the control group at 1 h, 2 h, and 4 h. \* indicates significant differences between the control group and the pleural lavage group at the same time points. BI: before injury.  $P < .05$  is considered as statistically significant difference.

sharply after injury and before treatment (0 hour) and rose a little in both the control group and the pleural lavage group. Then the values of 2 groups declined and reached the lowest value 8 hours after injury, with 78.8 (22.6) mm Hg in the control group and 153.8 (269) mm Hg in the pleural lavage group. The arterial oxygen pressure values at 1 hour, 2 hours, 4 hours, and 8 hours were significantly lower in the pleural lavage group than in the control group ( $P < .05$ ) (Figure 2A).

There were similar trends in arterial partial pressure of carbon dioxide ( $pCO_2$ ) between the 2 groups, and no significant differences were found at any given time (Figure 2B).

*Pathological changes in lung tissue*

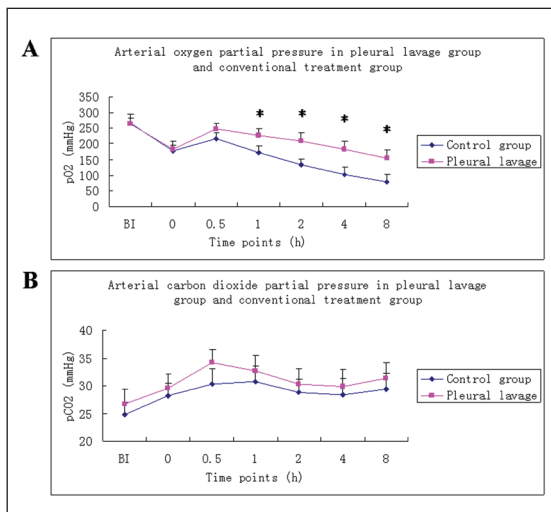
Morphological changes were observed and photographed under light microscopy ( $\times 400$  magnification). In control group, light microscopy examination showed alveolar wall collapse, alveolar visible serous effusion, alveolar interval widened, lymphocyte infiltration,

capillary congestion, and exudation of red blood cells (Figure 3A). The pleural lavage lung biopsy showed the milder alveolar wall collapse, less alveolar exudates, less obvious widened alveolar interval, no distinct capillary congestion, and exudation of red blood cells (Figure 3B).

**DISCUSSION**

Open chest trauma is frequent in accidents or violence with high mortality rate.<sup>5</sup> Seawater immersion of chest trauma may cause very complex physiological and pathological changes. Without early treatments with aggressive surgical procedures, multiple organs failure and even death occur in a short time.<sup>6</sup>

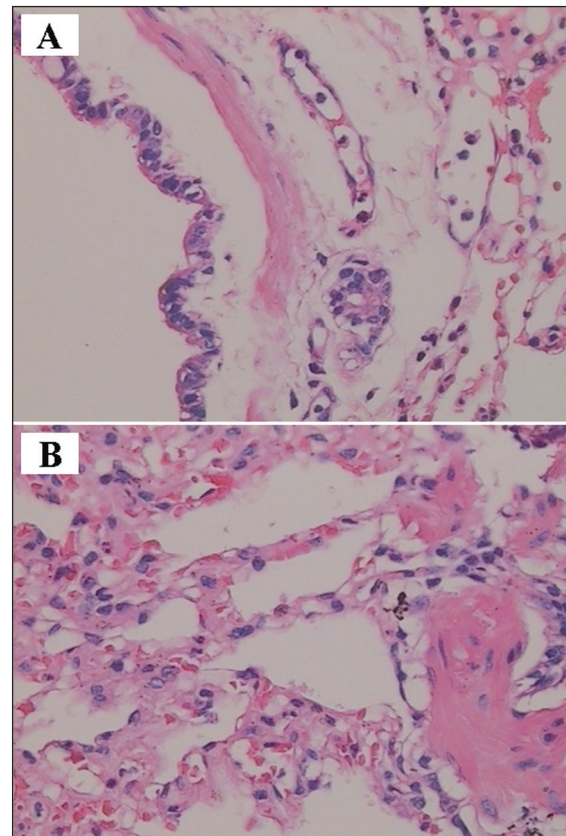
After entering into the thoracic cavity, the seawater elevates the internal pressure of the chest near the wound and causes the oppression on the contralateral lung tissue, resulting in hypoxemia and hypercapnia. The osmotic pressure of seawater is significantly higher than the plasma osmotic pressure, so except the intra-



**Figure 2.** Arterial partial pressure of oxygen ( $pO_2$ ) and partial pressure of carbon dioxide ( $pCO_2$ ) before and after injuries in the control group and the pleural lavage group (mm Hg). A) Arterial  $pO_2$  were significantly lower in the pleural lavage group than in the control group at 1 h, 2 h, 4 h, and 8 h. B) There were no significant differences in arterial  $pCO_2$  between the two groups at any time points. BI: before injury.  $P < .05$  is considered as statistically significant difference.

pulmonary oppression, the hypertonic characteristic of seawater can also lead to lung tissue degeneration, alveolar epithelial injury, and respiratory failure in a short time.<sup>7</sup> It is widely accepted that low tidal volume and appropriate “lung protective ventilation strategy” and the early closure of wound, oxygen inhalation, closed thoracic drainage, and infusion of isotonic solution can effectively improve the pulmonary function.<sup>8</sup> Some other scholars use B ketone theobromine, dexamethasone and other similar drugs to protect the function of lungs through inhibition of inflammatory response.<sup>9,10</sup>

The seawater is a hypertonic solution with sodium concentration about 500 mmol/L (normal plasma sodium concentration: 135~145 mmol/L), while the pleura are good biological semipermeable membranes. Therefore, the entrance of seawater into the thoracic cavity forms a big concentration gradient between the 2 pleural sides, which causes hypernatremia, acidosis, hypertonic dehydration, and intracellular dehydration, subsequently affecting the intracellular normal physiological activities. Hypertonic dehydration, hypernatremia, and water–electrolyte imbalance are important lethal factors that are related with the mass release of inflammatory mediators.<sup>11,12</sup> This can induce a systemic inflammatory response syndrome and make the body suffer a second hit in a short time, further aggravating the pulmonary function damage and inducing multiple



**Figure 3.** Morphological changes of pulmonary tissue in animals with open chest trauma caused by seawater immersion ( $\times 400$  magnification). A) Alveolar wall collapse, visible serous effusion, widened alveolar interval, lymphocyte infiltration, and capillary congestion were found in lung biopsy of the control group. B) Milder alveolar wall collapse, less alveolar exudates, less obvious widened alveolar interval, and no distinct capillary congestion were found in lung biopsy of the pleural lavage group.

organ failure and even death. Studies showed that in patients with acute hypernatremia, when the plasma osmotic pressure was higher than 330 mmol/L, the fatality rate was about 50%; when the plasma osmotic pressure was higher than 350 mmol/L, the fatality rate could reach almost 100%.<sup>13</sup> Therefore, once the open chest trauma is immersed in seawater, the pulmonary function should be preserved as far as possible; at the same time, fast and effective correction of hypernatremia should be carried out to reduce the plasma osmotic pressure and eliminate the adverse factors of seawater.

We performed pleural lavage on the basis of conventional therapeutic measures and showed that the repeated lavage with isotonic saline formed a concentration gradient between blood and pleural cavity, which promoted the internal high-concentration sodium ions to transfer from the blood to the pleural cavity and flow



out of the body through drainage. In this experiment, the blood sodium value in the conventional treatment group reached a peak in 1 hour after the injury and slowly declined in the subsequent treatment process, but the blood sodium value in the pleural lavage group was significantly lower than that in the conventional treatment group.

Plasma osmotic pressure in the conventional treatment group reached a peak in 2 hours after injury. But in the pleural lavage group, it reached the peak before treatment. After lavage the plasma osmotic pressure was statistically significantly lower in the pleural lavage group than in the conventional treatment group. This indicates that compared with low permeability rehydration and other traditional treatment methods, pleural lavage can quickly correct internal high sodium and high hypertonic conditions, and allow the victim break away from hypertonic environment earlier, creating better opportunities for successful rescue.

$pO_2$  of both groups decreased after immersion, but the drop of  $pO_2$  was significantly smaller in the pleural lavage group than in the conventional treatment group. This difference may be caused by the following 2 reasons: (1) In the conventional treatment group, the seawater was discharged by a closed thoracic drainage. However, a complete drainage is very difficult, and the residual seawater continued to function on the pleural cavity. While pleural lavage is able to dilute the residual seawater and reduce its persistent damage. (2) In the process of pleural lavage, the pleural membrane having the semipermeable property can effectively filter inflammatory mediators in the body and reduce a second hit

caused by inflammation, so as to attenuate the lung tissue damage, improve effective ventilation, and increase  $pO_2$ .

A comparative study on lung damages of the open chest trauma caused by seawater and fresh water immersion showed that after the seawater immersion, there was large quantity of edema fluid accumulated in the interstitial pulmonary tissue and alveolar cavity. The alveolar septa were widened obviously with the expansion and congestion of capillaries and massive leukocyte extravasation and aggregation. The integrity of some regional alveolar structures was damaged with the alveolar fusion; some regional alveolar structures were collapsed and atrophied. However, after the immersion of fresh water the lung tissue showed no such obvious changes.<sup>9</sup> In this study, the changes in the lung tissue in the conventional treatment group were similar to the changes caused by seawater immersion, while the damage in the lung tissue in the pleural lavage group was significantly less obvious than in the conventional treatment group. In both groups, the pulmonary pathological changes were mainly consistent with the  $pO_2$  changes. The  $pO_2$  in the pleural lavage group was generally higher than that in the conventional treatment group. However, inflammatory mediators cannot be completely removed, so 8 hours later  $pO_2$  in the pleural lavage group was still hard to return to the level before the injury.

In conclusion, repeated pleural lavage shows an improved treatment in the open chest trauma caused by seawater immersion, with clear effects on the correction of blood hypertonic state and hypoxemia, but the long-term effects of pleural lavage need further study.

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