Acute respiratory distress syndrome related to near hanging: a case report

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Summary

A 13-year-old male patient was admitted to our emergency unit because of near-hanging while playing with a rope. On physical examination, his general condition was not good, his state of consciousness was evaluated to be stuporous and his spontaneous respiration was weak. The patient had been intubated. The Glasgow coma score was 7. Rope marks were observed on his neck. Thorax computed tomography revealed widespread ground-glass appearance and bilateral parenchymal consolidation in the lungs. This findings correlated with acute respiratory distress syndrome. This case was reported for reminding that acute respiratory distress syndrome can be associated with near-hanging. (Turk Arch Ped 2012; 47: 298-300)

Key words: Acute respiratory distress syndrome, asphyxia, near-hanging

Introduction

Acute respiratory distress syndrome (ARDS) is acute respiratory insufficiency caused by damage to alveolar epithelium and epithelial cells, acute inflammation and protein-rich pulmonary edema (1). In the pathogenesis of acute respiratory distress syndrome, neutrophil migration and damage to the lungs as a result of activation of inflammatory mediators occur. Neutrophils lead to cell damage by releasing free radicals, inflammatory mediators and cytokines including proteases (elastase, collagenase, reactive oxygen species, tumor necrosis factor-α) (2).

This 13-year-old male patient who experienced near-hanging while playing with a rope was presented for reminding that acute respiratory distress syndrome can be associated with near-hanging, though rarely.

Case

A 13-year-old male patient was presented because of near-hanging while playing with a rope. It was not known for how many minutes he was hanged on the rope. There was no pathology in his personal and familial history. He had no signs in favor of mood disorder in recent days. His general condition was poor, his consciousness was stuporous, his spontaneous respiration was week and he was intubated. The patient’s ventilation was provided by ambu bag. There were rope marks on his neck because of near-hanging. Glasgow coma score was found to be 7. His blood pressure was found to be 130/70 mmHg, his apical heart beat was found to be 88/min, his body temperature was found to be 37°C (axillary) and his respiratory rate was found to be 22/min. Widespread crepitant rales were heard over both lungs. Deep tendon reflexes were found to be normoactive on neurologic examination. He responded to painful stimuli. Flexor response was present for Babinsky reflex on both sides.

Laboratory findings were as follows: WBC:39 290/mm3, hemoglobin: 12.6 g/dL, platelets: 391 000/mm3, blood glucose: 159 mg/dL. Follow-up blood glucose was found to be normal. Creatinine kinase: 5 878 U/L, creatinine kinase-MB: 162 U/L, lactate dehydrogenase 2 152 U/L, aspartate aminotransferase: 250 U/L, alanine aminotransferase: 65 U/L, gamma glutamyl
transferase: 17.8 U/L. Blood gases were as follows: pH: 7.38, PCO2 21 mmHg, PO2 69 mmHg, HCO3 12.1 mmol/L and O2 saturation 94%. FiO2 was found to be 36 and PaO2/FiO2 was found to be 191.6. Renal function tests were found to be normal. C-reactive protein: 18.2 mg/L, erythrocyte sedimentation rate: 20 mm/h, prothrombin time: 18.9 s, activated partial thromboplastin time: 30 s. After one week some of the laboratory findings were as follows: creatinine kinase: 466 U/L, creatinine kinase-MB: 39 U/L, lactate dehydrogenase: 1198 U/L, aspartate aminotransferase: 33 U/L, alanine aminotransferase 32 U/L, prothrombin time: 13.6 s and activated partial thromboplastin time: 26 s. Radiologic examinations revealed diffuse nodular appearance on antero-posterior lung graphy (Picture 1). Radiologic findings of the lung were observed to be improved in the follow-up (Picture 2, 3). Widespread brain edema was observed on computed tomography (CT). Cervical CT was found to be normal. Thoracic CT revealed widespread ground glass appearance compatible with ARDS and collapsed and consolidated areas in both lungs (Picture 4a, 4b).

The patient’s FiO2 was found to be 36 and PaO2/FiO2 was found to be 191.6. It was learned that no vomiting occurred during hanging and resuscitation. A diagnosis of ARDS was made based on the clinical status and a PaO2/FiO2 value of <200. The patient was hospitalized with diagnoses of near-hanging, hypoxic cerebral edema following near-hanging and ARDS. He had insufficient spontaneous respiration. Mechanical ventilation set on SIMV mode was started in the supine position. The initial ventilator settings were as follows: the highest PEEP: 12 cmH2O, PO2: 60 mmHg. The highest PEEP became 12 cmH2O2 and the highest FiO2 became 100. In the follow-up, these values were reduced gradually until the oxygen saturation became 90%. For brain edema limited fluid, albumin and dexamethasone treatment was given. In the follow-up, it was observed that the patient’s spontaneous respiration was sufficient and he was separated from the ventilator on the 4th day of hospitalization. The patient’s consciousness opened three days later. After the consciousness was opened it was observed that the patient could make eye contact, but had difficulty in giving responses directed to the target. His attention was...
weakened, his instantaneous and recent memory was weakend and his remote memory was normal. His thinking did not contain active phobia, delusions, obsession or suicidal ideation. He reported that the event occurred accidentally while playing with a rope. Cranial magnetic resonance imaging (MRI) revealed increased signal compatible with hypoxic ischemic changes in the posterior parts of both frontal lobes, left parietal lobe and cortico-subcortical areas of both occipital lobes. Three dimensional cervical tomography revealed tracheal narrowing prominent 2.7 cm distal to the larynx. On the 14th day, widespread nodular appearance on lung graphy disappeared. After 2 months lumbar, cervical and thoracic MRI examinations were found to be normal. The patient who was discharged in a healthy condition. He has no signs of complication on the 10th month of the follow-up.

Discussion

Acute respiratory distress syndrome is a very severe form of lung damage which impairs the lung. Its pathogenesis is characterized by widespread alveolocapillary wall damage and increased permeability of lung vessels related to alveolar and interstitial edema. The incidence of this disease in children is 22.7/1000 patients admitted to pediatric intensive care units (3). In pediatric patients, the mortality rate of ARDS has been reported to be 22% (4).

Although there are many cases of ARDS due to reperfusion damage after hypoxia and/or ischemia, ARDS cases related to near-hanging have not been reported frequently. In a 7-year retrospective study, ARDS was reported in three of 23 patients who were referred with near-hanging (5).

ARDS is defined as follows according to 1994 American-European consensus criteria: 1- Acute onset, 2- Severe arterial hypoxemia (PaO₂/FiO₂< 200), 3- Bilateral radiographic involvement, 4- Absence of left atrial hypertension (6). Dyspnea, tachypnea and hypoxemia which constitute initial clinical findings of ARDS can occur as early as 1-2 hours after the causative factor. However, they are usually observed in 12-24 hours. In the first 24-72 hours, the consistency of the lung decreases, dyspnea and hypoxemia become prominent (1). Widespread involvement is observed on lung graphy (1). Our patient had been intubated because of acute respiratory failure related to near-hanging. On physical examination, he had bilateral widespread crepitant rales. Widespread nodular appearance was found on lung graphy. PaO₂/FiO₂ ratio of the patient who was intubated was calculated to be 191.6. The patient who had no arterial hypertension was diagnosed as ARDS with these findings.

In one study, thoracic CT revealed ground glass appearance in 50.2% of the patients, air consolidations in 16% and empty area and severe abnormalities in peribronchial areas in 26.6% (7). Widespread brain edema was found on brain CT in our patient. His cervical CT was normal. His thoracic CT revealed widespread ground glass appearance in both lungs compatible with ARDS and widespread collapsed and consolidated areas.

Near-hanging affects all organs of the body. It leads to acute and chronic respiratory complications. Treatment consists of increasing oxygen level, controlling increased intracranial pressure and treatment of respiratory distress (8). Treatment in ARDS consists of symptomatic and supportive therapy. The mortality rate in ARDS is still very high. The mortality is primarily related to complications of multiple organ failure caused by the pathologies causing ARDS rather than ARDS itself. Owing to modern ventilation protocols only 15% of the patients are lost due to failure of oxygenation of the blood by the lungs with ARDS (9,10). Currently, new therapies including high frequency ventilation, facedown position, inhaled nitric oxide, surfactant treatment and extracorporal membrane oxygenation are being experimented (9). In our patient, supportive treatment with mechanical ventilation and steroid treatment were administered. In addition, anti-edema treatment was given for brain edema. In the follow-up, spontaneous respiration became sufficient and the patient’s consciousness opened. He was separated from the ventilator and discharged with cure on the 15th day of hospitalization.

This patient was presented to remind that ARDS due to near-hanging can be observed.

References