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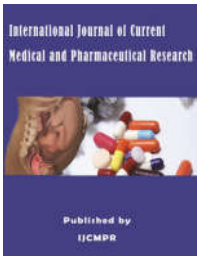

MANAGEMENT OF TRAUMATICASPHYXIA

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ABSTRACT

Background: Traumatic asphyxia (TA) is a clinical syndrome associated with cervicofacial cyanosis, edema-petechiae, subconjunctival hemorrhage, and neurological symptoms following a sudden, severe compressive and blunt thoracoabdominal trauma. This syndrome is also called "*acute thoracic compression syndrome", "ecchymotic mask", or "Perthez syndrome"*. The syndrome is an uncommon but it can potentially lead to serious clinical consequences.
Method: Among 568 blunt thoracic trauma patients, we retrospectively evaluated a total of 12 patients for the diagnosis of TA in our department between February 2010 and February 2017. All patients were male with a mean age of 44,5 ± 15,6 (18-64 years)
Results: The patients were admitted to various types of trauma: Four (33%) motorcycle accidents, three (25%) car crashes, two (16%) industrial accidents, one (8%) farm accident, one (8%) mining accident and one (8%) truck accident. The most common accompanying pathology was pneumothorax -hemopneumothorax in seven (58%) patients and tube thoracostomy was performed. Other accompanying traumatic pathologies were rib fractures in five patients, sternal fracture in one, pelvis and upper extremity fractures in one, clavicular fracture in one, cephalic haematoma in one and, pulmonary contusion in one. All patients were treated as conservatively. In three (25%) of the patient neurological findings were developed as agitation, disorientation and loss of consciousness during follow. These findings regressed after supportive conservative treatment within days. The mean time of length of hospital stay was 6 ± 2, 9 days (4-15 days). There was no mortality.
Conclusion: Traumatic asphyxia is a rare syndrome after blunt thoracic trauma. In the majority of cases supportive conservative therapy as head elevation of 30 degrees, continuous oxygen therapy and close follow up in terms of neurological and other complications may be sufficient for the management of the syndrome. However, injuries associated with trauma must be kept in mind and these injuries should be appropriately treated.

INTRODUCTION

Traumatic asphyxia (TA) is a clinical syndrome associated with cervicofacial cyanosis, edema-petechiae, subconjunctival hemorrhage, and neurological symptoms following a sudden, severe compressive and blunt thoracoabdominal trauma. This syndrome is also called" acute thoracic compression syndrome", "ecchymotic mask", or "Perthez syndrome". The syndrome is rare but it may lead to potentially serious clinical consequences (1). The physiopathological classification is not fully defined and controversial. However the acute pressure to the mediastinum, thorax and heart is commonly thought to be the cause. Four factors are responsible in the formation of this entity including deep inspiration, closed glottis, thoracoabdominal effort and compression that will lead to blood cervicofacial area (2). During the accident, holding deep inspiration and intrathoracic pressure increasing with the Valsalva maneuver and closure of the glottis also contributes to this process. Because of increased intrathoracic pressure blood fills valveless vein in the head and neck causes these findings. Experimental studies on animals have shown increased jugular venous pressure when the glottis closed (3,4).We present 12 consecutive patients with TA after blunt thoracic trauma who were admitted to our clinic.

MATERIAL METHOD

Among 568 blunt thoracic trauma patients, we retrospectively evaluated a total of 12 patients for the diagnosis of TA in our department between February 2010 and February 2017. All patients were male with a mean age of 44,5 ± 15,6 (18-64 years). Age, gender, type of trauma, applied treatments, complications and neurological findings, length of hospitalization were examined. The diagnosis was made from the physical appearance, history and examination, there were diffuse petechiae, ecchymoses and edema on the face, neck and upper chest, bilateral subconjunctival hemorrhages in all patients (Figure I). We evaluated all patients with Chest X-ray, full body computed tomography and if needed with ultrasonography. Routine hemostasis, hemogram parameters and biochemical blood tests examined. Additionally, the

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cardiac status of the patients was evaluated by serial serum cardiac enzyme determinations, electrocardiograms, and if need echocardiography. All patients treated conservatively. Oxygen saturation, arterial blood gases and blood pressure were closely monitored. Continuous nasal oxygen therapy, fluid replacement and head elevation of 30 degrees for reduce the intracranial pressure were applied. Demographic and clinical features of patients were summarized in Table I.

Data Analysis

A statistical analysis was performed using the Statically Package for the Social Science program (SPSS, 20.0) Data were expressed as mean ±SD. Frequencies and percentages were used for the categorical measures.

RESULTS

The most common accompanying pathology was pneumothorax -hemopneumothorax in seven (58%) patients and tube thoracostomy was performed (unilateral in five patients, bilateral in two patients).

Figure I.(A,B,C,D,E) Multiple petechial hemorrhagic areas in patients's head, neck, and upper chest and bilateral subconjunctival hemorrhages can see in five of the patients.

and wound infection. In one case that developed ARDS in intensive care unit after orthopedic surgery mechanical ventilation required for a total of 3 days. In three (25%) of the patient neurological findings were seen as agitation, disorientation and loss of consciousness. The prognosis was good in all patients. The mean time of length of hospital stay was 6 ± 2,9 days (4-15 days). There was no mortality. The patients were followed two weekly intervals during the three months.

DISCUSSION

Traumatic asphyxia is a rare condition. The majority of cases are motor vehicle accidents. Other causes are industrial-sporting accidents, violence, earthquakes, rarely deep diving, epileptic attack, difficult births etc.(5). Traumatic asphyxia is usually secondary to blunt chest-abdominal trauma, but may occur in any situation increasing intrathoracic pressure with the closed glottis. El Amraoui *et al* (6). have presented a 15 years old child with TA triggered an asthma attack during surgery.

Although the etiology of traumatic asphyxia is not completely known, acute compression of the mediastinum, chest, and heart is widely considered. This is related to the extravasation from the blood in the cervicofacial region after the patient takes a holding the deep breath and closure of glottis during the accident. This is not seen when the patient does a deep inspiration during the accident and does not hold his breath (7). The main principal therapy is supportive treatment.

Table I Demographic and clinical features of traumatic asphyxia patients.

No	Age/Sex	Type of trauma	Additional pathology	Treatment	Complication	Presence of neurological symptom	Length of hospital stay
1	26/M	Motorcycle accident	Clavicula and rib fracture	Conservative	-	Ø	4
2	34/M	Industrial accident	Pnx	TT	-	Ø	5
3	64/M	Farm accident	Rib fracture	Conservative	CPPS	Ø	4
4	52/M	Motorcycle accident	Sternal fracture	Conservative	-	Ø	6
5	28/M	Industrial accidents	HemPnx	TT	Wound infection	Ø	7
6	45/M	Truck accident	Pnx	TT	-	√	5
7	61/M	Car accident	Rib fracture pulmonary contusion	Conservative	Pneumonia	Ø	6
8	55/M	Car accident	Cephalic hematoma	Conservative	-	√	4
9	62/M	Mining accident	HemPnx, Rib fracture	TT	Atelectasis requiring FOB	Ø	5
10	18/M	Motorcycle accident	Pnx,Pelvis and extremities fracture	TT, Orthopedic surgery	ARDS	√	15
11	36/M	Car accident	HemPnx	TT	-	Ø	6
12	53/M	Motorcycle accident	Pnx Rib fracture	TT	-	Ø	5

HemPnx: Hemopneumothorax **Pnx:** Pneumothorax **TT:** Tube Thoracostomy **CPPS:**Chronic Postthoracotomy Pain Syndrome **FOB:** Fiberoptic bronchoscopy, **ARDS**: Acute Respiratory Distress Syndrome

Other accompanying traumatic pathologies were rib fractures in five patients, sternal fracture in one, pelvis and upper extremity fractures in one, clavicular fracture in one, cephalic haematoma in one and, pulmonary contusion in one and treated effective analgesic and mucolytic treatments and extremity fixation if needed. Neurologically signs followed by consciousness-pupil follow-up and glaskow coma scoring. Mean Glaskow Coma Score was 14,4 ±0.3(13-15) initially at the emergency department. Findings of syndrome related symptoms regressed in the following dates. Complications developed in five (42%) of the patients as post-thoracotomy pain syndrome, pneumonia, atelectasis requiring bronchoscopy, ARDS (Acute Respiratory Distress Syndrome)

Patient should be monitored, oxygen saturation, arterial blood gases and blood pressure should be closely followed and oxygen support should be obtained (8). The development of neurological sequelae depends upon the duration and severity of the crush injury. Prolonged compression may be life threatening due to apnea and hypoxemia and may increase mortality (9). Although the prognosis is good in the majority of cases; severe shock-cerebral edema, neurological sequelae and sudden death at an accident site within a few minutes can develop. Morbidity and mortality are usually associated with the presence and severity of pulmonary and neurologic crush injury (10). It should be kept in mind that cerebral edema and hemorrhage may develop, thus intracranial pressure

monitoring should be performed for this condition. Clinical symptoms may develop from mild to severe coma due to hypoxic cerebral damage. The 30 degree of head elevation and continuous oxygen support helps to reduce intracranial pressure. These neurological symptoms may be improved with conservative treatment within two weeks (9-11). Prolonged compression time increases neurological sequelae possibility and mortality due to apnea and hypoxia. The incidence of mortality and / or morbidity depends on the accompanying injuries (11,12). In our cases pneumothorax-hemopneumothorax were mostly detected as accompanying injuries.

Since the syndrome can lead to severe airway edema besides unconsciousness be prepared for airway management in terms of non-laryngeal intubation methods (11).

Rosato *et al* (13). have presented that cardiac injury complicating of TA as two cardiac contusions and a ventricular rupture for three patients in their study. They have seen one death in the series, a patient with rupture of the right ventricle and severe splenic and liver injuries. In our study we evaluated the cardiac status of the patients by serial serum cardiac enzyme determinations, electrocardiograms, and if need echocardiography and there was no cardiac injury and mortality.

Cyanosis and edema may be seen upper chest, head and neck due to increased venous pressure. Petechiae can be detected in the tongue, lips, mouth, nose, ears, and pharynx. Subconjunctival hemorrhage is often seen, but it may also be seen on the retina vitreous and optic nerve. In these cases, blindness may be permanent. Also exophthalmia may be occurred due to bleeding behind the eye globes. Tinnitus or temporary deafness may develop. Bilateral optic disc edema and temporary blindness in association with TA cases have been reported in the literature (14,15). In our cases, we observed neither deafness nor blindness.

CONCLUSION

Traumatic asphyxia is a rare prognostic syndrome after blunt thoracic trauma. Supportive conservative therapy as head elevation of 30 degrees, continuous oxygen therapy can be sufficient in patients who are followed up with neurological examination and monitoring. However, injuries associated with trauma must be kept in mind and these injuries should be appropriately treated.

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