

BLOOD GASES AS AN INDICATOR OF INHALATION INJURY AND PROGNOSIS IN BURN PATIENTS

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SUMMARY. Inhalation injury greatly increases the incidence of respiratory failure and the acute respiratory distress syndrome. It is also the cause of most early deaths in burn victims. The aim of our research was to study the incidence, early diagnosis, complications, and management of inhalation injury and to discuss the relation between inhalation injury and death in burn patients. This study included 130 burn patients with inhalation injury admitted to Menoufiya University Hospital Burn Center, Egypt, from January 2004 to April 2008 (61 males and 69 females). We found that the presence of inhalation injury, increasing burn size, and advancing age were all associated with increased mortality ($p < 0.01$). The incidence of inhalation injury in our study was 46.3% (130 patients were identified as having inhalation injury out of 281). The overall mortality for patients with inhalation injury was 41.5% (54 patients out of 130) compared with 7.2% (11 patients out of 151) for patients without inhalation injury. These statistical data make it clear that inhalation injury is an important factor for the prediction of mortality in burn patients. Approximately 80% of fire-related deaths are due not to the burn injury to the airway but to the inhalation of toxic products, especially carbon monoxide and hydrogen cyanide gases. Inhalation injury is generally caused by thermal burns, mostly confined to the upper airways. Major airway, pulmonary, and systemic complications may occur in cases of inhalation injury and thus increase the incidence of burn patient mortality.

Introduction

Arterial blood gas analysis is a fundamental diagnostic test which must be performed serially in patients clinically suspected of inhalation injury. Arterial hypoxaemia and reduced oxyhaemoglobin saturation (SaO_2) accompany smoke inhalation.¹

Thus, the presence of hypoxaemia is an indicator of inhalation injury until proved otherwise. However, arterial oxygen tension is often normal (over 71 mm Hg) during the early evaluation of patients who have sustained severe inhalation injury.²

Initial measurements of PaO_2 are therefore not a sensitive indicator. The onset of hypoxaemia may be delayed as much as 48 h in spite of the absence of clinical or X-ray evidence of inhalation injury.³

Severe hypoxia that is not improved by supplemental oxygen denotes shunt (due to pulmonary oedema or major atelectasis). Milder hypoxaemia (improved with O_2 therapy) suggests less severe ventilation/perfusion mismatch (V/Q) due to airway obstruction.⁴

Haemoglobin oxygen saturation can be monitored by pulse oximetry. However, oedema or decreased local perfusion of the oximeter probe can decrease its usefulness even after COHb levels have fallen.⁴

Aim of the work

The aim of this work was to study the role of blood gases in the early diagnosis of inhalation injury and the relation between inhalation injury and death in burn patients.

Patients and methods

The study included 130 burn patients with inhalation injury admitted to Menoufiya Burn Unit, General Surgery Department, from January 2002 to January 2008. Each patient was subjected to:

1. History taking
 - Name, age, sex and past history of illness, chronic or congenital diseases (age is a valuable factor in the prognosis of cases: at very young or very old age, the prognosis is very bad); sex is important as females are more exposed to burns; chronic diseases such as diabetes mellitus, ischaemic heart disease, and bronchial asthma can increase the mortality rate if associated with burns
2. General examination
 - On admission we measure vital signs such as blood



Fig. 1 - Burned male patient with inhalation injury at moment of admission; family used oil to deal with burn.



Fig. 2 - Child with suspected inhalation injury with severe face and lip oedema; eyes closed owing to severe oedema.

pressure, pulse, and temperature (blood pressure and pulse to know the degree of hypovolaemia and as a base line for whether progress of fluid intake is sufficient or not; we examine level of consciousness to determine the degree of hypoxaemia

3. Evaluation and assessment of inhalation injury

- We suspect smoke inhalation injury in anyone who was trapped in a confined space or who lost consciousness during a fire, especially in the presence of large quantities of heavy smoke. Clinical manifestations vary among victims, depending on their susceptibility to injury and degree of exposure. Injury may be limited to the upper airways (e.g. nasopharyngeal irritation, hoarseness, stridor, cough) or may extend distally with tracheobronchial and alveolar destruction (e.g. dyspnoea, chest discomfort, haemoptysis). Inhalation injury is likely in the presence of facial and upper cervical burns, singed eyebrows and nasal vibrissae, bronchial breath sounds, wheezing, râle, cyanosis, and carbonaceous sputum (Figs. 1-4).

We evaluate patients presenting with inhalation injury for the extent of the disease and the degree of hypoxaemia. Patients at low risk of such and with no clinical symptoms can usually be observed for 4-12 h and then be discharged with a close follow-up and instructions to return if symptomatic.

Investigations

All patients admitted to our department with suspected inhalation injury had an arterial blood gas analysis performed as a fundamental diagnostic test. In our department this test is performed serially in patients clinically suspected of inhalation injury. Arterial hypoxaemia and reduced oxyhaemoglobin saturation (SaO_2) accompany smoke inhalation.

The presence of hypoxaemia is thus an indicator of inhalation injury until proved otherwise. Initial measurements of PaO_2 are therefore not a sensitive indicator.

It was found that the onset of hypoxaemia could be delayed by as much as 48 h in spite of the absence of clinical or X-ray findings of inhalation injury.

- Chest X-ray: we perform serial chest x-rays in patients with suspected inhalation injury. Chest x-rays taken on the day of injury are an unreliable means of determining pulmonary injury due to smoke inhalation. However, we perform it on admission because it serves as a baseline for subsequent radiographs. Chest x-rays are used in patient follow-up in order to detect atelectasis and more or less extensive peribronchial cuffing or oedema. In our department we perform x-rays to detect complications in various therapeutic interventions (such as endotracheal intubation, intravenous catheterization, and replacement of the central venous line)
- Respiratory function tests: we use these tests as a bedside procedure in the early detection and quantitation of inhalation injury. They demonstrate the presence of airway obstruction
- Fiberoptic bronchoscopy: this investigation provides direct information about all respiratory passages. In addition to its diagnostic functions, bronchoscopy has important therapeutic and investigative applications - it is used to determine the severity of inhalation injury according to the following grading:
 - * Grade I: no laryngeal oedema
 - * Grade II: minimal laryngeal oedema and erythema
 - * Grade III: slight tracheal mucosal oedema and erythema
 - * Grade IV: moderate tracheal mucosal oedema and erythema
 - * Grade V: severe tracheal oedema and erythema



Fig. 3 - Deep burn in face, history of entrapment in closed space, soot around mouth, patient drowsy face with lip oedema.



Fig. 4 - Very deep burn in face; soot around mouth.

4. First aid and initial management

Initial management starts when burn patients with inhalation injury come to our emergency room, where we give them first aid as follows:

- a) airways: ensure patent airways
- b) breathing: we check for upper airway compromise, difficult breathing, stridor, and coughing. We administer 100% oxygen because of the possibility of carbon monoxide inhalation in fires
- c) circulation: patients are then admitted to the burns unit in accordance with our admission policy.

When patients arrive in the burns unit, there are two main problems. The first problem is severe pain, which may lead to neurogenic shock; the second is fluid loss, which may lead to hypovolaemic shock.

The patients start their journey of treatment from the dressing-room, where doctors and a nurse begin the dressing procedures. We put the patients on a trolley to take them to a water tank for removal of dead skin and aspiration of bullae. Next we paint the patients with Dermazinc (silver sulphadiazine). During dressing, a venous line (cannula) is inserted and fluid therapy is commenced (500 cc Ringer's acetate), for which we give an analgesic. We then weigh the patients and the burn percentage is calculated for fluid circulation therapy using the Evans formula ($2 \text{ cc} \times \text{weight in kg} \times \text{burn percentage}$). If the burn is more than 20% in children or more than 25% in adults, a urine catheter is inserted in order to calculate urine output, which should be 1 cc/kg of body weight per hour for adequate perfusion and fluid resuscitation.

In major burns (more than 50% of total body surface area [TBSA]) we insert a central venous line (Swan-Ganz catheter) for continuous measurement of central venous pressure.

Patients are then assessed for the presence of inhala-

tion injury. If inhalation injury is suspected a 100% oxygen mask is applied, followed by nebulized heparin.

5. Definitive treatment

- a) Fluid resuscitation: our unit follows the Evans formula ($2 \text{ cc/kg/percentage burn lactated Ringer's}$); patients are given a maintenance dose as glucose. All fluids should be warm to avoid hypothermia
- b) Dressing: the patient is dressed twice daily, using the open method, with micronized silver sulphadiazine in a hydrophilic base (Dermazinc) after hydrotherapy
- c) Medical treatment: if any patient is suspected of inhalation injury, we administer humidified oxygen using high oxygen flow rates and a non-rebreathing-type face mask with a tight seal that facilitates delivery of high levels of supplemental oxygen, which helps reverse the oxygenation defect created by the ventilation-perfusion mismatch. Inhaled oxygen also helps displace CO from haemoglobin, decreasing the half-life of carboxyhaemoglobin from 4-6 h in room air to 40-60 min in 100% FiO_2 . Oxygen therapy should be continued until the acidosis is corrected, the carboxyhaemoglobin levels have fallen below 15%, and neurological symptoms have resolved, which typically takes several hours. We give nebulized Farcoline bronchodilator as it relaxes bronchial smooth muscle by acting on beta-2 receptors, with little effect on cardiac muscle contractility. We administer it according to the following dosage: for adults, nebulizer, dilute 0.5 ml (2.5 mg) of 0.5% inhalation solution in 1-2.5 ml of saline every 6 h and according to the patient's symptoms; for paediatric patients < 5 yr, nebulizer, dilute 0.25-0.5 ml (1.25-2.5 mg) of 0.5% inhalation solution in 1-2.5 ml of saline every 6 h and ac-

ording to the patient's symptoms. We use bronchodilators carefully with hyperthyroidism, diabetes mellitus, and cardiovascular disorders; adverse effects include tachycardia, palpitations, tremor, insomnia, nervousness, nausea, and headache.

We add heparin to the nebulizer - heparin plays an important role in breaking the cast and debris formed as a result of inhalation injury.

Corticosteroids like Decardon and Soliocortif are added to medications. They play an important role as anti-inflammatory elements.

6. Surgical interference

In circumferential chest burns we perform escharotomy to prevent any restriction of chest expansion during respiration. This is done by making two lateral incisions in the chest and a mid-line incision in the chest wall plus two parallel incisions.

Tracheostomy can be performed in cases of severe inhalation injury and airway obstruction that makes intubation difficult.

Results

This study covered 130 burn patients (61 male and 69 female) with inhalation injury admitted to Menoufiya University Hospital Burn Center from January 2004 to April 2008 (Tables I-III).

Our study found that the presence of inhalation injury, increasing burn size, and advancing age were all associat-

ed with increased mortality ($p < 0.01$). The incidence of inhalation injury in our study was 46.3% (130 patients were reported as having inhalation injury out of a total number of 281). The overall mortality among patients with inhalation injury was 41.5% (54 of 130) compared with 7.2% (11 out of 151) among patients without inhalation injury. These statistical data make it clear that inhalation injury is an important factor in the prediction of burn patient mortality.

Discussion

Over the past twenty years, there has been a remarkable improvement in the chances of survival of patients treated in burn centres. A simple, accurate system for objectively estimating the probability of death would be useful in counselling patients and making medical decisions. This remarkable success can be attributed to a number of therapeutic developments, including vigorous fluid resuscitation, the early excision of burn wounds, advances in critical care and nutrition, powerful topical and systemic antibiotics, and the evolution of specialized, multidisciplinary burn centres. It has been over a decade since the present authors published their first manuscript on inhalation injury.⁵ In a review of the article published the following year it was reported that inhalation injury was one of the main factors - possibly the most important - responsible for mortality in thermally injured patients.^{6,7} Although it may still be true today, many new things have been learned that have reduced morbidity and mortality in

Table I - Blood gases of burn patients with inhalation injury in relation to burn percentage, showing that blood gas has a significant relationship with inhalation injury.

Gases	No inhalation injury	Inhalation injury	t-test	p-value
pH	7.37 ± 0.06	7.3 ± 0.12	2.6	<0.05
pO ₂	96.7 ± 18.6	77.2 ± 22.5	4.3	<0.001
pCO ₂	31.5 ± 6.6	35.8 ± 9.1	2.4	<0.05
Base deficit	4.8 ± 2.5	8.2 ± 3.3	5.9	<0.001

Table II - Mortality rate with and without inhalation injury, indicating that inhalation injury plays a significant role in the prediction of burn patient mortality.

Patients		Living		Deceased		Total		χ^2	p value
		Number	Percentage	Number	Percentage	Number	Percentage		
Total	Positive inhalation injury	76	65.5	54	83.1	130	46.3	46.1	<0.001
	Negative inhalation injury	140	34.5	11	16.9	151	53.7		
	Total	216	100	65	100	281	100		

Table III - Mortality among burn patients with inhalation injury in relation to age

Age (yr)	Living		Deceased		χ^2	p value
	Number	Percentage	Number	Percentage		
<10	11	15.3	3	5.6	14.1	<0.05
11-20	18	25.0	12	22.2		
21-30	20	26.3	16	29.6		
31-40	16	21.1	6	11.1		
41-50	7	9.7	5	9.3		
51-60	4	5.6	6	11.1		
>61	0	0	6	11.1		

Table III shows that all burn patients with inhalation injury and over 60 years of age died. This confirms that the presence of more than one risk factor increases mortality.

burn patients who have sustained a concomitant inhalation injury.⁸ While some have reported that little progress has been made in reducing the mortality associated with inhalation injury,⁹ others have noted significant progress,⁸ improved techniques for patient ventilation,¹⁰ more aggressive fluid therapy, and better techniques for trachea bronchial toilet. Better understanding of the pathophysiology caused by cutaneous burns and smoke in the lung will lead to even further advances in the treatment of such lesions.¹¹

The most commonly used predictors of mortality in burns are age, burn size, and the presence of inhalation injury. In the past, burns in over 80% TBSA that were mostly full-thickness were often considered fatal, especially in children and the elderly. In the past 15 years, advances in burn treatment have increased survival rates in patients treated in specialized burn centres, and a number of factors - larger burn size, presence of inhalation injury, delayed intravenous access, lower haematocrit and lower base deficit on admission, higher serum osmolarity on arrival at the authors' hospital, sepsis, inotropic support requirement, platelet count < 20,000, and ventilator dependency during the hospital course - significantly predict increased mortality. It has been found the patients most likely to die are the very young, those with limited donor sites, those who have inhalation injury, those with delayed resuscitation, and those with burn-associated sepsis or multi-organ failure.¹²

We conducted a review of all 130 burn patients with inhalation injury admitted in 2004/2008 to Menoufiya Burn Center General Surgery Department in Menoufiya University Hospital.

As already said, our study found that the presence of inhalation injury, increasing burn size, and advancing age were all associated with increased mortality, with an inhalation injury incidence of 46.3% (130 patients out of 281) and an overall mortality rate in patients with inhalation injury of 41.5% compared with 7.2% in patients without inhalation injury, clearly indicating the importance of

inhalation injury as a factor for the prediction of mortality in burn patients.

Another study was performed in Tokyo to evaluate the impact of inhalation injury on burn patient mortality. Out of 5560 patients admitted to 13 burn facilities of the Tokyo Burn Unit Association between 1984 and 2002, 1690 patients (30.4%) had experienced inhalation injury. The overall in-hospital mortality rate of patients with inhalation injury was higher than that of patients without inhalation injury (33.6% versus 8.1%), close to the ratio found in our study. It was found that the results of the multivariate analysis indicated that inhalation injury, full- and partial-thickness burn size, and age were independent predictors of outcome (relative risk, 2.58 [2.03-3.29], 1.10 [1.09-1.11], 1.06 [1.06-1.07], 1.05 [1.05-1.06], respectively). The authors concluded that inhalation injury was the most important predictor of overall mortality among burn patients in Tokyo.¹³

A retrospective study conducted in the Kansas University Medical Center, Burnett Burn Center, Kansas City, KS, USA, regarding all patients (no. = 201) aged 75 yr or older admitted to a university-based burn centre between 1972 and 2000, examined the following variables: age, sex, total body surface area burned, abbreviated burn severity index, inhalation injury, time interval between burn and operative intervention, number of surgical procedures, number of pre-morbid conditions, and mortality. Mortality significantly increased with inhalation injury ($p < 0.01$). As we found in our own study, the fatality risk increased by 400% with inhalation injury.¹⁴

In a study of 710 burn patients in a Spanish burns centre it was found that the mortality rate among burn patients with inhalation injury was 66%.¹⁵

In a retrospective study analysing 5264 patients treated in the burns centre at Gulhane Military Medical Academy in Turkey from 1 January 1986 to 31 December 1995, it was found that 134 patients had inhalation injury, of whom 82% died¹⁶ (in our study 83.1% of burn patients with inhalation injury died). Three risk factors for death

were identified: age greater than 60 yr, more than 40% TBSA burned, and inhalation injury. The mortality formula they developed predicts respectively 0.3%, 3%, 33%, or approximately 90% mortality depending on whether zero, one, two, or three risk factors are present. The results of the prospective test of the formula were similar. A large increase in the proportion of patients who choose not to be resuscitated has complicated comparisons of mortality over time.¹⁷

In another study the outcome of 1385 patients admitted to a burns unit over a 20-year period was evaluated in relation to the presence of three major risk factors for death: age 60 years or over, TBSA 40% or more, and the presence of inhalation injury. Mortality was respectively 9.9%, 48.0%, and 90.5%. It was found that nearly all the patients who died presented at least one risk factor. In the presence of three risk factors the prognosis following burns was particularly compromised.¹⁸

Our study found an overall mortality rate among all burn patients of 23.13% (65 patients out of 281).

In a study performed on all patients admitted to the burns unit of Alexandria Main University Hospital over a 1-year period (1999), the mortality rate was 33%.¹⁹

In a retrospective analysis of 435 consecutive admissions to a regional burns unit in Saudia Arabia over an 8-year period, the fatality rate was 7.4%.²⁰

The already mentioned study of 5264 patients treated in a burns centre in Turkey reported an overall mortality rate among in-patients of 18.2%,¹⁶ while the analysis of mortality rates and related factors in the Spanish burns centre referred to, based on 710 patients, reported an overall mortality rate of 6.6%.¹⁵ The marked difference in mortality rate between that of the Spanish centre and our own

was due to the average burn size of 14% TBSA in the Spanish centre.

Between February 1987 and July 1990, among the 844 patients admitted to the Grady Memorial Hospital Burn Unit, the mortality rate was 8.5%.²¹ One-half of the burns were less than 10% TBSA, which explains the low mortality rate compared with that of our Centre.

As a measure of outcome in a group of burn patients, the mortality rate is useless and misleading unless the burn severity parameter is standardized. For many years it was accepted that the extent of the burn and the age of the patient were the two most important determinants of mortality probability in burn patients, while it is now recognized that smoke inhalation is another important risk factor and must be included in the computation of mortality probability.

Inhalation injury greatly increases the incidence of respiratory failure and of the acute respiratory distress syndrome. It is also the cause of most early deaths in burn victims. The mortality rate following smoke inhalation ranges from 45% to 78%.²²

Conclusion

Blood gases are a good indicator for early detection of inhalation injury, which increases the incidence of mortality in burn patients - approximately 80% of fire-related deaths are due not to the burn injury itself but to inhalation of toxic products, especially carbon monoxide and hydrogen cyanide gases. Early detection and early management of inhalation injury are thus essential for the survival of burn patients.

RÉSUMÉ. Les lésions causées par l'inhalation provoquent un incrément notable de l'incidence de l'insuffisance respiratoire et du syndrome de détresse respiratoire aiguë. Ces lésions sont en outre la cause de la plupart des décès précoces des victimes des brûlures. Le but de cette recherche était d'étudier l'incidence, le premier diagnostic, les complications et la gestion des lésions par inhalation et de discuter le rapport entre les lésions par inhalation et la mort des patients brûlés. L'étude s'est occupée de 130 patients brûlés atteints de lésions par inhalation hospitalisés au Centre des Brûlés de l'Hôpital Universitaire de Menoufiya en Egypte entre janvier 2004 et avril 2008 (61 mâles et 69 femelles). Nous avons constaté que certains facteurs, comme la présence de lésions par inhalation, l'augmentation progressive de l'extension de la brûlure et l'âge avancée, étaient invariablement associés à un incrément de mortalité ($p < 0.01$). Dans notre étude l'incidence des lésions par inhalation était 46,3% (130 patients sur 281). La mortalité totale des patients atteints de lésions par inhalation était 41,5% (54 patients sur 130) par comparaison avec un taux de 7,2% pour les patients qui ne présentaient pas ce type de lésion (11 patients sur 151). Ces données statistiques démontrent que les lésions par inhalation constituent un facteur important pour ce qui concerne la prédiction de la mortalité des patients brûlés. Approximativement 80% des décès dus au feu ne sont pas causés par les lésions directes aux voies aériennes mais par l'inhalation des produits toxiques du feu, et particulièrement de certains gaz (oxyde de carbone et cyanure d'hydrogène). Les lésions par inhalation sont causées principalement par les brûlures thermiques, pour la plupart limitées aux voies aériennes supérieures. Dans les patients atteints de lésions par inhalation, il est possible d'observer des complications importantes dans les voies aériennes et le système pulmonaire, comme aussi des problèmes systémiques, ce qui augmente le taux de mortalité des patients brûlés.

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