International Journal of Biomedical and Health Sciences
Vol. 6, No. 1, March 31, 2010
Printed in Nigeria

0794-4748/2010 $12.00 + 0.00
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http://www.asopah.org

IJBHS 2009132/6101

Acute Respiratory Distress Syndrome in a Neonate: A case report

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(Received November 17, 2009)

ABSTRACT: Acute respiratory distress syndrome (ARDS) describes an acute diffuse inflammatory lung injury caused by permeability pulmonary edema which occurs as a result of alveoli capillary endothelial damage that develops in response to an initiating injury or illness.This condition occurs unnoticed among seriously ill patients in the hospital wards. Despite advance research in inflammatory mechanism , ventilatory support and haemodynamic control,the mortality rate of ARDS remains high,50 -75%.In this report, we present a term,3kg live female baby who became breathless immediately after birth. There was generalized refractory cyanosis(despite adequate ventilation),tachycardia (≥140b/minute) and bibasal coarse crepitations and rhonchi. Apgar score was $4^{1}3^{10}$ .She was delivered by a 29year old Primi gravida via emergency caesarian section(CS) indicated by passage of fresh meconium stained liquor.Findings during Surgery were cord round neck and meconium filled uterine cavity. Mother was not diabetic nor hypertensive.She neither had any major medical therapy nor trauma prior to delivery.

Key words: Acute Respiratory Distress, Inflammation, Endothelial damage, Non Cardiogenic Pulmonary oedema, Cyanosis, Meconium inhalation, Neonate.

Introduction

Acute respiratory distress syndrome (ARDS) is a form of acute respiratory failure resulting from permeability pulmonary edema as a result of alveoli capillary endothelial damage. It was initially regarded as adult respiratory distress syndrome because of its pathological similarities(hyaline membrane) to the infant respiratory distress syndrome caused by surfactant deficiency in premature babies.[1,2] However, It is now called acute respiratory distress syndrome and its pathophysiology is similar in all age groups(no age difference).[1,3,4] The risk factors for ARDS are listed in Table 1.

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Table 1: Risk factors for ARDS

Direct	Indirect
Aspiration Pneumonitis	Sepsis
Severe Pneumonia	Major Trauma
Smoke Inhalation	Multiple Blood Transfusion
Lung Contusion Fat Embolism	Acute Pancreatitis
Amniotic Fluid Embolism	Extensive Burns
Chemical Inhalation (Silo Filler's Lung)	Hypovolaemic Shock
Oxygen Toxicity	Anaphylaxis
Reperfution Pulmonary Oedema	Disseminated Intravascular Coagulation
(Post Lung Transplantation)	Malaria

The exact mechanism of the primary insult in the alveoli capillary membrane remains unknown,but,it is thought that injury on the lungs leads to compliment or kinin activation which chemo attracts neutrophils to the alveoli capillary membrane.[4,5,6] Neutrophils release various toxins including oxygen radicals which damage cell walls by lipid peroxidation.[5,6,7] Contrarilly,experimental ARDS has been demonstrated in neutropenic animals and revealed a leak of plasma protein from capillaries into the alveoli with inactivation of surfactant.[6,7,8] This causes interstitial and alveoli edema that collapses the lung unit causing shunt hypoxaemia,loss of gas volume ,stiff lungs and reduced compliance. Time interval may not be specific but ARDS features usually develop 12 to ≥48hours after the initial lung injury. It is characterized by the following:

- **History of an initial injury or illness**
- **Hypoxaemia refractory to Oxygen therapy e.g PO_2 < 8KPa (60mmHg) on ≥40% Oxygen**. The degree of hypoxaemia may be expressed as the ratio of arterial Oxygen tension (PO_2) to the fractional inspired Oxygen concentration (F_1O_2 /100% Oxygen = F_1O_2 of 1).In ARDS, PO_2 / F_1O_2 is < 26 KPa (200mmHg).
- **Bilateral diffuse infiltrates on Chest X ray**.
- **No evidence of cardiogenic pulmonary edema (e.g Pulmonary capillary wedge pressure ≤ 18mmHg)**.

Deterioration of patients condition is monitored with the pulse rate, respiratory rate, blood pressure, urine output and the level of consciousness. Arterial blood gas measurements or and pulse oximetry are very vital in the management of ARDS. Optimal management of the initiating illness or injury combined with respiratory and nutritional support are the main stay of treatment.[9,10] Multi organ failure(MOF) which complicates ARDS is often the main cause of death and requires additional specific management. Anti inflammatory drugs have failed in the management of ARDS and despite advance research in inflammatory mechanism , ventilatory support and hemodynamic control, the mortality rate of ARDS remains high(50 - 75%),especially in the developing world.[4,11,12] Few survivals may have lung fibrosis leading to impaired ventilation perfusion mismatch. Suffix to emphasis that some patients still make remarkable recovery. Prognostic factors include, the age of patients, risk factors for ARDS(Pneumonia 86%,trauma 38%), and the number of organs involved (3 organs involved for > 1 week is fatal).[12,13,14]

This is the first reported case of Acute Respiratory Distress Syndrome in neonates, from the Niger Delta region of Nigeria. The aim of reporting this case is to encourage surveillance of the disease among

Physicians because the risk factors are encountered in our daily medical practice. ARDS is not a rare disease, as it has been assumed over the past years. it is a common pathway for most seriously ill patients in the hospital.

CASE REPORT

A live female term baby was delivered at1300hours on the 12th of April 2008 by emergency caesarian section on account of fetal distress as evidenced by passage of fresh meconium stained liquor and fetal tachycardia (170b/m)at the University of Benin Teaching Hospital (UBTH),Benin City. Caesarian section findings revealed cord round neck, endometrium filled with concentrated meconium. Apgar score at birth was 4^{1},3^{10}. She had breathlessness immediately after birth(38cycles /minute), cyanoses, tachycardia (>140 b/minute)and bilateral diffuse coarse crepitations and rhonchi. She was intubated, suctioned and placed on 20 – 30% Oxygen,2-3liters/min. Hypoglycemia, hypovolaemia and low serum bicarbonate were corrected immediately. She was placed on intravenous ampicillin. Urea and creatinine levels were normal. Genotype and blood group were AA and O positive respectively. There was no malarial parasite seen on blood film. Pulse oximetry was always between 80% and 90%. Emergency chest Xray was not done as doctor battle to save the life of this baby. The working diagnoses was Acute Respiratory Distress Syndrome secondary to severe asphyxia caused by cord round neck and meconium aspiration.Baby's colour became pink temporarily during resuscitation. However, her condition deteriorated and she died 14hours after birth. Mother was a 29 year old primigravida. She was not a known hypertensive or diabetic. She attended antenatal clinic regularly and was not on therapy for any disease condition. However, her admission into the labour ward was delayed.

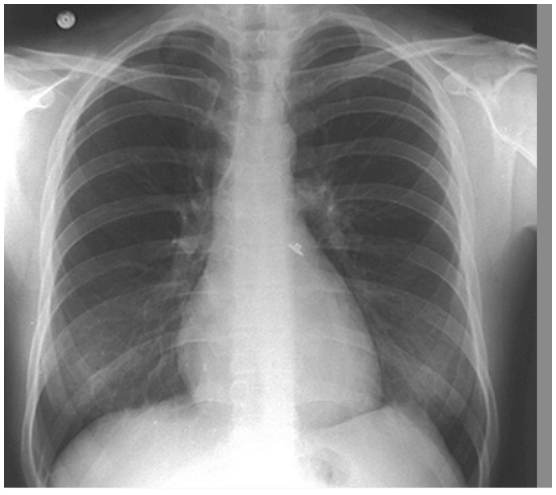

Figure 1:Posterior anterior view of a normal Chest Xray

Discussion

Acute respiratory distress syndrome occurs as a result of endothelial damage leading to permeability pulmonary edema. The risk factors for ARDS are very common especially in patients who have poor access to health facilities, whose treatment has been delayed and seriously ill inpatients. The factors that contributed to the development of ARDS in this report included delayed delivery of the baby, associated with meconium aspiration that was confirmed by the color and nature of the suctioned airway fluid.[15] Other suspected risk factors included hypovolaemic shock, sepsis which were corrected immediately after birth.

The time of onset of this present report is not known because inhalation of meconium occurred *in utero*. This seems to contradict the usual period of unset of 12 – ≥48hours in ARDS. This underscores the fact that the duration of the initiating risk factors is not as important as the dose of the risk factors.

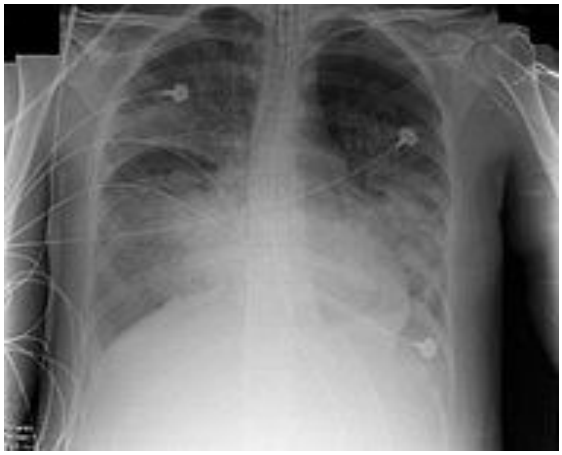

Figure 2: Anteroposterior chest radiograph of a patient with acute respiratory distress syndrome.There are bilateral diffuse alveolar and reticular infiltrates consistent with pulmonary edema. Note the absence of cardiomegally and pleural effusions that are prominent in cardiogenic pulmonary edema.

Furthermore, there is no age discrepancy in the risk factors and pathophysiology of ARDS. The critical condition of the patient as evidenced by the deep level of consciousness, tachypnea with subsequent hypopnea, tachycardia, bilateral coarse crepitations, refractory hypoxaemia, acidosis (low blood pH<7.2) and low bicarbonate level < 20mmol/L agree with the documented findings in ARDS reported by some researchers.[15,16] The prognosis was very poor due to the tender age, high dose of inhaled meconium and associated *in utero* neck cord tie. However, reports on prognosis of ARDS has been controversial. In spite of intensive medical research and advances in ventilatory techniques and hemodynamic control, the mortality rate is still very high.[9,11].This is at variance with the report of some other researchers who reported the 1993 National mortality follow back study database findings which stated that the annual ARDS mortality is slowly declining but that men and blacks have a higher mortality rate compared with women and other race.[17] This present report revealed that the mortality of ARDS in the developing countries will remain high because of poor health facilities, diagnostic dilemma and low disease surveillance. Mortality is also high in seriously ill inpatients and out patients who make orthodox facility a last resort.

However, a better understanding of the pathophysiology of the disease has produced management strategies that have translated into evidenced based improvement in outcome. Studies with small groups of patients indicated that the use of beta agonists in patients with ARDS is safe, with a trend towards improved oxygenation and a decrease in peak and plateau ventilatory pressures.[18] Steroid use is still controversial as it does not improve prognosis. However, judicious use of fluids, ionotropic agents and antibiotics is needed. Various studies demonstrated that the use of prostaglandins, antibodies, and receptor antagonists to various cytokines, N-acetylcysteine (Mucomyst) or aerosolized exogenous surfactant in multicenter trials failed to reduce mortality in ARDS.[19,20] Nitric oxide, hydrallazine, Nitroprussides have not been beneficial.[4] Investigations into the role of gene mutations and gene polymorphisms have given insight into possible genetic susceptibility for the development and outcome of ARDS.[21] As more strategies and drugs are developed, there is hope that control of this fatal disease will be possible. For now, treating the risk factors, avoiding ventilator-induced injury, managing fluids judiciously and providing supportive care remain the cornerstone of management.

ACKNOWLEDGMENT: Special thanks to **MRS GAIUS OBASEKI (ADNS -UBTH)** and her nursing crew (Matrons Evbuomwan, Obayanju etc) for their motherly care during the period.

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