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CASE HISTORY

Meconium aspiration syndrome and persistent pulmonary hypertension of the newborn

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Hypercapnia;
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Meconium aspiration syndrome;
Respiratory acidosis;
Continuous positive airway pressure;
Developmental care;
Prone position

Abstract The focus of this article will be to critically assess the nursing care provided to an infant, "James", with meconium stained aspiration (MAS) and persistent pulmonary hypertension (PPHN) with particular regard to James's hypercapnia, hypoxia, tachypnoea, low mean systemic blood pressure (BP), low pre and post ductal oxygen saturations and cool extremities. The hypercapnia, hypoxia and tachypnoea and underlying physiological mechanisms will be related to the pathophysiology of MAS, and the low mean systemic BP, oxygen saturations and cool extremities will be related to the PPHN. Knowledge of the underlying physiological mechanisms which will then be used to discuss the nursing care provided, and the impact it had on James's condition. The overarching aim of nursing care was to reduce James's need for respiratory support and return his blood gases to normal. The interventions that supported this such as positioning, fluid balance and comfort measures will be looked at in more detail. Other aspects, including drugs that were used will also be discussed.
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Case study

A pseudonym, James, has been used in order to protect the identity and confidentiality of the baby and his family (Nursing Midwifery Council, 2015). James was born at 40 + 1 weeks. His mother had presented to hospital in spontaneous labour with meconium stained liquor and reduced fetal movements. A fetal tachycardia, maternal tachycardia and maternal pyrexia, combined with a suspicious cardiotocography resulted in a decision to expedite delivery via forceps. James had a shoulder dystocia and was delivered through thick meconium. His mother did not receive intrapartum

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antibiotics — for unknown reasons. At birth James was centrally cyanosed with poor tone and required stimulation, airway management, oxygen via a facemask and Continuous Positive Airway Pressure (CPAP). (Resuscitation Council UK, 2010). His apgar scores were 3, 7 and 9. He was then transferred to the Neonatal Intensive Care Unit (NICU) for ongoing respiratory support, observation and partial septic screen.

James became tachypnoeic and developed an oxygen requirement of 100% FiO2. His blood gases showed hypercapnia, hypoxia (see Table 1 below) and his mean systemic blood pressure (BP) was low. James had cool extremities. A diagnosis of meconium aspiration syndrome (MAS) was made which was presumed to have resulted in secondary persistent pulmonary hypertension (PPHN). A decision was made not to intubate and mechanically ventilate but to continue with CPAP and to gradually attempt to reduce the amount of FiO2 depending on his clinical condition. His ongoing care included CPAP, oxygen, full cardiac and respiratory monitoring (including regular blood gases) with non invasive blood pressure checks, nursed prone in an incubator, intravenous fluids, intravenous antibiotics and minimal handling.

James's x-ray showed wet streaky lungs consistent with MAS. An echocardiogram was not carried out so the diagnosis of PPHN was not confirmed. Over the course of twelve hours his blood gases became more normal and his FiO2 was reduced to 21%. James's c-reactive protein came back as less than one.

Introduction

Meconium aspiration syndrome

Meconium aspiration syndrome (MAS) is defined as respiratory distress with changes on x-ray, unexplained by any other underlying pathology, and accompanied by meconium stained amniotic fluid (MSAF) at (like James), or prior to, delivery (Stenson and Smith, 2012). MSAF is associated with fetal hypoxia although Monen et al. (2014) suggest that, as it occurs in 30—40% of post term deliveries, it maybe a physiological consequence of foetal maturation. Of that number up to 10% go on to develop MAS (Edwards et al., 2013). Supplemental oxygen is the main therapy, although approximately one third of affected infants will require mechanical ventilation (Dargaville, 2012). James required CPAP to improve his lung function and gaseous exchange.

Persistent pulmonary hypertension

Persistent pulmonary hypertension (PPHN) is a failure of the fetal circulation to adapt to extra-uterine life. It is characterised by a high pulmonary vascular resistance (PVR) and can sometimes be associated with a low systemic vascular resistance (SVR) (Nair and Lakshminrusimha, 2014) and should be suspected when the baby's oxygen requirement is out of proportion to the level of pulmonary disease (Bendapudi et al., 2015). It affects 2 in 1000 live births, and is associated mostly with term or near term infants (Nair and Lakshminrusimha, 2014). Mortality remains at 10%. PPHN is associated with MAS, pneumonia, sepsis, pulmonary hypoplasia, congenital diaphragmatic hernia, and maladaptation of the pulmonary vascular bed both in utero and ex utero (Nair and Lakshminrusimha, 2014; Teixeire-Mendonca and Henriques-Coelho, 2013). Treatment strategies include respiratory support, which for James was CPAP, but for more severe PPHN high frequency ventilation, surfactant and pulmonary vasodilators such as nitric oxide may be required.

MAS, hypercapnia, hypoxia and tachypnoea

Fetal distress leads to fetal hypoxia at which point the fetus passes meconium resulting in meconium stained fluid (MSAF). Meconium is inhaled, and causes hypoxia in infants in three ways — airway obstruction, chemical pneumonitis, and surfactant dysfunction (Mokra and Calkovska, 2013). Meconium blocks the airways and can lead to gas

Table 1 Normal values taken from Boxwell (2010).

	pH	pCO2 (kPA)	pO2 (kPA)	Base excess (mmol/l)	cHCO-3 (mmol/l)	Sodium (mmol/l)	Lactate (mmol/l)	Systemic BP mean (mmHg)	Respiratory rate per minute	Oxygen saturations in air (%)
Normal Values	7.3 to 7.4	4.5 to 6.0	8.0 to 12	−7 to +3	18 to 25	135 to 146	0.5 to 2.0	30 to 55	30 to 60	⩾95
James's values	7.23	8.1	5.68	−11.3	15.5	132.9	3.9	28	70	40 to 50

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