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Review

The accuracy of clinical parameters in the prediction of perinatal pulmonary hypoplasia secondary to midtrimester prelabour rupture of fetal membranes: A meta-analysis

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ABSTRACT

Prediction of pulmonary hypoplasia after midtrimester preterm prelabour rupture of membranes (PPROM) is important for optimal management. We performed a systematic review to assess the capacity of clinical parameters to predict pulmonary hypoplasia. A systematic literature search in EMBASE and MEDLINE was performed to identify articles published on pulmonary hypoplasia in relation to midtrimester PPROM. Articles were selected when they reported on one of the following clinical parameters – gestational age at PPROM, latency period and degree of oligohydramnios – and when they allowed the construction of a two-by-two table comparing at least one of three clinical parameters to the occurrence of pulmonary hypoplasia. The selected studies were scored on methodological quality, and sensitivity and specificity of the tests in the prediction of pulmonary hypoplasia and lethal pulmonary hypoplasia were calculated. Overall performance was assessed by summary receiver operating characteristic (sROC) curves that were constructed with bivariate meta-analysis. We detected 28 studies that reported on the prediction of pulmonary hypoplasia. Prediction of lethal pulmonary hypoplasia could be analysed separately in 21 of these studies. The quality of the included studies was poor. The estimated sROC-curves showed that gestational age at PPROM performed significantly better than the two other parameters in the prediction of pulmonary hypoplasia. The accuracy in the prediction of lethal pulmonary hypoplasia was similar. In women with midtrimester PPROM, pulmonary hypoplasia can be predicted from the gestational age at PPROM. This information should be used in the management of women with early PPROM.

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Contents

1. Introduction

Midtrimester prelabour rupture of membranes is associated with an increased risk of altered pulmonary development leading to pulmonary hypoplasia. In fetal lung development a critical interval, the canalicular phase, exists between 16 and 28 weeks gestation.

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Preterm prelabour rupture of membranes (PPROM) before 26 to 28 weeks can delay lung development and can cause pulmonary hypoplasia [1].

Pulmonary hypoplasia poses a serious threat due to its high mortality and morbidity rates. It can occur as severe respiratory failure leading to early neonatal death, as respiratory insufficiency with pulmonary haemorrhage, bronchopulmonary dysplasia, or subacute lung disease, or as mild and even transient respiratory disease [2]. Perinatal mortality approximates 70% in most series (55–100%) [3].

In a review of 11 studies on midtrimester PPROM, the reported incidence of pulmonary hypoplasia secondary to midtrimester PPROM ranged widely, from 1% to 48% [4]. In another review the reported incidence also varied widely, from 0% to 24% [1]. This wide range in prevalence is partly explained by the absence of uniform pathological and clinical definitions. Histological findings form the basis of the diagnosis but autopsy is not always allowed or reported uniformly or informed consent is not obtained [2]. An internationally recognized definition of pulmonary hypoplasia does not exist and it rather is a diagnosis of exclusion [5]. Congenital pneumonia, infant respiratory distress syndrome (IRDS) and pulmonary hypoplasia sometimes occur simultaneously and have overlapping symptoms [1]. Moreover there are methodological problems, such as differences in follow-up and lack of blinding.

Once midtrimester PPROM has occurred, an assessment of the probability of pulmonary hypoplasia is important both for clinical decision making and counselling of patients. Previous studies have shown that gestational age at the time of rupture of the membranes has a strong relation with the occurrence of pulmonary hypoplasia [6,7]. Other factors associated with pulmonary hypoplasia are the duration of the rupture of the membranes and the degree of oligohydramnios [8]. To our knowledge, the predictive capacity of these clinical parameters for the presence of hypoplasia has not been assessed systematically. Therefore, we performed a meta-analysis on this subject. The aim of the present analysis was to assess the predictive capacity of clinical parameters in the prediction of pulmonary hypoplasia.

2. Materials and methods

We searched for studies that reported on neonatal outcome after midtrimester rupture of fetal membranes. We performed an electronic search of MEDLINE (Inception to 03/2008) and EMBASE (Inception to 03/2008), and checked reference lists of known reviews and primary articles to identify cited articles not captured by electronic searches. There were no language restrictions.

To be included, studies had to fulfil the following criteria. The study had to report on the outcome of pregnancies complicated by PPROM between 14 and 28 weeks of gestational age.

Neonatal outcomes after midtrimester PPROM had to include the presence of pulmonary hypoplasia. The diagnosis of pulmonary hypoplasia could be based either on clinical and radiological findings or on findings at autopsy. In the analysis we distinguished two types of hypoplasia, i.e. lethal hypoplasia and any form of hypoplasia. Lethal hypoplasia was defined as hypoplasia resulting in the death of the fetus or neonate due to hypoplasia. Fetuses with autopsy proven lung hypoplasia after early pregnancy termination were also included in the lethal group. Any form of hypoplasia was defined as the sum of lethal hypoplasia and non-lethal hypoplasia. For each study, we calculated the prevalence of any form of pulmonary hypoplasia, and if possible of lethal pulmonary hypoplasia.

Studies also had to report on one of the three clinical parameters: gestational age at PPROM, latency between PPROM and delivery, or oligohydramnios. The method by which oligohydramnios was defined was also documented, if applicable.

The following characteristics of each study were registered: (1) sampling (consecutive versus other), (2) data collection (prospective versus retrospective), (3) study design (cohort study versus case–control study), (4) blinding (present or absent), (5) verification bias and (6) selection bias [9]. Study characteristics were scored by two of the authors (*ASPvT* and *DvdH*). In case of disagreement, the judgement of a third author (*BWM*) was decisive.

2.1. Analysis

2.1.1. Data analysis

For each study, we constructed a two-by-two table cross-classifying one or more of the three clinical parameters and the presence of any form of pulmonary hypoplasia, and if possible, of lethal pulmonary hypoplasia separately. Two-by-two tables were constructed independently by two of the authors (*ASPvT* and *DvdH*). In case of disagreement, the judgement of a third author (*BWM*) was decisive.

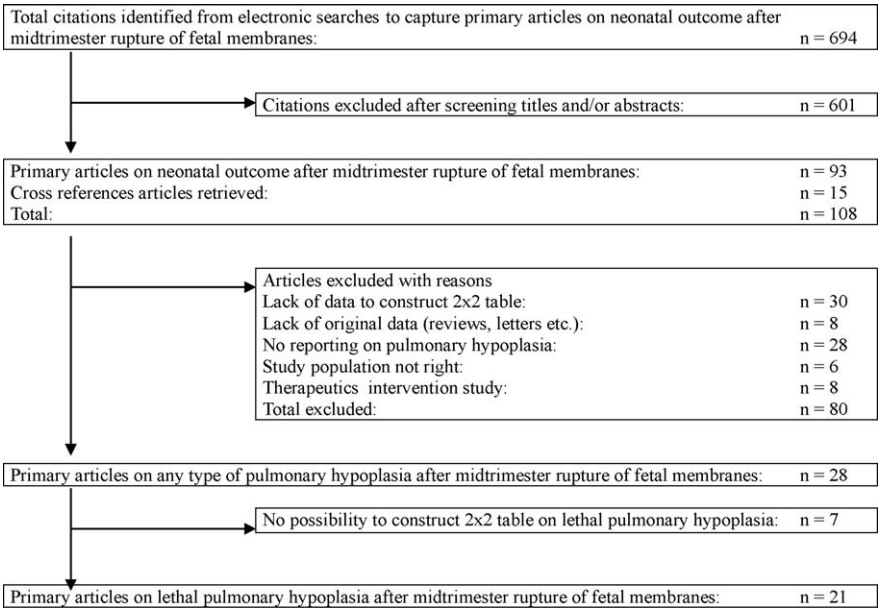

Fig. 1. Process of literature identification and selection.

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