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Neurodevelopmental outcome of late-preterm infants: literature review

Short title: Neurodevelopmental outcome of late-preterm infants

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Abstract

Late-preterm infants are characterized by a birth term from 34^{0/7} to 36^{6/7} weeks of gestation. A foetal brain at 34^{0/7} weeks of gestation weighs only 65% of the full-term newborn brain, which suggests a particular cerebral vulnerability to injury during this 6-week period. Epidemiological studies reporting the neurological outcomes of late-preterm infants exhibit large methodological heterogeneity that inhibits clarity on this issue. However, contradictory results and odds ratio values near neutral reveal probable moderate neurodevelopmental delay in late-preterm infants. This observation reflects the variable neurological outcomes of this population according to multiple perinatal factors. Therefore, the current challenge is to define efficient screening strategies to determine infants requiring specific follow-up.

1 Introduction

The neonatal brain is particularly vulnerable to various challenges in births before 32 weeks of gestation. Brain lesions specific to this developmental window consist of intra-ventricular haemorrhages and a diffuse disruption of brain development corresponding to the encephalopathy of premature newborns [1]. In parallel, neurodevelopmental alterations were described in children born before 32 weeks of gestation in several cohorts worldwide. These alterations concerned motor or sensorial functions as well as emotional, behavioural, and cognitive skills [2–6]. The follow-up of these vulnerable children, up to 7 years of age, is organized through regional networks in France. Furthermore, the French Health Authority (Haute Autorité de Santé) will soon publish guidelines to homogenize modalities of this follow-up.

Infants born from 34^{0/7} to 36^{6/7} weeks of gestation correspond to late-preterm infants (LPIs). This group of infants represent approximately 70% of infants born before 37 weeks of gestation in developed countries (i.e. 4–5% of live births) [7]. Few of these infants require intensive care during the neonatal period when compared with younger preterm infants [8]. Furthermore, neonatal brain lesions observed in early preterm infants are scarce, and do not arouse medical attention for potential neurodevelopmental problems [8]. The neurodevelopmental outcomes for this group of infants remain elusive for neonatologists.

This review will successively report on brain development from 34 weeks of gestation to term-equivalent age, on the available epidemiological data on the neurodevelopmental outcomes of LPI, and on neonatal risk factors associated with worse outcomes.

2 Methods

Eligible studies included relevant papers addressing LPIs issues associated with brain morphology, neurodevelopmental outcome, and neonatal factors influencing the neurologic prognosis. All studies were published in the Medline electronic database, in French or

English, between 1 January 1995 and 31 December 2018. Searches used the Medical Subject Headings (MeSH) “Infant, Premature” with subheadings “epidemiology” and “growth and development” or “Child Development” with subheadings “complications” and “physiology” and cross-referencing. In total, 59 articles were considered based on abstracts. No controlled trials were found out. A total of 26 observational studies, which reported on the neurodevelopmental follow-up of moderate-preterm infants and LPIs from 12 months of age to late childhood, were selected. Seven literature reviews that focused on various aspects of LPIs neurodevelopment (motor, cognitive, emotional, behavioural) up to adulthood were also selected.

Infants with congenital malformations or genetic syndromes were not addressed in this review even though they are twice as prevalent in the LPI population when compared with the full-term infant population [9]. Finally, infants from multiple pregnancies were also excluded.

3 Late prematurity and effect on early brain development up to term-equivalent age

3.1 Normal brain development from 34^{0/7} weeks of gestation to term-equivalent age

From 34^{0/7} to 40^{0/7} weeks of gestation, a foetal brain gains one third of its weight at term. Cortex and white matter volumes are multiplied by two and five, respectively, throughout this 6-week period [10]. This time period is therefore critical for immature brain growth. Intense synaptogenesis and dendritic arborisation are observed with a key role of subplate neurons essential for thalamic–subcortical connectivity [11].

3.2 Brain MRI of late-preterm infants at term-equivalent age

Moderate-preterm infants and LPIs exhibited maturation delay on magnetic resonance imaging (MRI) of the brain at term-equivalent age. Whole brain and cerebral sub-part measurements such as central grey matter (-1.4%, $p = 0.006$) and cerebellum (-1.9%, $p = 0.002$) revealed a significant size reduction in comparison with term infants. Sub-arachnoid

spaces were larger, evoking features observed in encephalopathy of early preterm infants. Cortical folding delay (odds ratio [OR] = 0.04, 95% confidence interval [CI] [0.01–0.3]) and myelination reduction of the internal capsule posterior limb (OR = 0.2, 95% CI [0.1–0.8]) were observed [12]. However, only cortical folding delay and volumetric measurements concerning the whole brain, grey matter, white matter, cerebellum, and peri-cerebral spaces demonstrated a significant association with neurodevelopmental outcome at 2 years of age [13]. Unfortunately, these parameters are not assessed routinely.

4 Contribution of epidemiological studies

4.1 Data concerning neurodevelopmental outcome of late-preterm infants

There have been numerous epidemiological studies focusing on the neurodevelopmental outcome of LPIs since the mid-2000s. This underscores the medical community's growing interest on this topic in developed countries. However, these studies demonstrate extensive methodological heterogeneity, which disallows conclusions on the real impact of late prematurity on neurological outcome. Methodological variations, for example, encompass: the frequent merging of moderate-preterm infants and LPIs [12,13], neonatal course [9,14–16], experimental group compositions [16,17], judgement criteria [17], and assessment tools [18,19].

4.1.1 Late prematurity and cerebral palsy

Cerebral palsy is a chronic and permanent disorder of movement and posture secondary to motor and cognitive brain impairment. The Gross Motor Function Classification System (GMFCS) is composed of five severity levels that describe global motor function self-initiated by patients [20]. Therefore, cerebral palsy is a well-defined pathology, easily diagnosed, that reflects severe developmental delay.

A population study based on the birth register in Finland from 1991 to 2008 ($n = 1,018,302$ live births) focused on the incidence of cerebral palsy according to birth term. The cerebral palsy incidence was 0.22% in the whole population and 0.1% in infants born after 37^{0/7} weeks of gestation. For LPIs, this incidence was 0.6%, which reflected a significant association (OR = 2.35, 95% CI [1.99–2.77]) [16]. A retrospective study on nearly 150,000 children born after 30 weeks of gestation in the United States reported the same association, with a hazard ratio of 3.39 (95% CI [2.54–4.52]) [21].

4.1.2 Late prematurity and neurodevelopmental outcome at pre-school age (i.e. up to 3 years of age)

Several epidemiological studies emphasized that neurodevelopmental assessment should be performed in reference to corrected age rather than chronologic age up to 2 years for moderate-preterm infants and LPIs [15,22]. A comparison of scores according to the corrected rather than chronologic ages in moderate-preterm infants and LPIs found a decrease in the number of infants identified with neurodevelopmental delay from 18.3% to 15% [18]. However, only few studies focused on LPIs. A Canadian study reported on the results from a parental questionnaire (Ages and Stages Questionnaire [ASQ], third version) for 52 LPIs and 156 term infants at a corrected age of 12 months. Infants were paired on sex (1:3), and small-for-gestational-age infants were excluded. Upon univariate analysis, communication and gross motor scores were significantly lower in LPIs. LPIs also demonstrated lower scores in other assessed domains, but did not reach a threshold of significance. Pathologic communication scores (i.e. < -2 standard deviations [SD]), were observed in 13.5% of LPIs and only in 4.5% of term infants. Concerning gross motor skill, 34.6% of LPIs and 20.5% of term infants exhibited a pathologic score. In logistical regression, however, after adjusting for maternal academic level, delivery method, hospitalization in neonatal unit, and breastfeeding, the statistical association between late prematurity and neurodevelopmental impairment at a

corrected age of 12 months did not persist [23]. Neurodevelopmental assessment was performed in 36-month-old infants through parental questionnaires in a Norwegian cohort (MoBa) that included full-term infants (i.e. those born from 39^{0/7} to 40^{6/7} weeks of gestation; $n = 30,641$), early-term infants (i.e. those born from 37^{0/7} to 38^{6/7} weeks of gestation; $n = 7,109$), and LPIs ($n = 1,673$). Pathologic communication skills were observed in 4.8% of full-term infants, 5.8% of early-term infants, and 6.3% of LPIs. Late prematurity was associated with communication delay (OR = 1.32, 95% CI [1.07–1.62]). However, this statistical effect did not persist after adjusting for emergency caesarean section requirements and neonatal morbidities such as quality of extra-uterine life adaptation, respiratory distress requiring mechanical ventilation, and intracranial bleeding [24]. One study assessed the neurodevelopment of 1200 LPIs in comparison with 6300 term infants at a chronological age of 2 years with the Bayley scale (second version). The mean mental and psychomotor indices were significantly lower in the LPI group (85 vs 89, $p < 0.0001$, and 88 vs 92, $p < 0.0001$, respectively). Furthermore, a mental index lower than 70 was observed in 21.2% of LPIs and 16.4% of term infants ($p = 0.007$) [25].

4.1.3 Late prematurity and neurodevelopmental outcome at school age

A North American study reported on the neurodevelopmental outcome of 7152 LPIs and 152,661 term infants at ages 3 and 5 years. This study focused on singleton pregnancy and infants who did not require more than three hospitalization days during their neonatal period. A significant association was observed between late prematurity and neurodevelopmental impairment (adjusted relative risk = 1.36, 95% CI [1.29–1.43]) [26]. A meta-analysis focusing on the neurodevelopment of LPIs up to 7 years of age was performed based on three observational prospective studies and seven retrospective ones. This meta-analysis remained descriptive owing to the wide heterogeneity of the selected studies. A moderate deficit was

constant in LPIs concerning neurological impairment, school skills, and early intervention programme requirement [17].

Memory, attention, and reading skills as well as an intellectual quotient were tested with a neuropsychometric scale (WISC-III) in an English cohort of moderate-preterm infants and LPIs at the ages of 8–11 years. No difference on intellectual quotient was detected in comparison with term infants (-1.38, 95% CI [-3.20–0.44], $p = 0.14$). However, memory and reading scores were lower in preterm infants. Furthermore, preterm infants exhibited more educational needs (OR = 1.56, 95% CI [1.18–2.07], $p = 0.002$) [27]. Behavioural outcomes of three birth term classes up to 33^{6/7} weeks of gestation ($n = 74$), from 34^{0/6} to 36^{6/7} weeks of gestation ($n = 342$), and equal to or more than 37^{0/7} weeks of gestation ($n = 2788$) were assessed at age 11 years through psychometric and parental scales [28]. LPIs were at greater risk for developing behavioural trouble (OR = 1.6, 95% CI [1.1–2.1]). However, this risk was no longer significant after adjusting for parental socioeconomic level and perinatal factors [28].

4.1.4 Late prematurity and neurodevelopmental outcome at teen and adult ages

Attention and teaching problems were screened in a North American cohort of infants born between 1976 and 1982 either from 34^{0/6} to 36^{6/7} weeks of gestation ($n = 256$) or at more than 37^{0/7} weeks of gestation ($n = 4419$). In total, 75% of the infants were followed up to 19 years of age. No difference was established before or after adjustment [29]. Another study reinforced this result and demonstrated identical cognitive, behavioural, and social skills at 15 years of age in healthy LPIs as in term infants. Healthy infants exhibited non-complicated neonatal courses, including hospitalization for fewer than 7 days, no significant maternal or neonatal morbidity, and an absence of malformation or genetic disease with neurological impact. However, mothers of LPIs were older and exhibited more complicated pregnancies, with arterial hypertension and diabetes. The LPI group ($n = 53$) consisted of 64% of infants

born after 36^{0/7} weeks of gestation and 81% of infants weighing more than 2500 g at birth. The term infant group ($n = 1245$) included only 60% of infants born from 39^{0/7} to 40^{6/7} weeks of gestation [9]. A recent meta-analysis focused on the outcome of infants born from 34^{0/7} to 38^{6/7} weeks of gestation at adult age. A total of 53 studies were selected, including 48 Scandinavian studies, based on registries or large cohorts, which allowed for a descriptive synthesis only. A modest cognitive deficit was observed in adults born late preterm but without reaching the significance threshold (-0.11--0.25 SD). Susceptibility for developing psychiatric disorders was present in several studies without crossing the threshold of significance (OR = 1.11, 95% CI [0.67–1.84]) [30].

Thus, late prematurity did not appear to exert a strong negative impact on school learning, attention, or psychiatric disorders in teenagers or young adults. However, complementary studies are needed underlining the methodological difficulties linked to this issue.

4.2 Neonatal factors associated with neurodevelopmental impairment in late-preterm infants

Epidemiological studies regularly reported significant neurodevelopmental impairment in LPIs in comparison with term infants despite large heterogeneity and low methodological quality. However, perinatal factors associated with late prematurity seemed to influence neurodevelopmental outcome. Data adjustment on perinatal factors or selection of infants without neonatal morbidities thus eliminated the significant effect of late prematurity on neurodevelopmental outcomes [9,19,28]. Determining these factors is an important challenge considering the large population of infants born between 34^{0/7} and 36^{6/7} weeks of gestation and the need to identify the infant sub-group at neurodevelopmental risk who require follow-up.

4.2.1 Birth term

Several studies highlighted that neurodevelopmental skills improved with each week of further gestation. Surprisingly, this effect was also observed in early-term infants (i.e. 37^{0/7}

and 38^{6/7} weeks of gestation) in comparison with full-term infants (i.e. 39^{0/7} to 40^{6/7} weeks of gestation) [24]. These data argued for a neurodevelopmental improvement continuum according to birth term. Shapiro-Mendoza et al. reported that birth term influenced the proportion of infants who benefitted from early intervention programmes in the United States: 34.9%, 24.9%, 19.9%, 16.7%, 14.1%, 12.6%, and 11.7% at 34, 35, 36, 37, 38, 39, and 40 weeks of gestation, respectively [31].

4.2.2 Maternal socio-educational level

The impact of the maternal socio-educational level on children's neurological skills was already demonstrated in very preterm infants [32]. This factor was therefore an adjustment variable in several studies that reported neurodevelopmental outcomes of LPIs [9,23,29,33]. Both studies aimed to assess the effect of maternal academic level on infant neurodevelopmental outcome. Morag et al. reported a negative effect of low maternal academic level on all infants born from 34^{0/6} to 41^{6/7} weeks of gestation (OR = 2.7, 95% CI [1.18–6.3]). However, this effect was more evident in LPIs (OR = 3.98, 95% CI [1.51–10.5]) [15]. In parallel, Coletti et al. reported a communication index increase at the age of 12 months in correlation with a rise in maternal educational level [34]. One study focused on the impact of low socio-economic level on neurodevelopmental delay in LPIs and full-term infants. A significant negative effect on neurodevelopmental outcome was found out in all-term infants of maternal low socio-economic status. However, lower gestational age associated with low socio-economic status had a synergic effect, worsening neurodevelopmental outcome [35].

4.2.3 Small for gestational age (birth weight < 10th percentile)

Hirvonen et al. demonstrated that LPIs who were small for their gestational age were at higher risk for developing cerebral palsy (OR = 1.85, 95% CI [1.25–2.75]) [16]. Furthermore, small-for-gestational-age infants who were moderate-preterm and late-preterm demonstrated a

significant neurodevelopmental delay at the age of 4 years, measured through a parental questionnaire (ASQ) [14]. By contrast, another two studies showed that this association was not consistent in the LPI group even when small for gestational age was a risk factor of neurodevelopmental impairment in all-term cohorts [15,36].

4.2.4 Delivery type: caesarean section

As with maternal educational level, caesarean section was a typical adjustment variable in the literature [9,23,24]. Gross motor function delay was significantly more frequent at the age of 4 years in infants delivered by caesarean section [19]. Morag et al. reported a significant association between delivery by emergency caesarean section and neurodevelopmental delay at the age of 12 months (OR = 4.72, 95% CI [1.88–11.8]) [15].

4.2.5 Neonatal course

Morbidities inducing hospitalization in a neonatal care unit appeared as an important factor conditioning the neurodevelopmental outcome of LPIs. Neurodevelopment at the age of 4 years was assessed through ASQ according to easy ($n = 116$) or complex ($n = 47$) neonatal course. A complex neonatal course was defined by respiratory distress, haemodynamic instability, phototherapy requirement, or hypoglycaemia (i.e. less than 40 mg/dL) in the first 48 h of life. Infants with a complex neonatal course exhibited lower gestational age and birth weight. Antenatal corticosteroids were administered more frequently in infants with neonatal complications. A significant increase of neurodevelopmental delay was observed in infants with a complex neonatal course. Surprisingly, LPIs with an easy neonatal course exhibited as identical neurological performance to full-term infants [19]. In a moderate- and late-preterm cohort, neonatal hypoglycaemia (OR = 2.42, 95% CI [1.23–4.77]) and Apgar scores of less than 7 at 5 min (OR = 3.18, 95% CI [1.01–10]) were associated with a global ASQ score more than -2 SD in univariate analysis. Hyperbilirubinaemia and admission to a neonatal intensive care unit were just below the significance threshold. In multivariate analysis, after

adjustment for sex and small for gestational age, neonatal hypoglycaemia only remained associated with significant neurodevelopmental impairment (OR = 2.19, 95% CI [1.08–4.40]) [14]. From Finnish birth registers, cerebral palsy was associated with: birth resuscitation (OR = 1.78, 95% CI [1.09–2.9]), neonatal antibiotics treatment (OR = 1.67, 95% CI [1.13–2.44]), an Apgar score of less than 7 in the first minute (OR = 1.8, 95% CI [1.21–2.67]), and intracranial bleeding (OR = 12.8, 95% CI [5.58–29.2]) in moderate-preterm infants and LPIs [16]. Therefore, the neonatal course may be a crucial factor in the early detection of LPIs with a worse neurodevelopmental prognosis who require specific follow-up.

5 Discussion

The neurodevelopmental outcomes of LPIs have generated abundant medical literature over the past decade. This literature review gives rise to several levels of reflection.

LPIs experience physiological brain immaturity associated with more complex pregnancies, and more neonatal morbidities (e.g. small for gestational age, perinatal asphyxia, hypoglycaemia, and hyperbilirubinaemia) compared with term infants [37]. This suggests neurodevelopmental vulnerability as observed in younger preterm infants.

Emerging data highlighted that each week up to 39 weeks of gestation contributed to a reduction in neonatal morbidities and to neurodevelopment improvement. Therefore, birth term may be a crucial factor for predicting neurological outcome to a greater extent than maturation sub-groups.

Although there are many published data on this topic, a lack of homogeneous and rigorous methodology concerning birth term, judgement criteria, and assessment tools renders any conclusions inconclusive. However, contradictory results or near-neutral ORs suggest a moderate neurodevelopmental risk in LPIs.

Systematic neurodevelopmental follow-up of LPIs may be considered based on the reviewed data. However, LPIs account for nearly 70% of preterm births. This new patient flow may

represent a sudden burden for follow-up networks, and could be potentially detrimental for patients with identified neurological impairment. Long-term systematic follow-up for LPIs is therefore unrealistic. Patient selection may be a safe strategy for detecting infants at risk of neurological impairment. Selection criteria may be based on perinatal factors, the neonatal period, or validated neurologic examinations at term-equivalent age [38]. Alternatively, selection may be performed later through a systematic parental questionnaire at key neurodevelopmental ages or at school entry.

Key leaders have not agreed on a clear follow-up strategy for LPIs. Nevertheless, the Spanish Society of Neonatology recently published recommendations for LPI follow-ups, which highlights an emerging desire to solve this issue [39].

6. Conclusion

Overall, the neurodevelopmental impacts of LPIs currently remain elusive. The low methodological quality of epidemiological studies and the heterogeneity of the neonatal course interfere with the insightful determinations of neurological prognoses. Therefore, the current challenge is to determine efficient screening strategies for selecting those requiring neurological follow-up.

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