Neurological outcome following isolated 10 12 mm fetal ventriculomegaly

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LETTERS

Neurological outcome following isolated 10–12 mm fetal ventriculomegaly

The outcome of non-progressive, isolated fetal ventriculomegaly is uncertain. The normal width of the atrium of the lateral ventricle is less than or equal to 9 mm.[1] Severe ventriculomegaly (or hydrocephalus) is defined as widths greater than 15 mm. Mild values of 10–15 mm confer an increased risk for aneuploidy, malformations and impaired postnatal neurological outcome, although the frequency of the association is variable.[2–4] However, the outcome of fetal non-progressive isolated forms of lateral ventriculomegaly with atrial widths between 10 and 12 mm has been little studied. It is, therefore, difficult to provide appropriate counselling in such cases. A recent series[5] suggested that outcomes in patients with non-progressive isolated mild ventriculomegaly with atrial widths between 10 and 12 mm were essentially the same as in controls.

A later recent series[6] suggested that the presence of mild ventriculomegaly does not necessarily correlate with good outcome, irrespective of the degree of the ventricular dilatation and irrespective of antenatal or neonatal resolution.

We evaluated outcome in 20 patients identified by retrospective review to have non-progressive isolated mild ventriculomegaly (atrial widths of 10–12 mm) on prenatal examination.

Between January 1998 and December 2005, 7056 infants underwent obstetric ultrasonographic first scans using a standard protocol at the Obstetric and Gynecology Unit at the University of Chieti (Italy). Twenty fetuses diagnosed with non-progressive isolated 10–12 mm lateral ventriculomegaly who underwent postnatal follow-up and had complete data available, entered the study. Figure 1 shows the selection procedure. Prenatal ultrasound examinations of the brain, including atrial width measurements of both the left and right lateral ventricles, and postnatal physical and neurological examinations (including complete electroencephalography) were performed. Briefly, we followed the same protocol as Vergani *et al*[2] with the addition of proximal and distal hemispheric evaluations and, in selected cases (suspected of malformations), fetal total body magnetic resonance imaging. In addition, we assessed intellectual and motor development using the Griffith Mental Development Scale scores for locomotor, personal-social, hearing and language, eye and hand co-ordination, and overall performance.

Postnatal follow-up times ranged from 13 months to 95 months (mean 42.7 (SD 25.8) months). Clinical characteristics are summarised in table 1. The condition was not detected at follow-up in eight of 20 fetuses who had non-progressive and isolated 10–12 mm lateral ventriculomegaly. Four cases (20%) presented with a mildly impaired neurological outcome; in comparison, the rate in the general population is 2.5%.[7] Two of these patients recovered after participating in a rehabilitation program specific for the observed neurological impairment, and two are still in rehabilitation.

The message from this study is clear: the majority of the cases of fetal non-progressive and isolated lateral ventriculomegaly with an atrial width of 10–12 mm have a good outcome, although there is evidence of neurological impairment in early childhood in some cases.

The number of cases reported with persistent abnormal development is 2/20. Whether this is "large" is debatable, but it is significant and comparable to that reported by Vergani[2] and in other studies. As the rate of neurological impairment (although moderate) is higher than that in the general population, postnatal follow-up is strongly recommended.

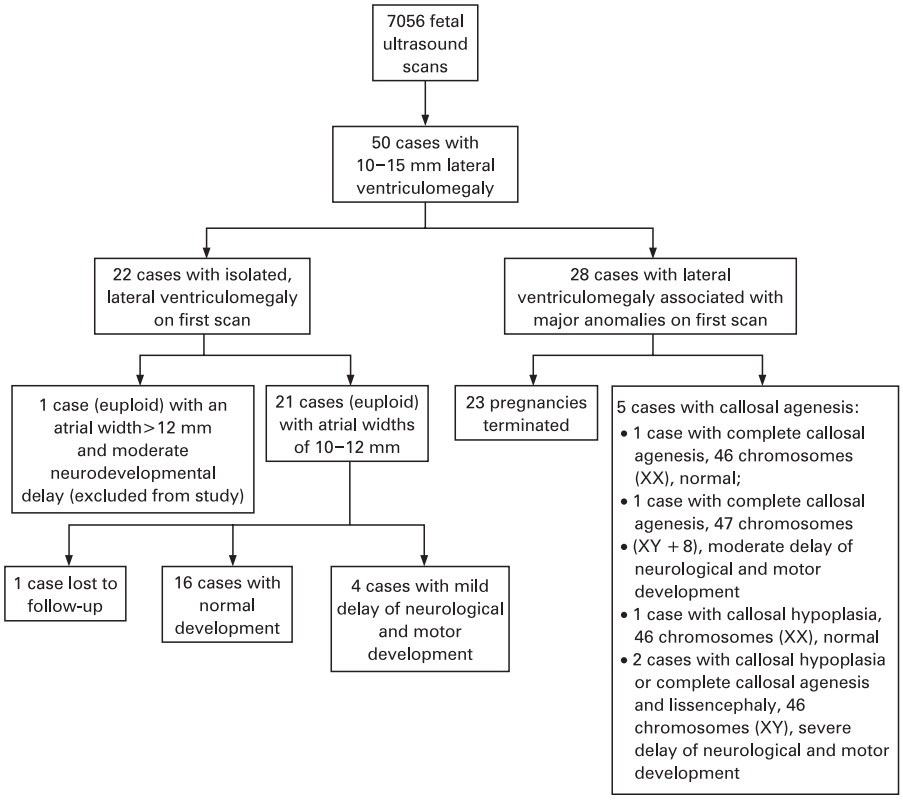

Figure 1 Flow chart of selection process. 7056 fetal were ultrasounds performed at our centre from 1998 to 2005 and 20 fetuses with non-progressive isolated mild ventriculomegaly (atrial widths of 10–12 mm) were identified.

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Table 1 Clinical characteristics of the 20 fetuses diagnosed with non-progressive isolated 10–12 mm lateral ventriculomegaly who were followed up postnatally

	All subjects, n = 20	Subjects with impaired neurological outcome, n = 4	Subjects with impaired neurological outcome who later recovered, n = 2		Subjects with impaired neurological outcome, n = 2	
			Fetus 1	Fetus 2	Fetus 3	Fetus 4
Sex, male/female	14/6	1/3	F	F	M	F
Gestational age at diagnosis, weeks	24.5 (SD 3.8), 18–32	25.5 (SD 4.2), 21–30	23	28	21	30
Type of lateral ventriculomegaly, ML/BL	11/9	2/2	ML	BL	BL	ML
Lateral ventriculomegaly at birth	8, resolved (6 ML, 2 BL) 12, persistent (6 ML, 2 BL)	2, resolved (1 ML, 1 BL) 2, persistent (1 ML, 1 BL)	Persistent	Persistent	Resolved	Resolved
Right lateral atrium width, mm	10.0 (SD 1.2), 6.6–12.0	10.4 (SD 0.5), 10.0–11.0	11.0	10.8	10.0	10.0
Left lateral atrium width, mm	9.5 (SD 1.6), 7.0–12.0	8.8 (SD 2.1), 7.0–11.2	7.0	11.2	10.0	7.0
Difference between lateral atrial widths, mm	1.63 (SD 1.56), 0–3.7	1.85 (SD 1.95), 0–4.0	4.0	0.4	0	3.0
Gestational age at birth, weeks	37.5 (SD 2.3), 34–42	37.5 (SD 0.6), 37–38	38	38	37	37
Weight at birth, kg	3.1 (SD 0.65), 2.0–4.65	3.0 (SD 0.5), 2.2–3.4	3.0	2.2	3.4	3.3
Apgar score at 1 min	7.9 (SD 1.02), 7–9	8.2 (SD 0.95), 7–9	8	7	9	9
Apgar score at 5 min	8.9 (SD 0.82), 7–10	9 (SD 0.81), 8–10	9	8	10	9
Age at first Griffith test, months	26.15 (SD 24.25), 3–75	7 (SD 4.9), 3–13	13	9	3	3
Age at last Griffith test, months	42.7 (SD 25.82), 13–95	22.5 (SD 8.8), 15–32	32	28	15	15

Values are mean (SD), range. BL, bilateral; ML, monolateral.

Surfactant replacement after acute massive milk aspiration in a very low birthweight infant

Massive milk aspiration during infant feeding is a severe event causing respiratory distress, asphyxia and sudden death.[1] To date, only experimental models have shown that administration of exogenous surfactant is a successful treatment for acute lung injury induced by acidified formula, or human breast milk, aspiration.[2 3] We describe a case of massive milk aspiration successfully treated with surfactant administration in a very low birthweight infant.

CASE REPORT

A 1180 g male infant of 30 weeks' gestation was delivered by caesarean section due to variable decelerations in cardio-tocographic readings. The infant's Apgar scores were 8 and 9 at 1 and 5 min, respectively. An umbilical venous catheter was placed, parenteral nutrition started and empiric antibiotic therapy (ampicillin plus gentamicin) instituted. The infant did not need respiratory support or supplemental oxygen after delivery and started enteral feeding on day 1.

On day 11, the infant vomited while being fed breast milk by gavage. The pharynx and the stomach were drained at once and immediately afterwards a bronchoaspiration was performed resulting in milk outflow. A chest radiograph showed massive milk inhalation. The infant required intubation and assisted ventilation because of severe respiratory distress. By 36 h after milk inhalation, the fraction of inspired oxygen (FiO_2) had gradually increased to 0.95 and the alveolar-arterial oxygen difference ($AaDO_2$) was ~565 mm Hg.

Following administration of one dose of natural porcine lung surfactant (~200 mg/kg), FiO_2 decreased to 0.50 ($AaDO_2$ ~245 mm Hg) and peak inspiratory pressure (PIP) was reduced from 20 to 16 cm H_2O. By 8 h after administration of surfactant, FiO_2 had declined to 0.40 and it was possible to switch from assisted to synchronised intermittent mandatory ventilation (rate 30/min). A second dose of surfactant (~100 mg/kg) was given 12 h after the first; the PIP was further reduced to 14 cm H_2O and the oxygen requirement decreased to 0.35 ($AaDO_2$ ~140 mm Hg). Owing to the persistent oxygen demand, a third dose (~100 mg/kg) was delivered and a slight decrease in FiO_2 (to 0.30) and $AaDO_2$ (to ~100 mm Hg) was registered.

By 48 h after the beginning of surfactant therapy, FiO_2 was 0.23 and the infant's clinical condition was continuing to improve. The infant was extubated and treated for a further 8 h with nasal continuous positive airway pressure (FiO_2 0.30). After 2 more days of supplemental oxygen, O_2 therapy was discontinued because blood oxygen saturation in room air (~93%) was satisfactory. The infant was discharged home on day 50. He is now 2 years old and shows normal growth and neurological development at ongoing follow-up examinations.

In our single experience, natural porcine lung surfactant proved to be very effective in reducing $AaDO_2$ and improving pulmonary compliance. We performed "late" surfactant replacement therapy because of our uncertainty about its possible effectiveness; however, "early" replacement therapy or a bronchoalveolar lavage with diluted surfactant, as in meconium aspiration syndrome,[4] may be a possible alternative.

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CORRECTION

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In the letter "Benchmarching neonatal anthropometric charts published in the last decade" in *Arch Dis Child Fetal Neonatal Ed* 2008:**94**:F233, the author's name is M De Curtis and not DeCurtis as published.