Ultrasound Obstet Gynecol 1999;14:320–326

The clinical significance of fetal isolated cerebral borderline ventriculomegaly: report of 31 cases and review of the literature

G. Pilu, P. Falco, S. Gabrielli, A. Perolo, F. Sandri and L. Bovicelli*

Department of Obstetrics and Gynecology, *Department of Neonatology, University of Bologna, Italy

Key words: VENTRICULOMEGALY, HYDROCEPHALUS, FETUS, ULTRASOUND, PRENATAL DIAGNOSIS

ABSTRACT

Objective *To assess the clinical significance of fetal isolated borderline cerebral lateral ventriculomegaly defined as a width of the atrium of the lateral cerebral ventricles of 10–15 mm in the absence of other sonographically demonstrable malformations.*

Design *Retrospective study of the outcome of fetuses with a sonographic diagnosis of isolated borderline cerebral lateral ventriculomegaly and review of the English-language literature.*

Results *Of 31 fetuses, two had chromosomal aberrations (trisomy 21 and trisomy 13) and three had neurological complications (one infant developed shunt-dependent hydrocephalus, one lissencephaly and one cerebral hemorrhage and periventricular leukomalacia). The literature search revealed eight independent studies. Including the present series, 234 cases were available for analysis. An abnormal outcome was documented in 22.8% of cases. Perinatal death occurred in 3.7%. Chromosomal aberrations, mostly trisomy 21, were present in 3.8%, malformations undetected at a second-trimester sonogram in 8.6% and neurological sequelae, mostly a mild to moderate delay in cognitive and/or motor development, were present in 11.5%. The risk of an abnormal neurological outcome was increased in females versus males (22.6% versus 4.6%, relative risk 4.892; 95% confidence interval 1.356–17.656), when the atrial width was 12 mm or more (13.9% versus 3.8%, relative risk 3.6, 95% confidence interval 1.035–12.846) and when the diagnosis was made in the second trimester versus later in gestation.*

Conclusions *In most cases, isolated borderline cerebral lateral ventriculomegaly has no consequence. However, this finding carries an increased risk of cerebral maldevelopment, delayed neurological development and, possibly, chromosomal aberrations. The optimal management of these cases remains uncertain.*

INTRODUCTION

Evaluation of the cerebral lateral ventricles is an essential part of the standard sonographic examination of the fetus[1]. Ventricles of normal size provide reassurance of the normal development of the cerebrum. Gross enlargement consistently indicates major cerebral anomalies. Borderline enlargement of the ventricles, a condition that is usually defined as an atrial width of 10–15 mm between 15 and 40 weeks' gestation, is frequently associated with neural and extraneural malformations and is a widely accepted indication for a targeted sonogram[2,3]. Over the past decade, however, several studies have pointed out that, even in the presence of an otherwise normal sonogram, borderline cerebral ventricles carry an increased risk of an abnormal outcome[2–8].

We report our experience with 31 consecutive cases of fetal borderline cerebral ventricles in the absence of other anatomic abnormalities demonstrable by antenatal sonography (a condition that will be referred to throughout this paper as isolated borderline cerebral ventriculomegaly) and review the English-language literature on this topic.

MATERIALS AND METHODS

In our ultrasound laboratory, the atrial width is measured in all fetuses examined in the second and third trimesters. The measurement is obtained in a transverse plane of the fetal head, from the atrium distal to the ultrasound transducer (Figure 1). Calipers are positioned at the level of the glomus of the choroid plexus, inside the echoes generated by the walls of the ventricles, and the obtained values are approximated to the closest millimeter[9]. The archives of the laboratory were searched between 1990 and 1995 for cases of isolated cerebral borderline ventriculomegaly, defined as an atrial width of 10–15 mm (Figure 2) with no demonstrable associated malformations. In all cases, fetal karyotyping, a TORCH screen and serial sonograms were

Correspondence: Dr G. Pilu, Clinica Ginecologica e Ostetrica, Policlinico S. Orsola, Via Massarenti 13, 40138 Bologna, Italy

Received 22–2–99
Revised 2–6–99
Accepted 23–6–99

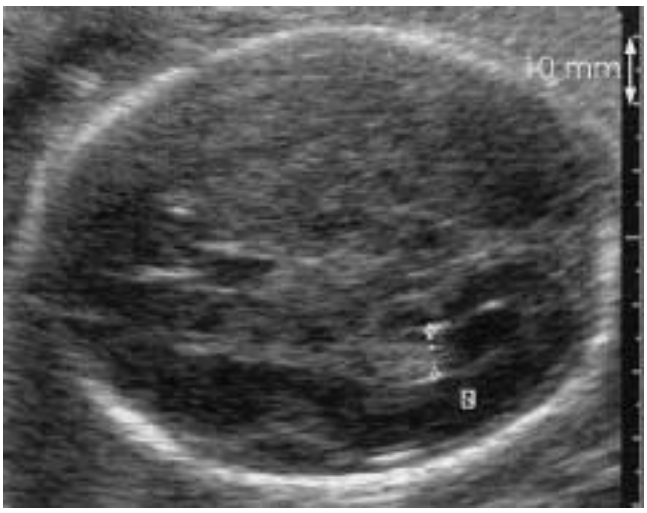

Figure 1 Measurement of the atrial width in a midtrimester fetus with normal lateral ventricles. Calipers are positioned at the level of the glomus of the choroid plexus inside the ventricular walls, and perpendicular to the ventricle

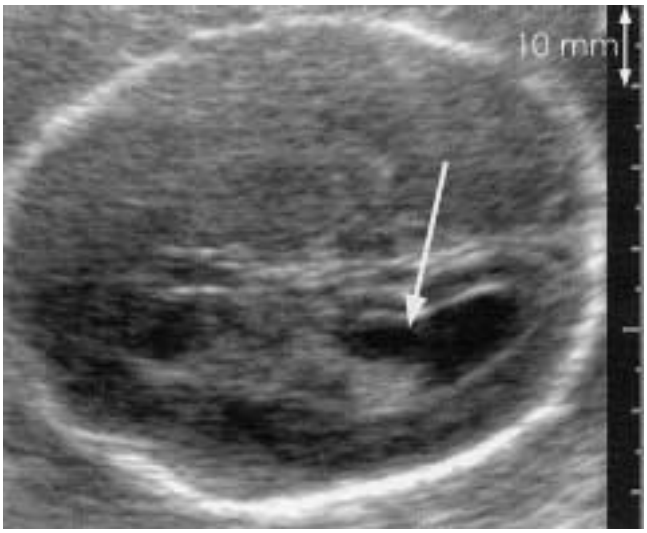

Figure 2 Borderline cerebral ventriculomegaly. The atrial width (arrow) is 10 mm. There is shrinking and anterior displacement of the glomus of the choroid plexus

offered to the couples. Postnatal follow-up was obtained by interviewing the couples and the pediatricians of the infants.

A search was conducted for reports of the condition in the English-language literature (Medline computer search, National Library of Medicine) by using ventriculomegaly and hydrocephalus as key words. A hand search was also made of peer-review journals in the field of diagnostic imaging, obstetrics and prenatal diagnosis.

Fisher's exact *t* test was used for statistical analysis. Relative risks were calculated by using the approximation of Katz.

RESULTS

Personal experience

Between 1991 and 1995, 31 fetuses with isolated cerebral borderline ventriculomegaly were seen in our institution. All cases were referred from other ultrasound departments with a suspicion of cerebral ventriculomegaly. The relevant clinical data are reported in Table 1. Gestational age ranged between 20 and 35 weeks (mean and median 24). Twenty-two fetuses (71%) were males. Two chromosomal aberrations were found, one trisomy 21 and one trisomy 13. These women were 39 and 29 years old, respectively. In both cases, other sonographic markers of chromosomal aberrations were present. Both pregnancies were terminated upon request of the couples. Four cases were lost to follow-up. In one fetus that also had a borderline value of the cisterna magna (10 mm) and an increased nuchal fold (7 mm) at 22 weeks, serial sonograms revealed progression of ventricular enlargement. The cisterna magna also increased in size, reaching a diameter of 34 mm at 33 weeks' gestation. At this time, asymmetry between the cerebellar hemispheres and a communication between the fourth ventricle and the cisterna magna were noted. The presence of Dandy–Walker variant was confirmed after birth, and a factor X coagulation defect was also found. The infant developed hypertensive hydrocephalus and was treated with a ventriculoperitoneal shunt. The neurological development, however, was normal at the age of 3 years. One fetus originally seen at 24 weeks' gestation developed severe enlargement of the entire ventricular system and was diagnosed as having lissencephaly after birth. The infant has profound intellectual and motor retardation. In one case, originally seen at 35 weeks' gestation, intraventricular hemorrhage and periventricular leukomalacia were documented following an unremarkable vaginal delivery at term. The infant has a mild delay in cognitive and motor development at the age of 2 years. One infant developed postnatal growth retardation and was found to have congenital leishmaniasis, but is otherwise doing well at the age of 2 years. Overall, postnatal information was available for 25 infants. Of these, seven had a birth weight greater than the 90th centile (28%). On follow-up at 21–72 months (mean 33, median 30 months), all infants were found to be developing normally, with the exception of the cases with associated lissencephaly and periventricular leukomalacia.

Review of the literature

Nine reports describing the outcome of fetuses with isolated cerebral borderline ventriculomegaly were found[2–8,10,11]. One report was not considered in the analysis, as the results were included in a subsequent article[2]. By adding our series, the overall number of cases was 234 (Table 2). Chromosomal aberrations were found in nine cases (3.8%). Trisomy 21 was most frequently encountered. In most cases, advanced maternal age and/or other sonographic findings of chromosomal aberrations were present (Table 3). In 19 of 221 cases (8.6%), structural anomalies that had not been detected at the time of the first

Table 1 Clinical data of 31 cases of isolated cerebral lateral borderline ventriculomegaly

Case	*Gestational age at diagnosis* (weeks)	*Atrium* (mm)	*Additional prenatal findings*	*Karyotype*	*Outcome*	*Follow-up* (months)	*Intellectual and motor development*
1	24	12	—	46,XY	lost to follow-up	—	—
2	23	11	—	46,XY	lost to follow-up	—	—
3	22	10	—	46,XX	lost to follow-up	—	—
4	22	11	—	46,XY	lost to follow-up	—	—
5	20	10	hyperechogenic bowel	47,XX + 13	termination of pregnancy	—	—
6	23	11	nuchal thickness 10 mm	47,XY + 21	termination of pregnancy	—	—
7	22	10	cisterna magna 10 mm nuchal thickness 7 mm	46,XY	3420 g at 39 weeks (AGA); Dandy–Walker variant; ventriculo-peritoneal shunt; coagulation factor X deficiency	38	normal
8	24	10	polyhydramnios	46,XX	2280 g at 41 weeks (SGA); lissencephaly	24	severe delay
9	35	10	—	46,XX	2800 g at 39 weeks (SGA); IVH with periventricular leukomalacia	24	mild delay
10	24	12	—	46,XX	3450 g at 41 weeks (AGA); congenital leishmaniasis; postnatal growth retardation	25	normal
11	29	12	—	46,XY	2850 g at 39 weeks (SGA)	49	normal
12	24	11	—	46,XY	3560 g at 40 weeks (AGA)	36	normal
13	24	11	—	46,XX	2780 g at birth, 38 weeks (AGA)	29	normal
14	24	11	—	46,XY	3250 g at birth, 38 weeks (AGA)	36	normal
15	24	10	—	46,XY	4450 g at 41 weeks (LGA)	36	normal
16	26	14	—	46,XY	2950 g at 36 weeks (AGA)	26	normal
17	22	10	—	46,XY	4000 g at 41 weeks (LGA)	24	normal
18	25	11	—	46,XX	3850 g at 42 weeks (LGA)	23	normal
19	23	10	—	46,XY	3600 g at 40 weeks (AGA)	23	normal
20	24	12	—	46,XY	4220 g at 40 weeks (LGA)	22	normal
21	23	10	—	46,XY	3650 g at 37 weeks (LGA)	21	normal
22	33	11	—	46,XY	3750 g at 40 weeks (AGA)	30	normal
23	24	10	—	46,XX	3650 g at 39 weeks (AGA)	43	normal
24	21	11	—	46,XY	3060 g at 39 weeks (AGA)	40	normal
25	22	10	—	46,XY	2950 g at 37 weeks (AGA)	36	normal
26	23	11	—	46,XY	3130 g at 36 weeks (AGA)	25	normal
27	22	11	—	46,XY	3200 g at 39 weeks (AGA)	48	normal
28	21	12	—	46,XY	3750 g at 39 weeks (AGA)	42	normal
29	30	12	polyhydramnios	46,XY	5000 g at 42 weeks (LGA)	26	normal
30	24	12	polyhydramnios	46,XY	4560 g at 41 weeks (LGA)	30	normal
31	23	11	SGA	46,XX	2400 g at 36 weeks (SGA)	72	normal

AGA, appropriate for gestational age; SGA, small for gestational age; LGA, large for gestational age; IVH, intraventricular hemorrhage

Table 2 Review of series of fetuses with isolated cerebral borderline ventriculomegaly

	Chromosomal anomalies		*Structural anomalies (normal karyotype)*		*Perinatal deaths (normal karyotype)*		*Abnormal neurodevelopment (normal karyotype)*		*Total cases with abnormal outcome**	
Authors	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%
Mahony *et al.*[4]	1/15	6.7	3/14	21.4	4/14	28.6	1/10	10	7/15	46.6
Bromley *et al.*[5]	0/27	0	1/27	3.7	0/26	0	5/26	19.2	5/26	19.2
Achiron *et al.*[7]	2/7	28.6	2/5	40	0/3	0	0/3	0	4/7	57.1
Patel *et al.*[6]	1/37	2.7	6/36	16.7	2/36	5.6	6/34	17.6	12/37	32.4
Alagappan *et al.*[10]	0/11	0	2/11	18.2	0/11	0	0/11	0	2/11	18.2
Bloom *et al.*[8]	0/30	0	1/30	3.3	1/30	3.3	9/29	31	10/30	33.3
Vergani *et al.*[13]	2/48	4.2	1/46	2.2	1/46	2.2	0/45	0	4/48	8.3
Lipitz *et al.*[11]	1/28	3.6	0/27	0	0/26	0	1/26	3.8	2/27	7.4
Present experience	2/31	6.4	3/25	12	0/25	0	2/25	8	6/27	22.2
Total	9/234	3.8	19/221	8.6	8/217	3.7	24/209	11.5	52/228	22.8

*Excludes cases lost to follow-up and termination of pregnancies of fetuses without associated anomalies

scan were found (Table 4). Major cerebral anomalies, including progressive hydrocephalus requiring shunt implantation after birth, cystic brain lesions and lissencephaly, were found in nine cases (4.1%). Heterogeneous extraneural anomalies of lesser clinical significance were identified in the remaining ten cases (4.5%). Perinatal or early neonatal death occurred in eight of 217 cases (3.7%), almost exclusively in association with severe growth restriction, premature delivery and/or perinatal infections (Table 5). Different modalities of neurological follow-up were employed. In two reports, a formal assessment of cognitive and motor development following an established evaluation scale was employed[3,8]. In three reports[4,5,6] as well as in the present series, information on the development of the infant was obtained by interviews with the family and the pediatricians. In three reports, the type of follow-up was not specified[7,10,11]. The duration of follow-up varied considerably, between 6 and 72 months. It was impossible to derive specific figures from several of the papers, but the majority of infants were followed up for more than 1 year, and a large number for more than 2 years. Very rare cases were presumably controlled after the 3rd year of life. Subnormal neurodevelopment was present in 24 of 209 cases with a normal karyotype (11.5%), with significant variations among the different series. In most cases, an idiopathic mild-to-moderate delay in cognitive and/or motor development was found (Table 6). Overall, an abnormal outcome (chromosomal aberrations, malformations, perinatal death, abnormal neurologic development) occurred in 22.8% of cases (Table 2).

Excluding cases with chromosomal aberrations, information on the fetal sex was available for 96 cases. Males were predominant (68%). Females were more frequently found to have abnormal neurodevelopment than males (7/31 (22.6%) versus 3/65 (4.6%); $p = 0.0118$; relative risk 4.892; 95% confidence interval 1.356–17.656). Information on the size of the ventricles was available in 141 cases. Abnormal neurodevelopment was more frequent when the atrial width was 12 mm or more (5/36 (13.9%) versus 4/105 (3.8%); $p = 0.0474$; relative risk 3.646; 95% confidence interval 1.035–12.846)

DISCUSSION

The cerebral lateral ventricles have a complex three-dimensional architecture that undergoes major developmental changes throughout gestation. Therefore, it is not surprising that sonographic assessment of these structures has been the object of many studies, and that many different approaches to the definition and diagnosis of fetal ventriculomegaly have been suggested from time to time. Reference charts have been established for all the various

Table 3 Chromosomal aberrations in fetuses with isolated cerebral borderline ventriculomegaly

Authors	*Aneuploidy*	*Maternal age* (years)	*Additional sonographic findings*
Mahony *et al.*[4]	trisomy 21	not reported	none
Achiron *et al.*[7]	trisomy 21	25	none
	trisomy 18	25	SGA, fisted hands on a follow-up scan at 23 weeks
Patel *et al.*[6]	trisomy 21	not reported	none
Vergani *et al.*[3]	trisomy 21	38	none
	trisomy 21	40	none
Lipitz *et al.*[11]	trisomy 21	not reported	none
Personal experience	trisomy 13	29	hyperechogenic bowel, nuchal fold
	trisomy 21	39	

SGA, small for gestational age

Table 4 Structural anomalies missed at the first scan in fetuses with isolated borderline ventriculomegaly and normal karyotype

Anomaly	*Cases*
Central nervous system	9
Hydrocephalus requiring surgery	3
Intraventricular hemorrhage with periventricular leukomalacia	2
Cystic brain lesions	1
Lissencephaly	1
Congenital glaucoma	1
Agenesis of the corpus callosum	1
Extracerebral	10
Club foot	2
Hypospadias	2
Distal mild sacral agenesis	1
Mild coarctation of the aorta	1
Patent foramen ovale	1
Small patent ductus arteriosus	1
Bilateral joint contractures	1
Ambiguous genitalia	1
Total	19

Table 5 Clinical data of infants with isolated cerebral borderline ventriculomegaly who died in the perinatal or early neonatal period

Case	*Gestational age at diagnosis* (weeks)	*Additional sonographic findings*	*Perinatal clinical data*
1	16	SGA, oligohydramnios, thick placenta	*in utero* demise at 25 weeks (260 g)
2	22	SGA	delivery at 36 weeks (1830 g); idiopathic failure to thrive
3	24	oligohydramnios	chorioamnionitis; *in utero* demise at 24 weeks (590 g)
4	31	twin pregnancy, SGA	delivery at 35 weeks (1455 g)
5	28	not reported	ornithine transcarbamylase deficiency
6	25	not reported	severe prematurity; cystic brain lesions
7	not reported	not reported	neonatal death from acquired immunodeficiency syndrome
8	not reported	not reported	unexplained intrauterine demise close to term

SGA, small for gestational age

Table 6 Developmental problems and neurological symptoms in infants with a prenatal diagnosis of isolated cerebral borderline ventriculomegaly and a normal karyotype

Developmental problems and neurological anomalies	*Cases*
Developmental delay	20
Severe	3
Mild/moderate	15
Unspecified	2
Others	4
Congenital seizures	2
Congenital glaucoma	1
Hearing impairment	1
Total	24

areas of the lateral ventricles, measured in standard axial planes[12–14], coronal and sagittal sections[15]. However, measurement of the transverse diameter of the ventricular atrium, at the level of the glomus of the choroid plexus, is currently favored[16,17]. This measurement is easily obtained and is reproducible[9]. Different studies have yielded very similar results in the midtrimester, reporting a mean value of the atrial width of approximately 7 mm, and a standard deviation of approximately 1 mm[7,10,13,17]. There is less agreement on the normal values in the third trimester. In two studies, the atrial width was found to remain constant between 15 weeks' gestation and term. However, these studies included relatively small numbers of third-trimester fetuses. Two larger reports were at variance with these results. One study of 503 fetuses demonstrated similar mean values in the second and third trimesters, but revealed an increase in the standard deviation throughout gestation. The mean + 2 standard deviations increased from 9.2 at 16 weeks' gestation to 10.2 at term[10]. Another series of 838 cases described a slight increase throughout gestation, with mean values of approximately 6 and 8 mm at 14 and 40 weeks, respectively, and a 95% confidence interval that exceeded 10 mm from 34 weeks to term[18]. Some degree of asymmetry of the lateral ventricles exists in the human fetal brain[19] and is detectable *in utero*[20]. Lateral ventricle measurements are slightly, but significantly, larger in male than in female fetuses[21,22].

Clinical series suggest that an atrial measurement of < 10 mm is indicative of normalcy, whereas a significant deviation from this value (> 15 mm) is indicative of major dilatation[16]. Borderline ventriculomegaly is usually defined as an atrial width of 10–15 mm between 15 and 40 weeks' gestation[2,3,7,8]. The prevalence of this finding is uncertain. Two prospective series on low-risk patients had conflicting results, ranging from 1 : 50 to 1 : 1600[7,10]. The reason for such a discrepancy is not clear. It may represent the consequence of a different technique for obtaining the measurement. Another possible explanation could be a discrepancy in the gestational age of the fetuses examined. The lower prevalence was obtained from a series of midtrimester fetuses[7]. The higher prevalence was derived from a group including a large number of third-trimester fetuses[10]. Ventricles exceeding 10 mm have been reported in 2–5% of late third-trimester fetuses[10,18]. Furthermore, one study of fetuses with ventricles of 10–12 mm detected at 25–37 weeks reported a normal outcome in all cases[10]. Conversely, the other seven studies together with our experience, which included different proportions of midtrimester fetuses, are consistent, and suggest that isolated cerebral borderline ventriculomegaly carries an increased risk of an abnormal fetal outcome.

Chromosomal aberrations, mostly trisomy 21, were found in 4% of fetuses. Many women were either of advanced maternal age or were carrying fetuses that demonstrated other sonographic markers of aneuploidy. It is therefore uncertain whether isolated cerebral borderline ventriculomegaly is an independent risk factor for chromosomal abnormalities. Further investigation is certainly warranted. It appears to be unlikely, however, that detection of isolated cerebral borderline ventriculomegaly will have an impact on screening for trisomy 21, as enlarged cerebral ventricles are infrequently seen in fetuses with Down's syndrome, particularly in the second trimester[23,24].

Several infants with isolated cerebral borderline ventriculomegaly were found to have malformations that had not been detected at the initial ultrasound examination. Major cerebral anomalies, including hypertensive hydrocephalus and cystic brain lesions, were present at birth in 4% of cases.

Independently from the presence of aneuploidies or underlying malformations, most of the available studies reported an excess of neurological symptoms, particularly abnormal intellectual and motor development. The delay was predominantly mild or moderate, and was severe only in a handful of cases.

In almost 80% of cases thus far reported, isolated cerebral borderline ventriculomegaly has had no clinical sequelae. At least in our experience, there was an excess of large-for-gestational-age infants, and in these cases the cerebral ventricles are probably proportional to the overall body size. However, we speculate that, in a distinct minority of cases, isolated cerebral borderline ventriculomegaly is the earliest manifestation of brain damage from heterogeneous causes including primary cerebral maldevelopment (e.g. obstructive hydrocephalus, lissencephaly) and destructive lesions (e.g. periventricular leukomalacia) arising from hypoxia and/or infections. It has been previously reported that individuals affected by trisomy 21 have degenerative brain changes and are frequently found after birth to have large lateral ventricles[7,25]. It is possible that idiopathic developmental delay may represent the consequence of subtle cerebral maldevelopment initiated by isolated cerebral borderline ventriculomegaly.

The risk of an abnormal outcome, and particularly of delayed neurological development, seems to be influenced by several factors, namely sex, size of the ventricles and gestational age at diagnosis. Male fetuses have a slightly greater atrial width than females[21,22]. Therefore, it is not surprising that males are found to have borderline ventriculomegaly more frequently, and to have a significantly lesser degree of neurological compromise than females. The available data suggest that the risk of delayed development for a male fetus with isolated cerebral borderline

ventriculomegaly and a normal karyotype is 5%. For a female fetus, the risk increases to 23%. An abnormal outcome was also significantly less frequent when the atrial width was < 12 mm (4% vs. 14%). We believe that this is important information, as three-quarters of cases thus far reported fall into this category. On the basis of the normative studies and clinical series available, we also suggest that the presently accepted definition of borderline cerebral ventriculomegaly applies principally to the second and early third trimesters, and that ventricles of 10–11 mm have very little, if any, significance in the late third trimester.

Intrauterine remission of borderline ventriculomegaly is frequently documented. It is unclear whether or not this implies an amelioration of the prognosis. Patel and coworkers failed to demonstrate a difference in the outcome between cases with stable or progressing ventriculomegaly and cases with spontaneous remission[6]. However, it is difficult to comment upon spontaneous remission when most studies have been based upon measurement of only one atrium, while it is now clear that many cases are unilateral[11]. It is obvious that, unless both atria are visualized and measured, demonstration of remission is not acceptable.

Although a significant number of cases of fetal isolated cerebral borderline ventriculomegaly have been described, it remains difficult to draw conclusions on the clinical significance of this finding. The available reports have several shortcomings. In most series, including our own, the diagnosis of ventriculomegaly was established by measurement of only one atrium. It has been more recently demonstrated that isolated cerebral borderline ventriculomegaly is frequently unilateral, and it has been proposed that the unilateral type is benign. We do not subscribe to this view. In the original report, two of 30 fetuses with the unilateral condition had an abnormal outcome (one fetus had trisomy 21, one infant had seizures)[11]. Furthermore, the duration of follow-up was shorter than in most other studies. There is a need for a prospective comparison between bilateral and unilateral isolated cerebral borderline ventriculomegaly.

Another shortcoming of the available experience is the type and duration of the postnatal follow-up. Most series reported an excess of neurodevelopmental abnormalities. However, different modalities for assessment of the infants were used, and the results are difficult to compare. In most cases, the evaluation was performed by the family pediatricians on a qualitative basis. Follow-up rarely extended beyond the 3rd year of life. It is important to stress that these results should be interpreted with caution.

The optimal management of the condition remains uncertain. We agree with others[6,7] that it would be prudent to offer fetal karyotyping, particularly when other sonographic markers of aneuploidy are present. A TORCH screen is commonly recommended, although thus far an association has not been demonstrated. Isolated cerebral borderline ventriculomegaly may be the harbinger of severe cerebral lesions that cannot be predicted in early gestation. This occurs rarely, but it is obviously a reason for major concern. Fetal magnetic resonance imaging has been recently employed to diagnose subtle cerebral maldevelopment in fetuses with isolated cerebral borderline ventriculomegaly[26]. Unfortunately, the diagnostic capability of this technique prior to fetal viability is still relatively undetermined. Infants with isolated cerebral borderline ventriculomegaly are at increased risk for developmental delay. It has been suggested that this finding could represent an indication for early childhood intervention, as special education programs maximize developmental potential[8]. However, counselling these couples is a major undertaking. It has been our experience that the communication that the infant has even a slightly increased risk of cerebral damage, or low intellect, causes major distress to most couples. We expect that at least some patients will request termination of pregnancy. This will raise a difficult ethical dilemma, as most cases result in the birth of healthy infants.

ACKNOWLEDGEMENT

This investigation was supported by the University of Bologna (funds for selected research topics).

REFERENCES

1. Leopold G. Antepartum obstetrical ultrasound guidelines. *J Ultrasound Med* 1986;5:241–2
2. Goldstein RB, La Pidus AS, Filly RA, Cardoza J. Mild lateral cerebral ventricular dilatation *in utero*: clinical significance and prognosis. *Radiology* 1990;176:237–42
3. Vergani P, Locatelli A, Strobelt N, Cavallone M, Ceruti P, Paterlini G, Ghidini A. Clinical outcome of mild fetal ventriculomegaly. *Am J Obstet Gynecol* 1998;178:218–22
4. Mahony BS, Nyberg DA, Hirsch JH, Petty CN, Hendricks SK, Mack LA. Mild idiopathic lateral cerebral ventricular dilatation *in utero*: sonographic evaluation. *Radiology* 1988;169: 715–21
5. Bromley B, Frigoletto FD Jr, Benacerraf BR. Mild fetal lateral cerebral ventriculomegaly: clinical course and outcome. *Am J Obstet Gynecol* 1991;164:863–7
6. Patel MD, Filly AL, Hersh DR, Goldstein RB. Isolated mild fetal cerebral ventriculomegaly: clinical course and outcome. *Radiology* 1991;192:759–64
7. Achiron R, Schimmel M, Achiron A, Mashiach S. Fetal mild idiopathic lateral ventriculomegaly: is there a correlation with fetal trisomy? *Ultrasound Obstet Gynecol* 1993;3:89–92
8. Bloom SL, Bloom DD, Dellanebbia C, Martin LB, Lucas MJ, Twickler DM. The developmental outcome of children with antenatal mild isolated ventriculomegaly. *Obstet Gynecol* 1997;90:93–7
9. Heiserman J, Filly RA, Goldstein RB. The effect of measurement errors on the sonographic evaluation of ventriculomegaly. *J Ultrasound Med* 1991;10:121–4
10. Alagappan R, Browning PD, Laorr A, McGahan JP. Distal lateral ventricular atrium: reevaluation of normal range. *Radiology* 1994;193:405–8
11. Lipitz A, Malinger G, Meizner I, Zalel Y, Achiron R. Outcome of fetuses with isolated borderline unilateral ventriculomegaly diagnosed at mid-gestation. *Ultrasound Obstet Gynecol* 1998; 12:23–6
12. Goldstein I, Reece EA, Pilu G, Hobbins JC, Bovicelli L. Sonographic development of the normal developmental anatomy of fetal cerebral ventricles: I. The frontal horns. *Obstet Gynecol* 1988;72:588–92

13. Pilu G, Reece EA, Goldstein I, Hobbins JC, Bovicelli L. Sonographic evaluation of the normal developmental anatomy of the fetal cerebral ventricles: II. The atria. *Obstet Gynecol* 1989;73:250–5
14. Goldstein I, Reece EA, Pilu G, Hobbins JC. Sonographic evaluation of the normal developmental anatomy of the fetal cerebral ventricles. IV. The posterior horn. *Am J Perinatol* 1990;7:79–83
15. Monteagudo A, Timor Tritsch IE, Moomjy M. Nomograms of the lateral ventricles using transvaginal sonography. *J Ultrasound Med* 1993;12:265–9
16. Filly RA, Goldstein RB, Callen PW. Fetal ventricle: importance in routine obstetric sonography. *Radiology* 1991;181: 1–7
17. Cardoza JD, Goldstein RB, Filly RA. Exclusion of fetal ventriculomegaly with a single measurement: the width of the lateral ventricular atrium. *Radiology* 1988;169:711–14
18. Snijders RJM, Nicolaides KH. Fetal biometry at 14–40 weeks' gestation. *Ultrasound Obstet Gynecol* 1994;4:34–48
19. Kier EL. The cerebral ventricles: a phylogenetic and ontogenetic study. In Newton TH, Potts DG, eds. *Radiology of the Skull and Brain: Anatomy and Pathology*. St Louis: CV Mosby, 1977:2787–911
20. Achiron R, Yagel S, Rotstein Z, Inbar O, Mashiach S, Lipitz S. Cerebral lateral ventricular asymmetry: is this a normal ultrasonographic finding in the fetal brain? *Obstet Gynecol* 1997; 89:233–7
21. Patel MD, Goldstein RB, Tung S, Filly RA. Fetal cerebral ventricular atrium: difference in size according to sex. *Radiology* 1995;194:713–15
22. Nadel AS, Benacerraf BR. Lateral ventricular atrium: larger in male than female fetuses. *Int J Gynecol Obstet* 1995;51:123–6
23. Nyberg DA, Resta RG, Luthy DA, Mahony BS, Hirsch JH. Prenatal sonographic findings of Down syndrome: review of 94 cases. *Obstet Gynecol* 1990;76:370–7
24. Deren O, Mahoney MJ, Copel JA, Bahado-Singh RO. Subtle ultrasonographic anomalies: do they improve the Down syndrome detection rate? *Am J Obstet Gynecol* 1998;178:441–5
25. LeMay M, Alvarez N. The relationship between enlargement of the temporal horns of the lateral ventricles and dementia in aging patients with Down's syndrome. *Neuradiology* 1990; 32:104–7
26. Greco P, Resta M, Vimercati A, Dicuonzo F, Loverro G, Vicino M, Selvaggi L. Antenatal diagnosis of isolated lissencephaly by ultrasound and magnetic resonance imaging. *Ultrasound Obstet Gynecol* 1998;12:276–9