

## ORIGINAL ARTICLE

## Neonatal lenticulostriate vasculopathy: further characterisation

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**Background:** Lenticulostriate vasculopathy (LSV) is sometimes detected on routine brain ultrasonography in neonates, and is often associated with various perinatal and neonatal abnormalities. However, most reports on LSV are retrospective with no controls.

**Objectives:** To compare the perinatal and neonatal clinical characteristics of neonates with LSV with matched controls and to summarise all published reports of LSV.

**Design:** A prospective study that summarises the clinical, laboratory, and neurosonographic data of neonates with LSV.

**Methods:** Of 1184 neonates admitted to the neonatal intensive care unit (NICU) during a three year period, 857 had a routine head ultrasound examination. Twenty one had LSV, and were compared with 42 matched controls with regard to gestational, perinatal, neonatal, laboratory, and neurosonographic characteristics.

**Results:** LSV was detected in 21 of the 857 (2.45%) neonates. It was bilateral in 10 of the 21 cases and located in the thalamus ( $n = 14$ ) and basal ganglia ( $n = 7$ ). Infants with LSV were not significantly different from matched controls in most tested variables. However, compared with the control group, the LSV group included significantly more multiple births and more disturbances in amniotic fluid volume, but less meconial amniotic fluid. In addition, the patients with LSV required fewer blood transfusions and less phototherapy.

**Conclusions:** Except for more multiple births, neonates with LSV did not display more adverse findings than their matched controls.

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Lenticulostriate vasculopathy (LSV) is detected in 0.4% of all liveborn neonates<sup>1</sup> and 1.9–5.8% of ill neonates,<sup>2–7</sup> using cranial ultrasound (US) as hyperchogenic arteries in the thalamus and basal ganglia.

LSV can be either unilateral or bilateral, with a branching, linear (strip-like), or punctate shaped pattern.<sup>1,4,5,8</sup> Histopathological examination shows that most lenticulostriate arteries involved in LSV are medium sized, have thickened and hypercellular walls without fibrosis or hyalinisation, but with intramural and perivascular deposition of amorphous basophilic material, iron, and calcium,<sup>4,5,9</sup> and signs of vessel wall damage.<sup>4</sup>

Since the initial description of LSV by Grant *et al*<sup>10</sup> in 1985, more than 25 reports of neonatal LSV have been published, in which LSV appeared to be associated with a variety of congenital and acquired neonatal conditions, such as fetal and neonatal infections, mainly cytomegalovirus,<sup>1–4,6–9,11–15</sup> chromosomal aberrations,<sup>2–4,6,8,9,16,17</sup> hypoxic/ischaemic conditions,<sup>4–6</sup> congenital heart disease,<sup>4</sup> fetal alcohol or drug exposure,<sup>3–6</sup> congenital malformations,<sup>1,4,6</sup> neonatal lupus erythematosus,<sup>3,6</sup> twin to twin transfusion,<sup>18</sup> sialidosis,<sup>19</sup> hydrops fetalis,<sup>6</sup> and diabetic fetopathy.<sup>1</sup>

Most published reports of LSV are either of large retrospective studies<sup>2–7</sup> or are case reports,<sup>10–24</sup> except for one prospective study by Kriss *et al*<sup>8</sup> in which prospective screening of brain sonograms performed in the neonatal intensive care unit (NICU), rather than of neonates, was conducted. From the literature on LSV, no clear conclusions can be drawn about the consequences of LSV on the sick neonate. Therefore we performed a prospective study with the following aims: (a) to summarise the clinical, laboratory, and neurosonographic data of all neonates with LSV in our NICU; (b) to compare the perinatal and neonatal clinical characteristics of neonates with LSV with those of matched controls; (c) to elucidate possible meaningful clinical trends from our observations. We expected

to discern characteristics that would delineate the relevance of a finding of LSV in the sick neonate.

## METHODS

## Study design and groups

This prospective study was conducted between 1 February 1999 and 31 January 2002 in the tertiary centre NICU at Rambam Medical Center. During the study period, 1184 neonates were admitted; 857 of them had a routine head US examination. The remaining 327 included neonates transferred to our NICU for surgical interventions, infants whose stay in the NICU was short term (transient tachypnoea of newborn, polycythaemia, jaundice), and some extremely low birthweight infants who died within 48 hours of admission.

Of the 857 neonates who had head US examinations, LSV was detected in 21 (2.45%); these comprised the study group. Each patient in the study group was matched with the two subsequent neonates born at the same gestational age ( $\pm 3$  days) and in the NICU, but who did not have LSV (control group;  $n = 42$ ). Neonates of both groups were born after 27–41 weeks gestation. When LSV occurred in one or both twin neonates, they were matched to control twin neonates, observing the same order of twinning (first with first, second with second).

## Data collection

In both the study and control group, data were prospectively collected on the neurosonographic characteristics of LSV (table 1), maternal, gestational, and perinatal characteristics

**Abbreviations:** LSV, lenticulostriate vasculopathy; NICU, neonatal intensive care unit; US, ultrasonography; TORCH, toxoplasma, other viruses, rubella, cytomegalovirus, herpes virus

**Table 1** Sonographic characteristics of neonates with LSV (n=21)

Variable	Number
NICU admissions during the study period	1184
Neonates with head sonograms	857 (72.4%)
Number of neonates with LSV	21 (2.45%)
LSV diagnosed on 1st US	19
LSV diagnosed on 2nd US	2
Age at examination (days) 1st US	8.8 (5.4)
LSV	
Unilateral	11
Bilateral	10
Location of LSV	
Thalamus	14
Basal ganglia	7
IVH	
Grade 1–2	3
Grade 3–4	2
Ventriculomegaly	2
Hydrocephalus	0

LSV, Lenticulostriate vasculopathy; NICU, neonatal intensive care unit; IVH, intraventricular haemorrhage.

(table 2), and neonatal variables and medical interventions (table 3). In addition, we conducted a comprehensive review of the literature on neonatal LSV (table 4). We could not perform a long term neurodevelopmental follow up in our series because of lack of data for most patients.

### Head US examination

A first head US examination was performed at 8.2 (5.4) days and 8.6 (6.1) days after birth in the study and control groups respectively (mean (SD)). Scans were performed with a 5 MHz convex transducer (SSD-1400; Aloka Co, Tokyo, Japan) and a 5–8 MHz curved transducer (HDI 5000; Advanced Technology Laboratories, Bothell, Washington, USA). Each examination included coronal and bilateral parasagittal views of the brain. Colour Doppler examination was performed in three cases.

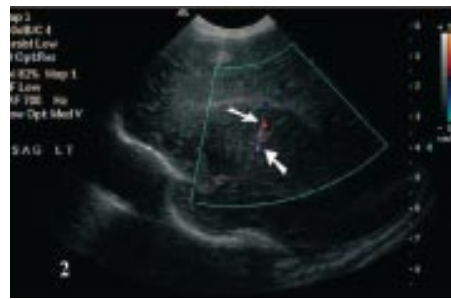
### Statistical analysis

For comparison of the results of the study and control groups, we used Student's *t* test for continuous variables (duration of mechanical ventilation), the Wilcoxon test for ordinal variables (Apgar score), and the  $\chi^2$  test for dichotomic variables.  $p < 0.05$  was considered significant.

## RESULTS

### Incidence and sonographic characteristics of LSV

Table 1 shows that the incidence of LSV in our study was 2.45% of NICU admissions. The gestational age distribution of the 21 patients with LSV was as follows: < 30 weeks, one; 30–33 weeks, six; 34–37 weeks, eight; > 37 weeks, six. LSV was mostly of linear branching pattern (fig 1A,B) and confirmed to be vascular by colour Doppler US in three neonates (fig 2). In



**Figure 2** Sagittal colour Doppler image showing the vascular nature of these thalamic echogenicities (arrows).

two of the 21 cases, LSV was first seen on the second US examination at 10 and 21 days of age. LSV was bilateral in 10 of the 21 (48%) cases, and was located in the thalamus in 14 cases and in the basal ganglia in seven. It was accompanied by intraventricular haemorrhage in five cases, and by ventriculomegaly in two.

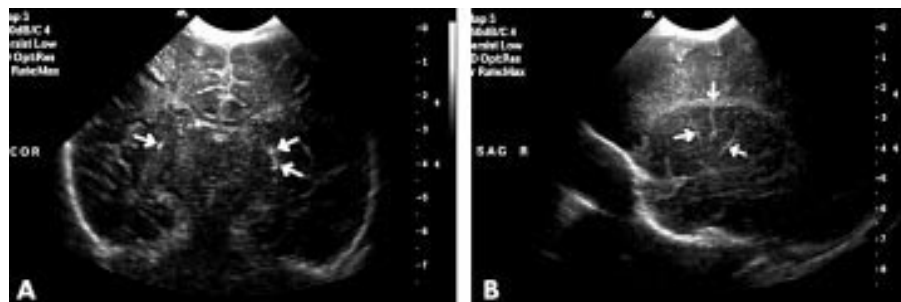
### Comparison of study and control groups

Table 2 shows a comparison of the study and control groups with regard to various maternal, gestational, and perinatal variables. Compared with the control group, the LSV group included significantly more multiple births ( $p = 0.013$ ), more disturbances in amniotic fluid volume (oligohydramnion or polyhydramnion;  $p = 0.015$ ), and less meconium stained amniotic fluid ( $p < 0.01$ ). All the remaining variables were not significantly different between the groups. The LSV group included five infants with congenital anomalies: two with cleft lip and palate; one with persistent omphalomesenteric duct; one with congenital contractural arachnodactyly; one with preauricular tags.

Table 3 shows a comparison of the study and control groups with regard to various neonatal variables, medical interventions, and outcome. Compared with the control group, the LSV group required significantly fewer blood transfusions ( $p < 0.05$ ) and less phototherapy ( $p < 0.01$ ). All the remaining variables were not significantly different between the groups. There were no cases of hypothyroidism or twin to twin transfusion.

## DISCUSSION

Our results indicate that LSV is not a rare finding during routine brain US examination of NICU neonates. We encountered LSV in 2.45% of our NICU admissions, conforming with a reported incidence of 1.9–5.8%.<sup>2–6</sup> In our series, 47.6% of LSV was bilateral, similar to a reported 40–82% bilateral location.<sup>3,7</sup> The incidence of LSV in our series was similar to that previously reported, in spite of including relatively more mature neonates (66.6% were more than 33 weeks gestation), compared with those described by Chamnanvanakij *et al.*<sup>7</sup> Of note is the fact that in only 72.4% of our NICU admissions was



**Figure 1** Ultrasound through anterior fontanelle showing branching echogenic linear structures in the thalamus (arrows). (A) Coronal scan; (B) sagittal scan.

**Table 2** Maternal, gestational, and perinatal characteristics of the study and control groups

Variable	Study group (n=21)	Control group (n=42)
Mother's age (years)	27.8 (6.7)	30.6 (5.4)
Jewish ethnic origin	14 (66.6%)	29 (69%)
Spontaneous fertilisation	16 (76.2%)	33 (78.6%)
Multiple births	11 (52.3%)	9 (21.4%)*
Maternal smoking in pregnancy	0	3 (7.1%)
Maternal drugs during pregnancy		
MgSO <sub>4</sub>	2 (9.5%)	1 (2.4%)
Indomethacin	1 (4.8%)	1 (2.4%)
Ritodrine	0	1 (2.4%)
Betamethasone	2 (9.5%)	7 (16.6%)
Gestational problems		
Vaginal bleeding	2 (9.5%)	4 (9.5%)
PET/PIH/CHT	2 (9.5%)	5 (11.9%)
GDM/IDDM	0	3 (7.1%)
Poly/oligohydramnion	3/3	1/1*
Alcohol/drug consumption	0	0
Gestational age (weeks)	35.0 (3.3)	35.1 (3.4)
Birth weight (g)	2352 (752)	2388 (768)
Male	12 (57.1%)	26 (61.9%)
Non-reassuring fetal heart rate	1 (4.8%)	0
PROM >12 hours	3 (14.3%)	12 (28.5%)
Chorioamnionitis	0	3 (7.1%)
Meconial amniotic fluids	1 (4.8%)	15 (35.7%)*
Mode of delivery		
Vaginal	10 (47.6%)	21 (50%)
Caesarean	10 (47.6%)	19 (45.2%)
Vacuum/forceps	0	2 (4.8%)
Five minute Apgar score	9.5 ± 0.6	9.3 ± 1
Small for gestational age	3 (14.3%)	7 (16.7%)
Microcephaly	1 (4.8%)	4 (9.6%)
Congenital malformations	5 (23.8%)	3 (7.1%)
Chromosomal aberrations	0	1 (2.4%)

If not indicated otherwise, values are mean (SD).

\*Significantly different from study group.

PET, Pre-eclamptic toxemia; PIH, pregnancy induced hypertension; CHT, chronic hypertension; GDM, gestational diabetes mellitus; IDDM, insulin dependent diabetes mellitus; PROM, prolonged rupture of membranes.

**Table 3** Neonatal morbidities, medical interventions, and outcome in the study and control groups

Variable	Study group (n=21)	Control group (n=42)
Respiratory distress syndrome	8 (38%)	13 (30.9%)
Patent ductus arteriosus	1 (4.8%)	1 (2.4%)
Apnoea	1 (4.8%)	7 (16.7%)
Metabolic acidosis (pH <7.25)	3 (14.3%)	3 (7.1%)
Hypoglycaemia (serum glucose <45 mg/dl)	5 (23.8%)	18 (42.9%)
Haematological		
Haemoglobin <14 g/dl	7 (33.3%)	20 (47.6%)
Packed cell volume >65%	0	1 (2.4%)
Leucocytes <5000/mm <sup>3</sup>	0	4 (9.6%)
Platelets <100000/mm <sup>3</sup>	3 (14.3%)	2 (4.8%)
Hyperbilirubinaemia		
Serum bilirubin >13 mg/dl	6 (28.6%)	18 (42.9%)
Peak serum bilirubin (mg/dl)	11 (2.2)	12.2 (3.4)
Congenital viral infection	1 (CMV)	0
Acquired bacterial sepsis	3 (14.3%)	6 (14.3%)
Acquired fungal sepsis	1 (4.8%)	1 (4.8%)
Necrotising enterocolitis	0	0
Intraventricular haemorrhage		
Grade 1–2	4 (19%)	6 (14.3%)
Grade 3–4	1 (4.8%)	2 (4.8%)
Seizures	0	2 (4.8%)
Mechanical ventilation	7 (33.3%)	13 (30.9%)
Duration (days)	2.1 (5.2)	1.1 (2.1)
Umbilical artery catheterisation	3 (14.3%)	3 (7.1%)
Duration (days)	0.48 (1.6)	0.33 (1.4)
Umbilical vein catheterisation	6 (28.6%)	10 (23.8%)
Duration (days)	1.52 (2.9)	0.83 (1.8)
Surfactant replacement therapy	4 (19%)	4 (9.6%)
Blood transfusion	1 (4.8%)	11 (26.2%)*
Exchange transfusion	1 (4.8%)	1 (2.4%)
Phototherapy	5 (23.8%)	25 (59.5%)*
Duration (days)	0.95 (1.9)	1.98 (2.3)
Hospital stay (days)	39.8 (81.1)	23.8 (17.3)
Mortality	0	0

If not indicated otherwise, values are mean (SD).

\*Significantly different from study group.

CMV, Cytomegalovirus.

head US examination carried out, with those not imaged being mostly healthier than those examined. When we matched our LSV cases with controls of the same gestational age, no trends of LSV with gestational age emerged. Therefore we could not determine the incidence of LSV in the normal term neonatal population. However, the gestational age distribution of our LSV cases shows that 28% (6/21) were term infants, whereas only 5% (1/21) were less than 30 weeks gestation.

Routine ultrasound examination of the brain in NICU neonates appears to be the best tool for detecting LSV; vessel patency can be confirmed by colour Doppler US examination.<sup>3–5 8 11</sup> In contrast with US, computed tomography of the brain generally failed to show increased attenuation of vessels in the thalamus or basal ganglia in LSV cases. Brain computed tomography in 60 sonographically diagnosed LSV cases failed to display the lesion,<sup>1 3–6 8 9 11</sup> except in five cases (two with cytomegalovirus; one with toxoplasmosis; one with AIDS; one with fetal alcohol syndrome).<sup>6</sup> Magnetic resonance imaging also failed to show LSV in most patients studied,<sup>3–5</sup> and in only four of 23 patients did it show areas of linear increased signal in regions corresponding to the thalamus and basal ganglia.<sup>3</sup> The superiority of US in detecting LSV is further emphasised by its ability to detect LSV prenatally, as shown by Estroff *et al*<sup>24</sup> in the brain of a fetus with cytomegalovirus.

LSV has been proposed as a marker of diffuse insult to the fetal and neonatal brain.<sup>4 25 26</sup> Various underlying perinatal and neonatal conditions have been reported to be associated with LSV, mainly fetal TORCH (toxoplasma, other viruses, rubella, cytomegalovirus, herpes virus) infections,<sup>1–7 9</sup> chromosomal

aberrations,<sup>2–6 8 9</sup> congenital malformations,<sup>1 5 6 8</sup> congenital heart disease,<sup>4</sup> and asphyxia.<sup>4</sup> Our LSV group was not significantly different from the control group in most variables tested, in spite of including significantly more multiple births. Overall, the neonates with LSV in our study were not sicker than controls, but were actually healthier, with less meconial amniotic fluid, fewer blood transfusions, and less phototherapy. We do not have a sound explanation for the increased incidence of LSV in twins and with abnormal liquor volumes. One speculation is that there is a higher risk of impaired placental flow in these two conditions. Similarly, we could not explain the lower incidence of LSV in infants with meconium stained fluids, or the fact that infants with LSV seem to be less sick. This, however, indicates that LSV did not pose any additional risk in the early neonatal period. Unfortunately, we do not perform routine head US examinations on all infants discharged from the NICU during follow up and therefore we may have missed cases of later LSV.

Table 4 summarises the largest published reports of LSV, including the data from this study. We could not perform a meta-analysis because most of the reports were either retrospective or included a review of brain sonograms without direct reference to patients. In addition, these reports included heterogeneous populations of neonates: premature infants weighing <1250 g,<sup>7</sup> NICU admissions,<sup>2 5</sup> all neonatal admissions,<sup>1</sup> and also infants up to 11 months of age.<sup>3</sup> A summary of LSV cases from large reports with study populations of known size (table 4) shows that, compared with the general neonatal population, patients with LSV have a higher incidence of asphyxia, respiratory disease, congenital heart disease, fetal TORCH infection, chromosomal aberrations, and

**Table 4** Summary of large reports of neonatal lenticulostriate vasculopathy (LSV)

	Coley <sup>4</sup> 2000	Chamnanvanakit <sup>7</sup> 2000	Shefer-Kaufman <sup>2</sup> 1999	Kriss <sup>8</sup> 1996	Wang <sup>3</sup> 1995	Cabanias <sup>5</sup> 1994	Weber <sup>1</sup> 1992	Hughes <sup>6</sup> 1991	Teale <sup>9</sup> 1988	This study 2002	References 1-6 & Makhoul <sup>10</sup>
Number of infants	1500	193	3700	1746	586	1893	3600	1324	4500	857	13460
Number of head US	63 (4.2%)	10 (5.1%)	75 (2%)	3	34 (5.8%)	4272	15 (0.4%)	2320	12	21 (2.45%)	270 (2%)
LSV	24-41	28 (1.4)	27-43			28-41	26-40	25 (1.9%)	35-40	35 (3.3)	
Gestational age (weeks)	2458 (1107)	907 (224)	880-4500			1075-4250	900-4300	9 (36%)	1400-3700	2351 (752)	
Birth weight (g)	29 (46%)	10 (100%)	26 (35%)			12 (32.4%)	5 (33.3%)	10/15	2 (16.6%)	14 (66.6%)	98 / 270 (36.3%)
Prematurity	37/26	5/5	40/35			18/19	9/6	5	4/8	12/9	126/110
Male/female ratio	5	0				4	2	0	0	0	16 / 140 (11.4%)
Asphyxia	15	5				3	3	1	0	8	30 / 161 (18.6%)
Respiratory disease*	13	0				1	1	3	0	0	18 / 140 (12.9%)
Congenital heart disease	5	4	1	0	6	5	7	4	8	1	29 / 270 (10.7%)
Viral infection†	9	0	4	3	3	1	0	3	3	0	20 / 270 (7.4%)
Chromosomal aberrations‡	4	0		0	2	3	0	1	0	0	10 / 195 (5.1%)
Fetal exposure (alcohol, drugs)	4	0		3	0	1	2	3	6/12 (50%)	0	25 / 140 (17.8%)
Congenital malformations	15/63 (23.8%)			3/3 (100%)	0	11	2	3		0	19 / 121 (15.7%)
Mortality (%)						4/37 (10.8%)					

Some neonates had more than one diagnosis; cells without numbers indicate no available data.

\* Includes respiratory distress syndrome, meconium aspiration syndrome, pneumonia, bronchopulmonary dysplasia.

† Includes cytomegalovirus (26), rubella (4), rotavirus (5).

‡ Includes trisomy 13 (16), trisomy 21 (8), other (1).

§ Reference 7 was not included because it included a selected population (premature infants < 1250 g); references 8 and 9 were not included because the size of the study population was not recorded.

congenital malformations. These findings imply that LSV is most probably associated with these adverse conditions.

Sonographic follow up of patients with LSV up to 15 months of age showed progression of LSV in 14.7–85% of cases,<sup>3,4</sup> no change in 15–86.5%,<sup>1,3,5</sup> or resolution of lesions in 36.4–50% of cases.<sup>1,7</sup> However, the extent of LSV progression was determined sonographically, but was without clear criteria for the estimation of progression and was operator dependent in terms of awareness of this finding. Nonetheless, progression of LSV could reflect the consequences of brain insult by unfavourable underlying conditions.

Follow up studies reported a neurodevelopmental delay in 18.9–55% of patients with LSV.<sup>1,5,11</sup> Furthermore, in premature infants of birth weight < 1250 g, those with LSV had lower scores for mental development, motor quality, and emotional regulation than matched controls.<sup>7</sup> Most infants with LSV and developmental delay experienced severe disease states, such as cytomegalovirus infection,<sup>9,11</sup> major malformations or chromosomal aberrations,<sup>1,5</sup> fetal exposure to alcohol,<sup>5</sup> and systemic *Streptococcus* sepsis.<sup>11</sup> Prematurity by itself, with or without respiratory distress syndrome, does not appear to adversely affect the short term developmental outcome of patients with LSV.<sup>3,4,7</sup> In the absence of severe underlying conditions such as fetal TORCH infections, chromosomopathy, major malformations, or hypoxic-ischaemic states,<sup>25,26</sup> survival without adverse clinical outcome is the rule in patients with LSV. However, in survivors with LSV who did not have known associated factors, an initially normal development did not ensure a subsequent favourable course, as long term follow up has shown that some patients develop tics, attention deficit, or hyperactivity disorders.<sup>27</sup> However, this needs to be confirmed.

The reported mortality in patients with LSV is 8.1–50%.<sup>4,5,9</sup> Review of the literature shows that those who died had severe underlying conditions, such as trisomy 13,<sup>4,9</sup> TORCH infections,<sup>3,6</sup> major congenital heart diseases,<sup>4,6</sup> fetal heroin exposure.<sup>5</sup> In our series, the mere presence of LSV did not increase mortality and there were no deaths in either the study or control groups.

We conclude that patients with LSV do not have more adverse findings than their matched controls with regard to most gestational, perinatal, and neonatal characteristics and outcome, except for an increased proportion of multiple births. LSV appears to represent a perinatal diffuse insult to the fetal and neonatal brain. A poor outcome in a patient with LSV can be expected when a severe underlying condition coexists. In the absence of known associated conditions, LSV has a good short term prognosis, but unfavourable behavioural and neurological features may become apparent in childhood. Therefore neonatal LSV mandates close neurodevelopmental follow up beyond infancy.

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## Neonatal lenticulostriate vasculopathy: further characterisation

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