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# Subdural Neomembranes and Sudden Infant Death Syndrome*

**REFERENCE:** Rogers CB, Itabashi HH, Tomiyasu U, Heuser ET. Subdural neomembranes and sudden infant death syndrome. J Forensic Sci 1998;43(2):375–376.

**ABSTRACT:** Cranial dura maters of 36 consecutive infants with sudden infant death syndrome (SIDS) and 16 control infants coming to the Department of Coroner were examined microscopically to determine if subdural neomembranes are associated with cases submitted as SIDS. Thirty-one percent (31%) of the infants with SIDS and 13% of control infants had organizing subdural neomembranes ($p > 0.05$). Overall prevalence of organizing subdural neomembranes was 25% in the group examined.

In all but two cases, birth trauma could be excluded as a cause of head trauma by aging neomembranes histologically. No association was found between type of delivery (vaginal or Cesarean) and presence of a subdural neomembrane.

Subdural neomembranes are common in infants autopsied in a forensic setting, but they may be missed without a microscopic examination. Subdural neomembranes have no demonstrated association with SIDS.

**KEYWORDS:** forensic science, head trauma, subdural hemorrhage, sudden infant death syndrome

Sudden unexpected infant death accounts for the large majority of deaths in infants under the age of one year autopsied in forensic offices. An extensive literature exists on various possible causes of sudden infant death (1), but no single cause of SIDS has been identified.

Little published work exists on the relationship of trauma to SIDS. Kinney et al. (2) showed a statistical increase in reactive astrocytes in the medulla oblongata of victims of SIDS compared to controls. It is unclear whether this finding is due to injury or to some other cause. We have recently noted a large number of subdural neomembranes in infants autopsied at our office. The usual autopsy techniques may miss such neomembranes. For example, the California Sudden Infant Death Protocol (3) requires gross examination of the dura, but not microscopic examination.

The present study is a prospective controlled comparison of infants who died of SIDS with those who died unexpectedly for other reasons. It addresses the hypothesis that there is a difference in the prevalence of subdural neomembranes between the two groups. We also determined the estimated age of the subdural neomembranes to see if they were consistent with birth trauma.

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## Methods

Cases for study included consecutive liveborn infants, age one year or less, autopsied at the Los Angeles County Department of Coroner. Cases were excluded from the study if there was a history of head injury. Each case had a complete autopsy, including gross examination of the dura, microscopic examination, toxicology and full-body X-rays according to the California Sudden Infant Death Protocol (3). Thirty-six (36) cases of SIDS and sixteen (16) cases of sudden infant death from other causes were studied. In the latter group, four deaths were due to congenital anomalies, three to bronchopneumonia, two to consequences of prematurity, two to seizure disorder, and one each to interstitial pneumonitis, sickle cell anemia, aspiration, viral gastroenteritis, and homicidal asphyxia.

The medical history was examined when possible to determine whether the delivery had been vaginal or by Cesarean section. If subdural hemorrhage were due to birth trauma, then subdural neomembranes should be rare in infants delivered by Cesarean section.

In each case, six sections of dura mater were examined microscopically. One section was taken from each side of the middle cranial fossa, posterior cranial fossa, and parasagittal region. Hematoxylin and eosin stained sections were examined by two neuropathologists without knowledge of the infant's history. The case was considered to show a neomembrane if one was present in any section. The ages of neomembranes were determined according to modified criteria after Leestma (4): 0–2 days—Intact red blood cells, with few or no fibroblasts (possibly autopsy artifact); 3–5 days—Proliferation of fibroblasts aligned parallel to dura, and macrophages; breakdown of red blood cells and hemosiderin, 6–10 days—Layered fibroblasts of several cells thickness and hemosiderophages; 11–21 days—Fibroblastic layering and neovascularization 12 or more cells thick; many hemosiderophages; 3–6 weeks—Giant capillaries and early collagen deposition; >6 weeks—Hyalinization of dense collagen, regressing neovascularization.

Results were analyzed by the Chi-square method to determine if SIDS is associated with subdural neomembranes. In cases with neomembranes, the ages of the neomembranes were compared with the ages of the infants to determine if the neomembranes could be due to birth trauma.

## Results

Neomembranes were present in 19 of the 36 cases of SIDS (53%) and in 3 of 16 control cases (19%). This difference was statistically significant ($p < 0.05$). However, when the very recent subdural hemorrhages (0–2 days), which might be related to removal artifact, are excluded, the prevalence of neomembranes

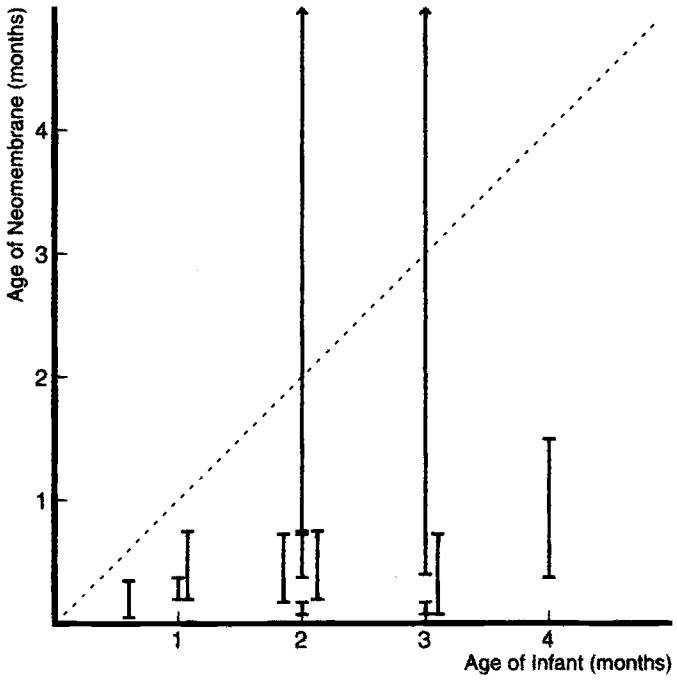


FIG. 1—*Ages of neomembrane (Y-axis) as a function of age of infant (X-axis). Bars show ranges of age estimates.*

in cases of SIDS was 11 in 36 cases (31%), and in control cases, 2 in 16 cases (13%). This difference was not statistically significant ($p > 0.05$). The overall prevalence of subdural neomembranes, excluding very recent hemorrhages, was 25%.

Neomembranes occurred in the posterior fossa (12 on the right and 9 on the left), the middle cranial fossa (4 on the right and 10 on the left) and the parasagittal regions (7 on the right and 4 on the left). There were 14 neomembranes aged 0–2 days, four aged 3–5 days, four aged 6–10 days, four aged 14–21 days, and two aged over three weeks.

Information on the method of delivery, whether vaginal or Cesarean, was available in 23 cases. Neomembranes were present in 6 of 15 infants delivered vaginally (40%), and in 2 of 8 infants delivered by Cesarean section (25%). This difference was not statistically significant ($p > 0.05$). After very recent neomembranes were excluded, neomembranes were present in 4 of 15 infants delivered vaginally (27%) and 1 of 8 infants delivered by Cesarean section (13%) ($p > 0.05$).

A comparison of the ages of the subdural neomembranes with the ages of the decedents is given in Fig. 1. The bars in the Fig. 1 show the ranges of the age estimates for neomembranes. In every case except two, the neomembrane was not present at the time of birth. In the remaining two cases, the neomembrane was over 6 weeks old. It was therefore not possible to determine whether it was present at birth.

## Discussion

This study did not show any association between the presence of organized subdural neomembranes and SIDS. However, there was a high prevalence of subdural neomembranes in the infants, suggesting that many subdural neomembranes are not detected by the gross examination of the dura used in most infant autopsy procedures. Other investigators have found a prevalence of 11 to 49% in infant autopsies (5,6).

The differential diagnosis of subdural hemorrhage in infancy includes trauma (7) and blood clotting disorders (8,9). There was no history of significant head trauma in any of our cases. In most of the cases studied, birth trauma was excluded by comparing the age of the subdural neomembrane with the age of the infant (Fig. 1), as well as by the lack of association between vaginal delivery and subdural neomembrane. In addition, SIDS cases almost by definition have almost no incidence of difficult delivery. While clotting disorders may have been present in our cases, they are not documented and do not explain the high prevalence of subdural neomembranes. Nontraumatic rupture of bridging veins has not been reported in the absence of hematologic disease.

Friede and Schachenmayr (10,11) have hypothesized that any pathologic condition separating the dura and arachnoid meninges will produce a neomembrane. This hypothesis might explain the roughly uniform distribution of subdural neomembranes observed in this study. If the subdural neomembranes were traumatic, they should occur most commonly over the superior convexities, where there is the greatest freedom of movement between the brain and dura.

It is not clear from the data why the subdural neomembranes occurred. They have no demonstrated relevance to cause of death. However, because of the known association between subdural hemorrhage and trauma, the finding of a subdural neomembrane should prompt examination of the eyes for retinal hemosiderin. The conjunction of these two findings would be strongly indicative of prior shaking.

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