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Choroid Plexus Hemorrhage in Premature Neonates:
Recognition by Sonography

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In 34 consecutive infants admitted to the neonatal intensive care unit with birth weight of less than 1,500 g, 80 cranial real-time sonograms were obtained to determine the incidence of choroid plexus hemorrhage. Choroid plexus hemorrhage was diagnosed only in the absence of germinal matrix hemorrhage. Diagnostic criteria included choroid plexus nodularity, enlargement (greater than 12 mm in anteroposterior diameter), or asymmetry between right and left (greater than 5 mm). Ipsilateral intraventricular clots or occipital horn dilatation supported the diagnosis of choroid plexus hemorrhage in most cases. Choroid plexus hemorrhage appeared to be the sole bleeding site in 10 (59%) of the 17 patients with intracranial hemorrhage. Hemorrhage in the region of the caudate nucleus was seen in the other seven cases (41%). Ventricular dilatation and/or intraventricular hemorrhage accompanied nine (90%) of the 10 cases of choroid plexus hemorrhage. This study suggests that in very low-birth-weight premature neonates, the choroid plexus may be a more frequent site of intracranial hemorrhage than previously believed.

An upsurge of interest in neonatal intracranial hemorrhage has occurred within recent years due to technical advances that have permitted noninvasive recognition and more effective management of the problem. Sonography has contributed greatly; its advantages over computed tomography (CT) have been reviewed elsewhere [1–3].

Although events at the arteriolar and capillary levels seem to be responsible for most neonatal germinal matrix hemorrhages, elevated venous pressure from asphyxial or mechanical factors may also result in hemorrhage [4]. The veins of the germinal matrix and the choroid plexus are components of the deep galenic system that participate in the vascular confluence near the foramen of Monro forming the internal cerebral vein. One would suspect that factors that contribute to germinal matrix hemorrhage might similarly affect the choroid plexus [5]. High fibrinolytic activity, which has been detected in the germinal matrix and is implicated as a cause of subependymal hemorrhage extension, has also been noted in the choroid plexus [6]. Despite pathophysiologic similarities to germinal matrix hemorrhage, choroid plexus hemorrhage has been reported as uncommon, in 3%–7% of cases of intracranial hemorrhage [7–9]. Other reports indicate that choroid plexus hemorrhage more often affects full-term infants, especially those who experience birth trauma [2, 4, 10].

Some authors, however, report a higher incidence of choroid plexus hemorrhage. Craig [11] detected choroid plexus bleeding in 18 (82%) of 22 cases of intraventricular hemorrhage and Hemsath [12] in six (30%) of 20 cases at autopsy. In both of these reports, most infants were premature.

These discrepancies in incidence of choroid plexus hemorrhage reflect the difficulties in diagnosing choroidal pathology, especially in the presence of intraventricular hemorrhage. In this paper, we provide additional data on the incidence of choroid plexus hemorrhage in premature infants and discuss its sonographic recognition.

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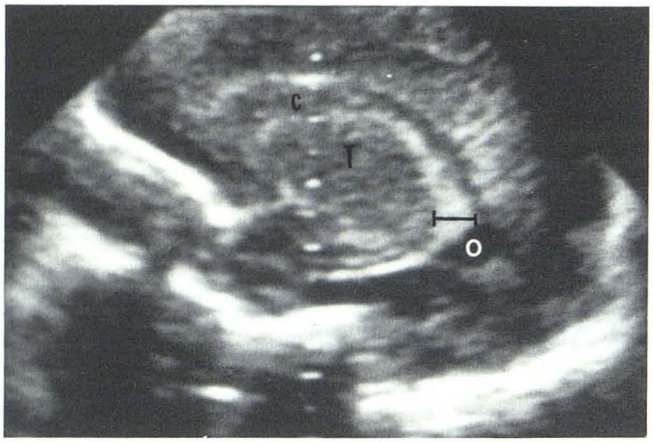

Fig. 1.—Normal choroid plexus measured on parasagittal scan. Normal caudate nucleus (C) and thalamus (T). Occipital horn of lateral ventricle (O).

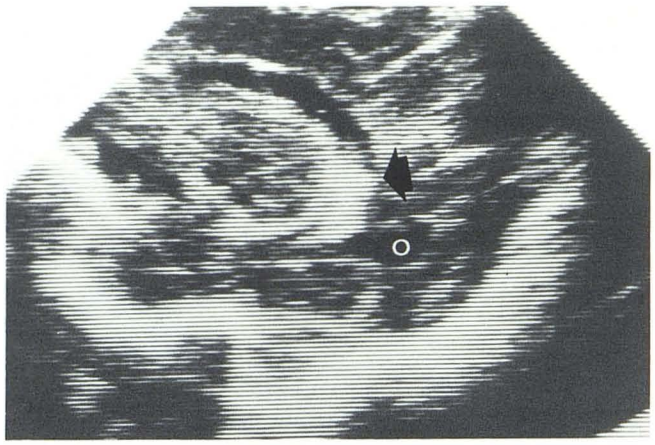

Fig. 2.—Parasagittal scan of choroid plexus hemorrhage in premature neonate (gestational age 28 weeks) confirmed at autopsy. Irregular enlargement of choroid plexus (*arrow*) accompanied by lateral ventricular dilatation. Occipital horn (O).

Subjects and Methods

We performed 80 cribside intracranial sonographic examinations on 34 premature neonates admitted consecutively to the University of Florida Shands Teaching Hospital neonatal intensive care unit. During this period, all infants weighing less than or equal to 1,500 g at birth who survived longer than 24 hr received sonographic evaluation. A portable real-time sectoral scanner with a 5 MHz transducer was used (Advanced Technology Labs., Bellevue, WA). The examinations were videotaped and hard-copy photographs were obtained during subsequent review of the tapes. Of the 34 neonates, 17 were also evaluated by CT.

Choroid plexus hemorrhage was diagnosed sonographically when the structure appeared enlarged, asymmetric, or irregular in contour. The presence of ipsilateral occipital horn dilatation or intraventricular blood without other source of pathology further strengthened the diagnosis. In determining the incidence of choroid plexus hemorrhage, we eliminated those cases in which germinal matrix hemorrhage was evident, as intraventricular hemorrhage from a source other than the choroid plexus can produce choroidal irregularity due to adherent clot.

To establish quantitative indices of choroidal pathology, we reviewed our prior experience in neonatal intracranial sonography and obtained measurements of the choroid plexus in both normal and abnormal neonates (fig. 1). In 53 newborns, no intracranial pathology was demonstrated and the choroid plexus appeared smooth in contour. The largest choroid plexus, chosen from the larger of the two sides and in many cases from serial examinations, averaged 7.9 ± 1.6 mm (range, 5–12 mm). In 31 neonates with irregularity or nodularity of the choroid plexus contour accompanied by ipsilateral occipital horn dilatation, the largest average anterioposterior diameter of the glomus was 12.2 ± 2.7 mm (range, 8–19 mm). The difference between these two means proved to be statistically significant by T test with $t = -8.139$ at 41° of freedom (p value < 0.01). Thus, in our current series, we considered a choroid plexus diameter greater than 12 mm suggestive of abnormality.

In addition to these absolute measurements, asymmetry between the right and left choroid plexus was used as a guide to pathology. To establish a normal range of right-to-left variation in choroidal diameter, we reviewed 225 examinations. In 193 cases, the choroid plexus appeared bilaterally smooth in contour with a mean side-to-side difference of 1 mm (range, 0–5 mm). In 32 neonates with unilateral irregularity, the abnormal choroid plexus measured an average of 3.4 mm greater than on the contralateral side (range, 0–13 mm). Choroid plexus asymmetry greater than 5 mm was considered abnormal in our study.

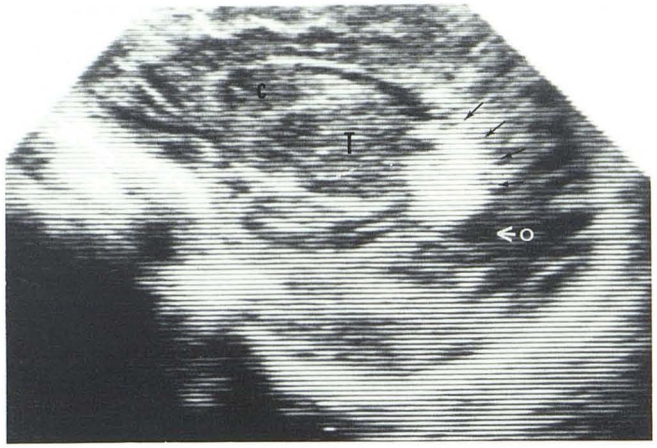

Fig. 3.—Parasagittal scan of choroid plexus hemorrhage in neonate confirmed at autopsy. Irregular enlargement of choroid plexus (*arrows*). Normal caudate nucleus (C) and thalamus (T). Occipital horn (O).

Results

Sonographic evidence of intracranial hemorrhage was observed in 17 of the 34 consecutively admitted low-birth-weight neonates. In the absence of germinal matrix hemorrhage, the choroid plexus appeared unilaterally enlarged or nodular in five neonates and bilaterally abnormal in five. Thus, choroid plexus hemorrhage was diagnosed in 10 of the 17 newborns with intracranial hemorrhage (figs. 2 and 3). Intraventricular hemorrhage and/or ventricular dilatation were present in nine of these 10 newborns. Choroid plexus hemorrhage was accompanied by intraventricular hemorrhage alone in two cases and by ventriculomegaly alone in four cases. Both ventricular dilatation and intraventricular hemorrhage developed in three of the 10 neonates.

Seven (41%) of the 17 infants with intracranial hemorrhage had hemorrhage near the caudate nucleus in the germinal matrix. Five of the seven also had choroidal irregularity, but the possibility of adherent intraventricular clot originating from germinal matrix bleeding precluded the definitive diagnosis of choroid plexus bleeding in these cases. In six of the seven infants with germinal matrix hemorrhage (four with choroid plexus irregularity and two without), intraventricular clots were noted. All seven infants with germinal matrix hemorrhage had mild to moderate ventricular dilatation. In four patients of the total population of 34 newborns, ventricular dilatation occurred in the absence of recognizable intracranial hemorrhage.

Serial sonographic examinations were available in six of the nine newborns with choroid plexus hemorrhage accompanied by intraventricular hemorrhage and/or ventriculomegaly, three of the seven newborns with germinal matrix hemorrhage, one of the four neonates with ventricular dilatation without evidence of hemorrhage, and eight of the 13 normal newborns.

Seventeen of the 34 neonates had CT evaluation. Five cases of intraventricular hemorrhage diagnosed by sonography were confirmed by CT. In three of these neonates, sonography demonstrated germinal matrix hemorrhage as the probable source for the intraventricular blood; CT confirmed hemorrhage in the region of the head of the caudate nucleus in one of these cases and could not identify the source of the intraventricular hemorrhage in the two other newborns. In the other two intraventricular hemorrhage cases, sonography suggested choroid plexus hemorrhage as the source of the intraventricular blood; in one of these cases, CT revealed blood in the region of the velum interpositum, and, in the other case, the source of intraventricular blood could not be determined. Normal sonograms in six neonates were accompanied by normal CT examinations. A discrepancy in results occurred in three cases: in two newborns, sonography revealed mild occipital horn enlargement and the CT scans were interpreted as normal, and, in one case, sonography revealed nodular enlargement of the choroid plexus with mild ventriculomegaly whereas CT revealed a small amount of blood in the occipital horns of the lateral ventricles but was otherwise normal. In the other three cases, an excessive time interval between CT and sonography prevented reliable comparison.

Autopsies were performed on three newborns from this series who died during the neonatal period. In one case, sonography revealed an abnormal choroid plexus accompanied by intraventricular hemorrhage. At autopsy, hemorrhage involved the margin of the choroid plexus anterior to the glomus, with extension into the brain parenchyma near the anterior choroidal artery. Intraventricular blood and an acute subependymal hemorrhage were also evident. In the two other cases, the sonographic diagnosis of subependymal hemorrhage was confirmed at autopsy.

Discussion

On the basis of the sonographic criteria we selected, the choroid plexus represented a frequent site of intracranial hemorrhage in our series of premature neonates. Although this finding agrees with certain autopsy studies concerning choroidal hemorrhage in premature newborns [11, 12], more recent reports have suggested that choroidal plexus hemorrhage rarely occurs as a complication of premature birth [2, 4, 7–10].

It remains difficult, if not impossible, to diagnose hemorrhage into the choroid plexus in the presence of concomitant germinal matrix hemorrhage in a living infant. Frequently, intraventricular blood originating from the subependymal germinal matrix will adhere to the choroid plexus giving the false impression of intrinsic choroidal enlargement or nodularity [13]. Flodmark et al. [8] could not distinguish choroid plexus hemorrhage from subependymal blood on CT; Pape and Wigglesworth [10] stated that choroid plexus hemorrhage may represent a pathologic diagnosis of exclusion. Before our study, we confirmed the sonographic diagnosis of choroid plexus hemorrhage at autopsy in three neonates. In two other newborns with germinal matrix hemorrhage and choroidal irregularity on sonography, autopsy revealed an intrinsically normal choroid plexus encased by intraventricular clot. Therefore, the sonographic identification of choroidal hemorrhage should be reserved only for those cases in which no other explanation for intraventricular hemorrhage can be found.

Even when no associated germinal matrix hemorrhage is detected, care must be taken in assessing choroidal size and nodularity. When the trigone of the lateral ventricle is narrow, echoes from parenchyma or sulci may be superimposed upon those of the choroid plexus giving a "false-positive" nodularity. Real-time sonographic examination is particularly useful in tracing the choroidal contour in both coronal and parasagittal planes and in imaging it free of periventricular cortex. Transient enlargement of the choroid plexus may occur due to venous congestion without hemorrhage [2, 7] and, therefore, in borderline cases, follow-up examination proves valuable. Conceivably, a microscopic germinal matrix hemorrhage, not apparent on sonograms, could account for intraventricular hemorrhage and resultant irregular enlargement of the choroid plexus glomus. Some authors have suggested that the observation of choroidal pulsations may separate choroidal echoes from those of intraventricular hemorrhage [14]. We have found pulsations in this region to be highly variable, independent of pathology.

To determine the accuracy of sonography in the diagnosis of choroid plexus hemorrhage, further pathologic sonographic correlation is required. The neurologic sequelae in survivors of choroid plexus hemorrhage also require evaluation. Although ventricular dilatation frequently accompanied choroid plexus hemorrhage, both in our series and in autopsy studies [5, 11], the clinical significance of this form of intracranial hemorrhage awaits further investigation.

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