Quantitative analysis of cerebrospinal fluid spaces in children with occipital plagiocephaly

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The etiology of occipital plagiocephaly (OP) is not fully understood. The authors have observed that many infants with this condition have external hydrocephalus. This study was undertaken to quantify cerebrospinal fluid (CSF) space caliber in children with OP and to compare these measurements to those derived from normal age-matched controls to further elucidate the pathogenesis of this condition.

Thirty-one infants with isolated unilateral OP (mean age 6 months) were studied. Infants with multiple cranial suture abnormalities, impaired neurological function, developmental delay, and associated craniofacial anomalies were excluded. Twenty normal infants were evaluated as controls. The volumes of the sylvian fissures, frontal and occipital subarachnoid spaces, as well as the cross-sectional areas of the suprasellar and perimesencephalic cisterns, were calculated from computerized tomography (CT) studies. Ventricular size was also assessed.

Generalized subarachnoid space dilation was observed in 29 (93.5%) of the 31 children with OP. Head circumference was significantly greater in the case group (71.4 vs. 50.8 percentile; p = 0.0002 by analysis of variance). The sylvian fissure volume was significantly larger in the case group (5.8 ml vs. 0.7 ml in controls, p < 0.0001). The volume of the contralateral sylvian fissure was greater than that ipsilateral to the side of OP (7.1 ml vs. 4.5 ml, p = 0.001). Frontal subarachnoid space volume was greater in infants with OP (27.5 ml vs. 0.6 ml in controls, p < 0.0001). Both the suprasellar and perimesencephalic cisterns were of greater caliber in the case group (p = 0.007 and p < 0.0001, respectively). No difference in ventricular size or occipital subarachnoid space volume was noted between groups.

The extraventricular CSF spaces in neurologically unimpaired infants with OP are significantly larger than those in age- and sex-matched controls. Enlarged subarachnoid spaces may increase the compliance and malleability of the calvaria and sutures, predisposing to positional deformity. External hydrocephalus may be a fundamental etiological factor in OP.

KEY WORDS • craniosynostosis • external hydrocephalus • lambdoid suture • occipital plagiocephaly • positional molding • children

NONSYNDROMIC craniosynostosis is a relatively uncommon clinical entity, encountered in approximately one per 1000 live births. Isolated unilateral involvement of the lambdoid suture comprises 1% to 15% of all cases of craniosynostosis in published series. The incidence of infantile occipital calvarial flattening appears to have increased in recent years, a phenomenon that is coincident with the American Academy of Pediatrics’ recommendation advocating the “non-prone” (that is, supine) position for healthy infants during sleep as a preventive measure against sudden infant death syndrome. This apparent increase in incidence may be partially artifactual as well, the result of greater scrutiny of calvarial dysmorphism by primary care physicians.

Lambdoid “synostosis” remains a controversial entity in terms of pathophysiology, clinical diagnosis, and treatment. Posterior calvarial flattening may arise from premature fusion of the lambdoid suture (true synostosis) or may be the consequence of positional molding. Indeed, the two conditions may be indistinguishable by clinical and radiological studies. Morphologically, the hallmark feature of both is the rhomboid calvarial deformity, with occipital flattening, prominence of the forehead, and anteroinferior displacement of the ear, all ipsilateral to the side of lambdoid suture involvement. Classic imaging findings such as sutureal sclerosis, perisutural thinning, and ipsilateral petrous bone deformation may be identified in association with either condition and do not correlate well with lambdoid suture fusion using histological criteria. In fact, Becker and Hinton and Hinton and colleagues confirmed histological bone union in only three of 41 clinically synostotic lambdoid sutures, suggesting that bona fide suture fusion (a requisite finding in all other forms of craniosynostosis) is typically absent in the lambdoid deformity.

Infants with lambdoid “synostosis” often benefit from conservative treatment measures such as positional modification, helmet use, or cranial banding; some will improve with no treatment at all. True synostosis of any suture does not respond to such intervention; in fact, the resultant calvarial deformity is exacerbated by the passage of time. Furthermore, the morphology of the involved lambdoid suture is unlike that of other synostotic sutures, displaying fibrous thickening and an inward prominence.
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along the inner calvarial table. Clearly, the affected lambdoid suture is structurally and functionally abnormal but in most cases is not truly synostotic. Consequently, we have adopted a more generic term, occipital plagiocephaly (OP), to denote the general condition of posterior calvarial flattening in the infant.

The pathogenesis of OP is poorly understood. We have observed that many neurologically unimpaired infants with this condition also have generalized dilation of the subarachnoid spaces. The significance of this finding is unknown. This investigation was undertaken to quantify the caliber of cerebrospinal fluid (CSF) spaces in children with OP and to compare these measurements with those derived from normal age-matched controls. We hypothesized that the subarachnoid spaces would be larger in infants with OP than in normal controls and that this excess of extraaxial fluid could contribute to enhanced calvarial malleability, thereby predisposing the calvarium to lambdoid positional molding.

Clinical Material and Methods

Patient Data

Clinical records and computerized tomography (CT) scans obtained in 31 infants treated for nonsyndromic unilateral OP at our institution between January 1990 and January 1995 were retrospectively reviewed. Children with multiple cranial suture abnormalities, impaired neurological function, developmental delay, concurrent craniofacial anomalies, or incomplete radiographic data were excluded from the analysis. Twenty age- and gender-matched control infants without intracranial and calvarial pathology were selected at random for comparison with the case group. For all subjects, head circumference, length, and weight (all parameters denoted as percentiles with reference to national normative data) were recorded at the time of CT scanning.

Imaging Regions of Interest

Computerized tomography studies from the case and control groups were analyzed to determine the caliber of the major subarachnoid spaces. Volumetric measurements were derived bilaterally from the sylvian fissures and from the frontal and occipital subarachnoid spaces. For each of these regions of interest, the cross-sectional area was determined on each image slice and multiplied by slice thickness to yield the slice volume (in cubic centimeters) for each region in a given image. Slice volumes were calculated for each image panel in which the region of interest appeared; the sum of the slice volumes determined the total volume for each region of interest. The relationship of the subarachnoid space to the side of occipital flattening (ipsilateral or contralateral) was recorded in the case group; for the control group, right and left sides were noted. The cross-sectional areas of the perimesencephalic and suprasellar cisterns were calculated at the level of the interpeduncular fossa and optic chiasm, respectively. The irregular geometry of the perimesencephalic and suprasellar cisterns and the slice thickness of the CT data rendered an accurate volume estimation of these regions impossible. Thus, cross-sectional areas were determined at readily defined anatomical landmarks to facilitate size comparisons between subjects. Relative lateral ventricular size was estimated by the Evans ratio (the ratio of the maximum distance between the lateral boundaries of the right and left frontal horns at the level of the caudate head and the maximum distance between the right and left inner calvarial tables).

Measurement Methods

All measurements were made using calipers calibrated to the nearest 0.1 mm. Data were collected by a single observer in blinded fashion; the observer was presented with CT scans in random order with the identity, age, and gender of the subject concealed. All scans were recycled, again at random, until each had been measured in triplicate. The three numerical values derived from each region of interest were averaged in an effort to minimize the influence of intraobserver variability.

Statistical Analysis

Individual subject data generated from each region of interest were compiled into case and control groups; means and standard deviations were calculated for the frontal and occipital subarachnoid spaces, sylvian fissures, perimesencephalic cistern, and suprasellar cistern. The composite data from each region of interest were compared between the case and control groups using a two-tailed analysis of variance test. The threshold of statistical significance was placed at p = 0.05.

Results

Demographic and Clinical Data

Patient data are summarized in Table 1. The mean age of the case group was 6.0 ± 3.1 months (mean ± standard deviation (SD)); control group age was 6.4 ± 3.8 months (p > 0.05). The gender ratio was approximately 2:1 (male/female) in both groups. The mean head circumference of the case group was significantly greater than that of the control group (71.4 ± 11.6 percentile vs. 50.8 ± 7.3 percentile, p = 0.0002). Length and weight measurements
The CSF space caliber in 31 infants with functional lambdoid craniosynostosis and 20 age- and sex-matched controls*

<table>
<thead>
<tr>
<th>CSF Space</th>
<th>Group (mean ± SD)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>sylvian fissure (ml)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case</td>
<td>4.5 ± 2.4 (ipsilat)</td>
<td>0.7 ± 0.4</td>
</tr>
<tr>
<td>Control</td>
<td>7.3 ± 3.8 (contralat)</td>
<td>0 ± 0.1</td>
</tr>
<tr>
<td>frontal SA space (ml)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case</td>
<td>30.1 ± 14.8 (ipsilat)</td>
<td>0.6 ± 1.4</td>
</tr>
<tr>
<td>Control</td>
<td>25.7 ± 12.2 (contralat)</td>
<td>0 ± 0.1</td>
</tr>
<tr>
<td>occipital SA space (ml)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case</td>
<td>0.3 ± 0.3 (ipsilat)</td>
<td>0.5 ± 0.4</td>
</tr>
<tr>
<td>Control</td>
<td>0.6 ± 0.3 (contralat)</td>
<td>0 ± 0.1</td>
</tr>
<tr>
<td>perimesencephalic cistern (cm²)</td>
<td>2.1 ± 1.2</td>
<td>0.9 ± 0.4</td>
</tr>
<tr>
<td>suprasellar cistern (cm²)</td>
<td>4.7 ± 3.6</td>
<td>2.4 ± 0.6</td>
</tr>
<tr>
<td>lateral ventricles (Evans ratio)</td>
<td>0.29 ± 0.06</td>
<td>0.26 ± 0.03</td>
</tr>
</tbody>
</table>

* Contralat = contralateral to side of occipital flattening; CSF = cerebrospinal fluid; ipsilat = ipsilateral to side of occipital flattening; NS = not significant (p > 0.05); SA = subarachnoid; SD = standard deviation.

were not significantly different between populations (p > 0.05). Right-sided OP was noted in 21 (67.7%) of the 31 children in the case group. All infants were neurologically unimpaired and had achieved appropriate developmental milestones without delay.

Comparison of CSF Space Caliber

A comparison of CSF space caliber between groups is presented in Table 2. The mean sylvian fissure volume was significantly larger in the case than in the control group (5.9 ± 3.1 ml vs. 0.7 ± 0.4 ml, p < 0.0001). Within the case group, the volume of the sylvian fissure contralateral to the side of flattening was greater than the ipsilateral counterpart (7.3 ± 3.8 ml vs. 4.5 ± 2.4 ml, p = 0.001) (Fig. 1). Frontal subarachnoid space volume also was greater in the case group (27.9 ± 13.5 ml vs. 0.6 ± 1.4 ml in controls, p < 0.0001) (Fig. 2). No difference in frontal subarachnoid space caliber was observed within the case group with respect to the side of flattening (p > 0.05). The occipital subarachnoid space volume was minimal in both the case and control groups (p > 0.05). The cross-sectional area of the perimesencephalic cistern at the level of the interpeduncular fossa was significantly greater in the case group (2.1 ± 1.2 cm² vs. 0.9 ± 0.4 cm² in controls, p < 0.0001) (Fig. 3). Similarly, the case group exhibited a larger suprasellar cistern at the level of the optic chiasm (Fig. 4) than the control group (4.7 ± 3.6 cm² vs. 2.4 ± 0.6 cm², p = 0.007). Relative ventricular size was similar in both groups (Evans ratio 0.29 ± 0.06 vs. 0.26 ± 0.03, p > 0.05).

Standard errors of the means for individual calculations of volume (for the sylvian fissures and frontal/occipital subarachnoid spaces) and cross-sectional areas (for the perimesencephalic and suprasellar cisterns) were < 0.1 ml and < 0.08 cm², respectively.

Discussion

This investigation clearly demonstrates the generalized enlargement of the extraventricular CSF spaces in neurologically and developmentally normal children with OP, as compared with those of age- and sex-matched controls. The pattern of subarachnoid space dilation is remarkably consistent: 1) the sylvian fissures are enlarged bilaterally, with the contralateral fissure of greater magnitude than that ipsilateral to the side of occipital flattening; 2) bilateral frontal subarachnoid space dilation is evident and is not correlated with the side of plagiocephaly; and 3) the suprasellar and perimesencephalic cisterns are both enlarged. Ventricular size remains similar to that of control subjects, although a mild degree of lateral ventricular dysmorphism may be observed accompanying the calvarial deformity. Typically, the occipital horn and atrium of the lateral ventricle ipsilateral to the side of plagiocephaly are compressed; the relatively larger contralateral ventricle retains a more normal contour (Fig. 5).

The demographic and clinical characteristics of the present case group are similar to those reported by previous investigators.1,28,34 Males were affected slightly more than females as frequently as females (21:10), a distribution which seems representative of OP as well as many forms of bone fide craniosynostosis, particularly that involving the sagittal suture.19,23,24 The marked predilection for right lambdoid suture involvement, reported in most previous series, was also observed in the present study. The mean patient age at the time of initial clinical evaluation and radiographic imaging was 6 months in this series; the age distribution reported herein is similar to that reported elsewhere.11,28,34 Thus, based on clinical criteria, this series reflects the norm thus far established for children with OP.

Normal CSF Spaces in Infants

As a component of their comprehensive assessment of the CSF dynamics and structural alterations accompanying childhood macrocephaly, Gooskens and colleagues4,15 suggested the following guidelines indicative of CSF space enlargement. Ventrilocumegaly was defined by an Evans ratio exceeding 0.35 in children younger than 3 years of age. The subarachnoid space was considered to be dilated when its width surpassed 7.5 mm in children less than 1 year old. Using these criteria, four (12.9%) of the 31 infants with OP in the present series exhibited ventricular enlargement; none of the normal control population had ventriculomegaly. Subarachnoid space enlargement was present in 29 (93.5%) of 31 of those with OP, whereas no child in the control group exhibited such findings. Of note, the two children with OP who did not have generalized subarachnoid space dilation were the two oldest children in the case population; both were 11 months old at the time of CT scanning.

Some caution must be exercised when interpreting subarachnoid space caliber in the infant. The literature fails to provide definitive values for the absolute and/or relative sizes of these anatomical regions in the normal child. This concern is compounded by the fact that ventricular size and subarachnoid space caliber are dynamic within the first years of life. This emphasizes the need for an age-matched control population to which the case group may be compared, as was incorporated in the present study. Previous studies of CSF space anomalies associated with craniosynostosis have not examined a cohort population of normal controls using either clinical or radiographic criteria.7,20

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Subarachnoid Space Anomalies in Craniosynostosis

Other investigators have noted subarachnoid space abnormalities in association with various forms of craniosynostosis. Carmel, et al., as part of their CT evaluation of skull base and calvarial deformities in craniosynostosis, commented briefly on CSF space abnormalities identified in four patients with unilateral lambdoid synostosis. They noted modest distortion of the lateral ventricle (occipital horn) as well as compression of the posterior fossa and occipital subarachnoid spaces, all ipsilateral to the side of flattening. Although not specifically mentioned in the text, CT data from a representative case also demonstrated generalized dilation of the subarachnoid spaces that was most pronounced over the frontal lobes. These authors postulated that CSF pathway obliteration at the site of suture fusion promotes redistribution of fluid to regions of reduced pressure gradient between the brain and skull. They believed that CSF space distortion may be responsible, at least in part, for intracranial hypertension associated with single-suture synostosis.

Muakkassa, et al., published a CT image obtained in a child with lambdoid synostosis that demonstrated enlarged frontal subarachnoid spaces and distortion of the lateral ventricles but did not elaborate on these findings.

Chadduck, et al., have contributed the most comprehensive assessment of subarachnoid space abnormalities associated with craniosynostosis to date. These investigators reviewed CT and magnetic resonance imaging data from 75 patients treated for various forms of craniosynostosis. Eleven of these children presented with lambdoid suture abnormalities. Five had “radiographic evidence of suture fusion;” the remaining six had “positional deformities.” Four of those with apparent suture fusion demonstrated subarachnoid space dilation only over the frontal lobe ipsilateral to the side of deformity. In contrast, the majority of those with positional molding had generalized enlargement of the subarachnoid spaces. Apparently, these infants also had historical evidence of cerebral insults with resultant motor and developmental delay antedating their calvarial abnormalities. These investigators speculated that generalized subarachnoid space dilation was indicative of cerebral damage and brain atrophy and that positional lambdoid deformities may be the consequence of motor delay. The clinical and imaging data derived from the present series seem to contradict this conclusion, because generalized subarachnoid space enlargement is apparent in children with unremarkable perinatal histories and normal psychomotor development who nevertheless develop OP.

Pathophysiology of Subarachnoid Space Dilation in Craniosynostosis

The pathophysiology underlying subarachnoid space dilation...
dilation in association with craniosynostosis remains obscure. Hassler and Zentner, who noted dilatation of the bifrontal and interhemispheric subarachnoid spaces in 70% of their patients with sagittal synostosis, suggested that bone compression of the superior sagittal sinus and adjacent pacchionian granulations induced a state of chronic CSF malabsorption with consequent subarachnoid space enlargement. Subsequent investigators have criticized this assumption, because similar dilation of the subarachnoid spaces has been identified in other forms of craniosynostosis not involving the sagittal suture, in which the superior sagittal sinus and/or pacchionian granulations would not be affected. Certainly, it is difficult to explain subarachnoid space enlargement in OP using this theory. Chadduck, et al., suggested that transient elevations in CSF pressure induced by sutural synostosis may be responsible for the preferential redistribution of fluid into more expandable skull compartments. These regions, they postulated, would then be prone to compensatory calvarial growth as a consequence of augmented brain pulsations through the enlarged fluid-filled spaces. Thus, in the case of lambdoid deformity, the frontal subarachnoid space ipsilateral to the side of plagiocephaly would be dilated preferentially, thereby inducing compensatory bone expansion exclusively overlying this region. However, it is apparent from the present study that the frontal subarachnoid spaces are bilaterally dilated in OP. There is no significant difference in the caliber of the frontal subarachnoid spaces with respect to the side of lambdoid deformity. Therefore, it is difficult to explain isolated compensatory ipsilateral frontal bone expansion on the basis of subarachnoid space dilatation alone. Furthermore, the enlargement of both sylvian fissures and the basal cisterns is not explained on the basis of preferential CSF distribution within the affected hemisphere. Clearly, subarachnoid space dilatation occurs bilaterally in OP and involves the basal CSF cisterns as well as the convexities.

The aforementioned theories feature a common fundamental assumption: the bone deformity (suture fusion) is the primary abnormality and is responsible for inducing secondary alterations in the subarachnoid spaces. We offer an alternative explanation. It must be reemphasized, however, that OP is an entity dissimilar to the true synostoses of other cranial sutures in many respects. Thus, because the pathology in OP is distinct from the other craniosynostoses, the pathophysiology underlying the condition is also likely to be different.

We postulate that generalized subarachnoid space dilatation occurs as the primary anomaly in OP, a predisposing condition that permits subsequent calvarial deformity. The enlarged fluid-filled posterior subarachnoid spaces possess a compliance to mechanical deformity that far exceeds that of underlying cerebral tissue. Chronic pressure exerted by the child’s position, coupled with the enhanced compliance of the enlarged subarachnoid spaces, permits progressive flattening and infolding of the posterior parietal and occipital bones. Because the patent lambdoid suture is relatively pliant and yielding to mechanical deformation, parietooccipital flattening is most pronounced in this region. Over time, the posterior subarachnoid spaces are obliterated by the progressive plagiocephalic deformity. Secondary alterations in suture morphology occur as a consequence of chronic pressure, manifesting as fibrous thickening and an inward-projecting prominence along the inner calvarial table.

As the deformity progresses, reactive suture changes and further infolding of the occipital and posterior parietal bones exert mass effect against the ipsilateral cerebral hemisphere. This is evidenced by compression and distortion of the lateral ventricle and sylvian fissure ipsilateral to the side of plagiocephaly. Recall that the ipsilateral sylvian fissure, although dilated in comparison to normal controls, is significantly smaller in caliber than its contralateral counterpart. If the subarachnoid space abnormalities in OP were secondary to CSF redistribution as suggested by others, the ipsilateral sylvian fissure should be enlarged relative to the contralateral side, a conclusion that is clearly not supported by the present data. Chronic compression of the cerebral hemisphere from the parietooccipital plagiocephaly induces frontal and temporal bone remodeling ipsilateral to the side of lambdoid deformity, in much the same manner that discrete unilateral mass lesions such as intraaxial tumors may actuate asymmetrical skull expansion in the neonate and infant. This creates the unilateral frontal bossing, anteroinferior auricular displacement, and rhomboid calvarial deformity characteristic of OP.
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Fig. 5. Unenhanced axial computerized tomography scan demonstrating compression of the lateral ventricle ipsilateral to the side of lambdoid deformity. The contralateral ventricle retains a more normal contour.

Etiology of Subarachnoid Space Dilation in OP

The etiology of generalized subarachnoid space dilation in children who develop OP remains speculative. However, it is apparent from the present investigation that the subarachnoid space enlargement observed in these infants does not represent diffuse brain atrophy or global cerebral dysfunction. Although conclusive data are lacking, circumstantial evidence suggests an association with idiopathic external hydrocephalus, benign enlargement of the subarachnoid spaces, benign extracerebral fluid collections, and benign communicating hydrocephalus of infancy). Children with idiopathic external hydrocephalus exhibit demographic characteristics that are similar to those reported for OP in this and other series. In both conditions, males are affected approximately twice as frequently as females. Enlargement of the head circumference, a prerequisite for diagnosis in idiopathic external hydrocephalus, is often encountered in OP as well. The neurological examination is typically unremarkable in children with either disorder, and the vast majority have normal prenatal, perinatal, and developmental histories. Both conditions present within the 1st year of life, with peak incidence generally occurring within the first 3 to 6 months.

The pattern of CSF space dilation is also similar in idiopathic external hydrocephalus and OP. Generalized subarachnoid space enlargement is characteristic of both conditions, involving the convexities and sylvian fissures bilaterally, as well as the basal CSF cisterns. Ventricular size is frequently normal, although mild enlargement of the lateral ventricles may be manifest on occasion. Finally, the natural history of the CSF space aberrations in both conditions is similar. Without surgical treatment, the subarachnoid space anomalies appear to be self-limited, with spontaneous resolution typically occurring in early childhood.

Although the association between OP and external hydrocephalus is provocative, a causal relationship remains speculative. Subarachnoid space enlargement may, as we propose, be a primary anomaly that precedes and facilitates the position-induced calvarial deformity. Conversely, however, primary changes in the calvaria (particularly those involving the skull base) induced by chronic pressure and an undefined permissive factor may alter CSF flow dynamics, yielding secondary permutations in subarachnoid space morphology. Finally, subarachnoid space dilation may be purely an epiphenomenon that does not contribute to the pathogenesis of progressive occipital deformity at all.

Ideally, to elucidate more precisely the nature of the association between external hydrocephalus and OP, infants with the former would be identified within the first few months of life and thereafter be followed prospectively for the development of calvarial deformity. Identification of such a cohort would be difficult, however, because neurologically and developmentally normal infants rarely undergo CT and magnetic resonance studies early in the postnatal period and are even less frequently referred for neurosurgical evaluation. In contrast, infants with abnormal head configurations often undergo diagnostic imaging and enter the province of the neurosurgeon. Thus, whereas it is apparent that most infants with OP have enlarged basal and convexity subarachnoid spaces at the time of diagnosis, the prevalence of calvarial deformity in children with external hydrocephalus is unknown.

Conclusions

This investigation represents the largest single series to date that analyzes the CSF spaces in OP. The following conclusions are suggested by the data.

1) Generalized subarachnoid space enlargement is associated with OP in the vast majority of cases and is not correlated with diffuse cerebral injury, neurological dysfunction, or developmental delay.

2) Ventriculomegaly is encountered only infrequently in children with OP.

3) Children with OP have larger head circumferences than do normal controls.

4) The pattern of subarachnoid space dilation in OP is predictable and involves both frontal regions, sylvian fissures (contralateral less often than ipsilateral), and basal cisterns.

5) External hydrocephalus may be a fundamental etiological factor in the development of OP.

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