

Bifrontal Subdural Effusion Crossing the Midline in Semilobar Holoprosencephaly

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Background: Holoprosencephaly (HPE) is a congenital forebrain cleavage disorder that often results in absence or hypoplasia of midline structures, including the falx cerebri. Subdural effusions typically remain confined to one hemisphere, as the falx acts as a barrier to its contiguous spread. We present a pediatric semilobar HPE case with an anterior falcine defect, leading to a bifrontal subdural effusion that crossed the midline.

Case Details: A 6-month-old female with Trisomy 13 and semilobar HPE (fused frontal lobes, single ventricular cavity, absent anterior falx) developed signs of increased intracranial pressure. MRI revealed a large subdural collection over both frontal convexities, crossing the midline in the region of falx agenesis. Posteriorly, where a partial falx was present, the fluid was compartmentalized. No hemorrhage was evident. This case confirms that an absent falx cerebri permits the communication of subdural space across the midline. We suspect that embryologically, failure of the prosencephalic diverticulation in HPE resulted in fused hemispheres and no interhemispheric fissure, thus no falx to constrain the effusion.

Discussion: For radiologists, recognition of falx agenesis is key to avoiding misdiagnosis, such as mistaking a bilateral subdural for subarachnoid fluid. Awareness of an absent falx is vital for surgical navigation and understanding that bilateral subdurals may represent a single entity in such patients. This rare case broadens our understanding of dural anatomy variations and their clinical consequences. It emphasizes the importance of integrating embryologic knowledge when interpreting neuroimaging in congenital malformations. By delineating how a developmental anomaly (semilobar HPE) can lead to unique pathological findings (midline-crossing subdural effusion), it offers insights into the physiology of dural compartments. Such a finding in the right clinical context can be highly valuable in the diagnosis of HPE. These insights enhance our ability to diagnose and treat neurological complications in the context of congenital brain anomalies, contributing to improved outcomes in this vulnerable patient population.

