Neonatal infarction within basal cerebral vein territory

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In this report, an unusual intracranial haemorrhage in a term male infant born to a mother with diabetes is explained on the basis of occlusion of both basal veins of Rosenthal. This diagnosis relies on anatomical location and iconographic aspect of the clots. Evidence that this vessel was occluded cannot be ascertained from ultrasound or MR angiographic techniques in the neonatal period. The basal vein has not been implicated in previous reports of neonatal brain haemorrhage.

This is an account of a hitherto unreported type of neonatal intracranial haemorrhage. Understanding the pathogenesis depends on exploration of the behaviour of the blood clots.

Case history
The infant was born at 37 weeks’ gestation to an insulin-dependent 41-year-old, gravida 6 para 6, obese, and diabetic mother, who was a heavy smoker. She was treated with methyldopa for hypertension in the third trimester of pregnancy. Her first trimester HbA1c was 9.6%. Two days before delivery the foetal heart rate had been variable without decelerations on routine check-up. On the day of the birth, the mother was admitted with reduced foetal activity and a foetal heart rate with low variability but no decelerations. Labour was induced with amniotomy. For five hours before delivery the foetal heart rate lost all variability. A male weighing 3560 grams (90th centile) was delivered by Caesarean section. His umbilical artery pH was 6.86. He was given Apgar scores of 4, 5, and 7 at 1, 5, and 10 minutes and was ventilated endotracheally after 5 minutes. Clinical features included generalized hypotonia, normal fontanelle tension, and a palpable right kidney. Ventilation was purely supportive. Hypoglycaemia required hypertonic glucose infusion. Clinical seizures presented as apnoic attacks and inappropriate smacking of the lips at 14 hours of age. Biochemical analysis of blood on admission into the unit revealed anaemia (haemoglobin 14.0 g/dL). On day 3, the platelet count dropped to 65,000/mm³, and without preceding administration of coagulation factors the activated partial thromboplastin time was 40.7 seconds (normal range 22 to 45), fibrinogen 443 mg/dL (normal range 180 to 455), prothrombin time was 55% (normal range 15 to 60), protein C activity 15% (normal range 15 to 45), protein S total antigen 39% (normal range 25 to 40), antithrombin activity 33% (normal range >30), and activated protein C resistance 2.88 (normal range >2.15). Lupus anticoagulans was absent.
Four hours after birth, ultrasound revealed extensive bilateral hyperechoic changes in temporal and parieto-occipital but not frontal white matter (Fig. 1). A tongue of hyperechoic tissue extended into the left basal and posterior striatum, sparing the thalamus. There was a moderate amount of intraventricular haemorrhage. Brain MRI on day 4 (Fig. 2) revealed a haemorrhagic lesion involving bilateral occipitotemporal and parietal periventricular white matter, the left striatum, and a caudal portion of the posterior limb of the left internal capsule. The border of the lesion facing parietal white matter was feathered by scattered small haemorrhages. The midline was free from bleeding. Both internal but not the basal veins were seen on magnetic resonance angiogram (MRA). Power and colour Doppler sonography on day 5 enabled us

Figure 1: Four hours after birth, ultrasound revealed extensive bilateral hyperechoic changes in temporal and parieto-occipital white matter, a tongue of hyperechoic tissue (arrows) extended into left basal and posterior striatum, sparing the thalamus.

Figure 2: MRI on day 4 showed a haemorrhagic lesion on both sides, involving left striatum and caudal crus posterius capsulae internae, on T1-weighted images, signal from pallidum and posterior putamen appeared increased in relation to internal capsule and thalamus; borders of lesion facing parietal white matter carried scattered smaller haemorrhages (arrows).
to see the internal cerebral but not the basal veins. Clinical evolution was complicated by prolonged refractory tonic seizures (with epileptic EEG) and posthaemorrhagic hydrocephalus. A definitive ventriculotoarial shunt was inserted on day 47. In the meantime, generalized cystic periventricular leucomalacia, including frontal areas not involved in the primary lesion, had been identified by ultrasound. The right kidney was unique and hypertrophied. On day 60 the infant was discharged with oral feeding. The infant was lethargic with bouts of irritability and tremor. He did not make visual contact but had unconjugated roving-cyc excursions. At 2 years of age he had spastic quadriplegia and learning disability due to white matter injury it was impossible to extract the influence on development of basal vein damage per se.

Discussion

The history of this case study suggested that the brain bleeding detected with ultrasound at 4 hours of age may have been of antepartum origin. Because the extensive lesion did not directly involve subcortex, a subcortical type of arterial infarction (stroke) was excluded (de Vries et al. 1997). Middle cerebral artercy posterior trunical stroke would include (sub)cortex behind the insula and would not reach as far as the ventricle wall. Complete middle cerebral artery infarction would involve frontal and parietal cortex and the entire putaminal area, not just its caudal part. Posterior cerebral artery pial stroke would involve the calcarine cortex and complete infarction within this vessel would cause damage to the thalamus. The feathered appearance of the haemorrhage resembles venous infarction of periventricular white matter in association with subependymal/intraventricular haemorrhage (SEH/IVH) of the preterm infant (Perlman et al. 1993). These are arguments in favour of a venous pattern of injury in this infant.

In the event of internal cerebral vein occlusion, haemorrhage in to the thalamus and/or caudate head is expected. This so-called thalamo-caudate-ventricular bleeding carries a component of IVH due to bleeding into the germinal matrix or choroid plexus secondary to deep venous thrombosis (Govaert et al. 1992). Venous infarction within the area of the thalamostriate vein (the main terminal tributary of the internal cerebral vein) involves periventricular white matter from the caudate area to the level of the upper half of the atrium. The brunt of the injury in this infant was in the parietal and occipitotemporal white matter including a rim of white matter near the ependyma. Lower and caudal putamen were involved on the left. These findings suggest both basal veins were primarily implicated. Bilaterality without midline involvement and left striatal involvement were in accord with this pattern. Absence of basal vein perfusion could not be documented with certainty, neither with colour Doppler imaging nor with MRA, but an inability to demonstrate flow in the basal vein with these techniques may also occur in the normal newborn infant (Taylor 1992). Even in adults 10% of these veins are missed with cerebral MR venography (Ayazncz et al. 2000). Thrombosis was the probable mechanism against a background of diabetes, obesity, and heavy smoking. Currently known mutations with neonatal thrombotic effect were excluded.

The basal cerebral vein of Rosenthal (Fig. 3) lies on the surface of the base of the brain, runs around the brain stem and drains posteriorly into the Galenic system (Newton and Polts 1974). It is divided into three major segments. The first or striate segment originates on the surface of the anterior perforated substance at the union of the anterior cerebral, deep middle cerebral and inferior striate veins. Tributaries of the first segment include the olfactory vein, posterior frontal orbital veins, and branches from the optic chiasm. The second or peduncular segment runs around the basis pedunculi to the plane of the lateral mesencephalic sulcus. Tributaries include the peduncular vein, inferior thalamic veins, inferior ventricular vein, and branches from the optic tract and hypothalamus. The third or mesencephalic segment joins other vessels to form the great cerebral vein of Galen in the quadrigeminal cistern. Tributaries of the third segment are the lateral mesencephalic vein, posterior thalamic veins, and veins from adjacent medial aspects of the temporal and occipital lobes. The basal vein is a secondary vein which develops around the 11th fetal week from the formation of longitudinal anastomoses.

Figure 3: Basal vein anatomy (adapted from Newton and Polts 1974). (I) anterior or striate segment; (II) middle or peduncular segment; (III) posterior or mesencephalic segment. 1, olfactory vein; 2, pericallosal vein; 3, orbitofrontal vein; 4, anterior cerebral vein; 5, peduncular vein; 6, inferior ventricular vein; 7, inferior choroidal vein; 8, anterior longitudinal hippocampal vein; 9, inferior temporoparietal vein; 10, calcarine vein; 11, internal cerebral vein.
between embryonic telencephalic, ventral diencephalic, mesencephalic, metencephalic, and myelencephalic pial veins. Since its formation follows sequential changes involving anastomoses, delections, and reanastomoses, it is not surprising to encounter anatomic variations. In rare cases, tributaries of various segments of the basal vein drain almost independently of each other into various sinuses.

In the event of basal vein thrombosis, haemorrhagic infarction, similar to the case study described here, would be expected. Surprisingly, this venous lesion pattern has not yet been reported in human medicine.

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References


