

Fetal closed head injuries following maternal motor vehicle accident

A clinicopathologic case report

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Abstract

Rationale: The clinicopathologic appearance of fetal closed head injury (FCHI) due to a maternal motor vehicle accident has not been fully investigated because of its extreme rarity.

Patients concern: A 22-year-old woman at 31 weeks of gestation was riding in the front passenger seat of a car, and another rightward-turning car struck the right side of her vehicle.

Diagnosis: Uterine injury with placental abruption was strongly suspected.

Intervention: A live female infant in breech presentation was delivered by emergency caesarean section.

Outcomes: Although the female infant was and showed no evidence of trauma on her body surface. She exhibited a convulsion on the day of birth, and subsequent ultrasonography revealed possible intracranial hemorrhage. Although laboratory parameters associated with circulatory and respiratory function suggested a good response to the intensive care administered during the treatment course, the infant died 6 days later despite intensive care. Autopsy showed severe brain softening, subarachnoid hemorrhage with cerebral and cerebellar contusion, and bilateral thalamic hemorrhage. No hypoxic/ischemic changes of the thoracoabdominal organs were evident at autopsy.

Lessons: This was a clear case of FCHI by both shear and tensile forces. Multiple factors including the structural vulnerability of the fetal brain, the head posture of the fetus, the crash location and direction of force on the vehicle, and the employment of safety equipment may have contributed to the occurrence of FCHI in the present case.

Abbreviations: FCHI = fetal closed head injury, MVA = motor vehicle accident.

Keywords: autopsy, brain, closed head injury, fetus, motor vehicle accident

1. Introduction

Direct fetal injury is infrequent, and head injury is most common in such cases.^[1] A motor vehicle accident (MVA) is the main cause of direct fetal injuries.^[2] Several studies have validated the association of safety devices, such as seat belts and airbags, with maternal traumatic injuries.^[3,4]

Many cases of fetal direct head injury have a poor prognosis; they are often associated with skull fracture and usually occur in the third trimester.^[5,6] Previous reports have indicated that fetal skull fracture frequently occurs when the fetal head is situated low in the pelvis, often in the setting of pelvic fracture.^[1,7,8] Severe maternal injuries can generally predict a fatal outcome of the fetus.^[9] However, it has been accepted that fetal traumatic head injuries infrequently occur in cases of mild maternal injury.^[10,11] In contrast, fetal closed head injury (FCHI) by blunt abdominal trauma in a pregnant woman is considered extremely uncommon, and the neuropathologic appearance and pathogenesis of such cases have not yet been fully explored.

We herein present a considerably rare autopsy case of FCHI that was caused by maternal blunt abdominal trauma associated with an MVA. Informed written consent was obtained from the next kin for publication of this case report and accompanying images. This study was approved by the Ethics Committee of the University of Toyama (#24-1) and was performed according to the recommendations of the Declaration of Helsinki.

2. Case presentation

A 22-year-old woman who was approximately 31 weeks pregnant with her 1st child was transferred to the emergency room. She had been riding in the front passenger seat of a car that was forcefully struck on the right side by a rightward-turning car, causing her car to overturn toward the left. She was wearing a

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seat belt, and the airbag was activated. While being transferred to the hospital, she continuously complained of lower abdominal pain. When she arrived at the emergency department about 35 minutes after the MVA, she was hemodynamically stable with the following vital signs: blood pressure of 103/79, pulse of 85/min, and oxygen saturation of 99% on 100% oxygen. She had no external injuries other than an abrasion in the left iliac region; however, a small amount of genital bleeding was found. The fetal heart rate was 180 beats/min, and sonographic examination revealed a breech presentation, thickening of the placenta, a small amount of ascites, and decreased amniotic fluid. A live female infant was delivered by emergency caesarean section that started 133 minutes after admission; the infant's Apgar score was 2 at 1 minute and 4 at 5 minutes, her body weight was 1512 g, and umbilical blood analysis showed a pH of 7.10 and base deficit of 11.5 mmol/L. At the time of the operation, an incomplete laceration of the uterine myometrium was found at three points: 2 in the anterior region and 1 in the posterior region of the myometrium. All wounds were repaired after delivery of the infant. Bloody ascites of 100 mL was found, and no hematoma was found in the uterine cavity. The operative duration was 49 minutes, and the total blood loss during the operation was 833 mL. The mother showed no clinical complications after caesarean section. The placenta weighed 340 g and measured 14 × 11 × 3.5 cm. No retroplacental hematoma was found, but a subamniotic hematoma that had probably been caused by rupture of chorionic vessels due to traction force from the umbilical cord was evident (Supplemental Fig. 1, <http://links.lww.com/MD/C601>).

The infant showed no anomalies or external trauma, and she was immediately transferred to the neonatal intensive care unit. Although subsequent chest X-ray showed a left diaphragmatic hernia, a significant difference in blood pressure between the upper and lower extremities suggestive of pulmonary hypertension was not evident throughout the infant's course. However, a generalized convulsion occurred about 7 hours after delivery, and ultrasonography a short time after the event clearly showed increased echogenicity in the occipital lobe and central gray matter (Fig. 1A, B). A bulging anterior fontanel followed by the absence of pupil movement and loss of light reflex then occurred 1 day after delivery. However, both her circulatory and respiratory conditions were continuously maintained throughout critical care by medical interventions: the fraction of inspiratory oxygen decreased from 0.75 to 0.25 on the day after delivery, the ventilator index remained at around 0.2, the alveolar-arterial oxygen gradient ranged from 20 to 30 mm Hg, and the partial pressure of carbon dioxide ranged from 20 to 40 mm Hg. Administration of nitric oxide started 1 hour after delivery and was stopped after 70 hours because the infant was becoming stable. However, follow-up ultrasonography showed further increased echogenicity and difficult visualization of the cerebral ventricles. She died 6 days after delivery.

At medicolegal autopsy, no skull fractures were found (Fig. 1C), and the brain weighed 750 g (839 ± 290 g for corresponding gestational age).^[12] The developmental stage of cerebral convolution was consistent with the typical appearance at 25 to 32 weeks of gestation.^[13] Macroscopic investigation

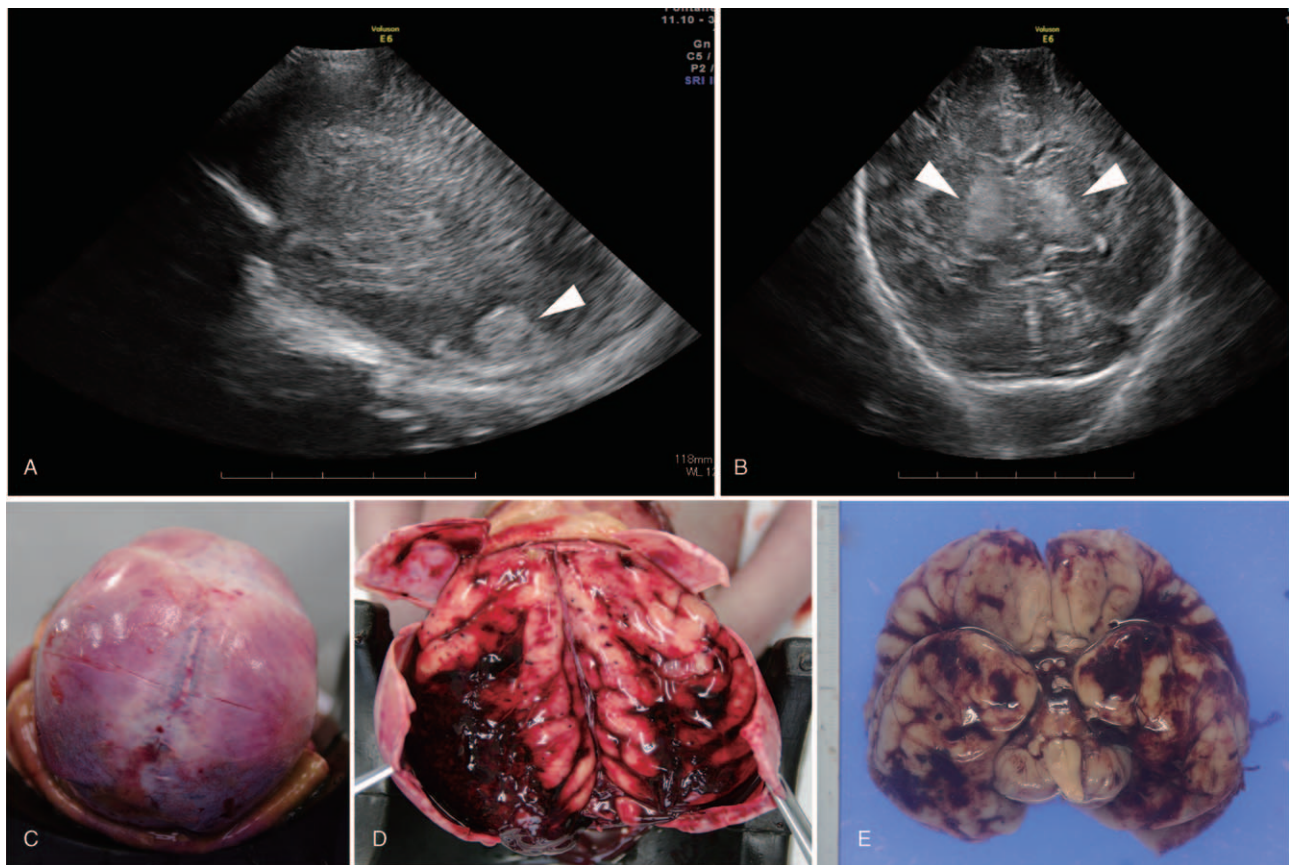


Figure 1. Ultrasonographic findings and gross appearance of the head. On ultrasonographic examination, a highly echogenic area suggestive of hemorrhage was found in (A) the posterior cerebrum and (B) bilateral basal ganglia. (C) Skull without fractures. (D, E) Fresh subarachnoid hemorrhage was seen in both the convexity and basal side.

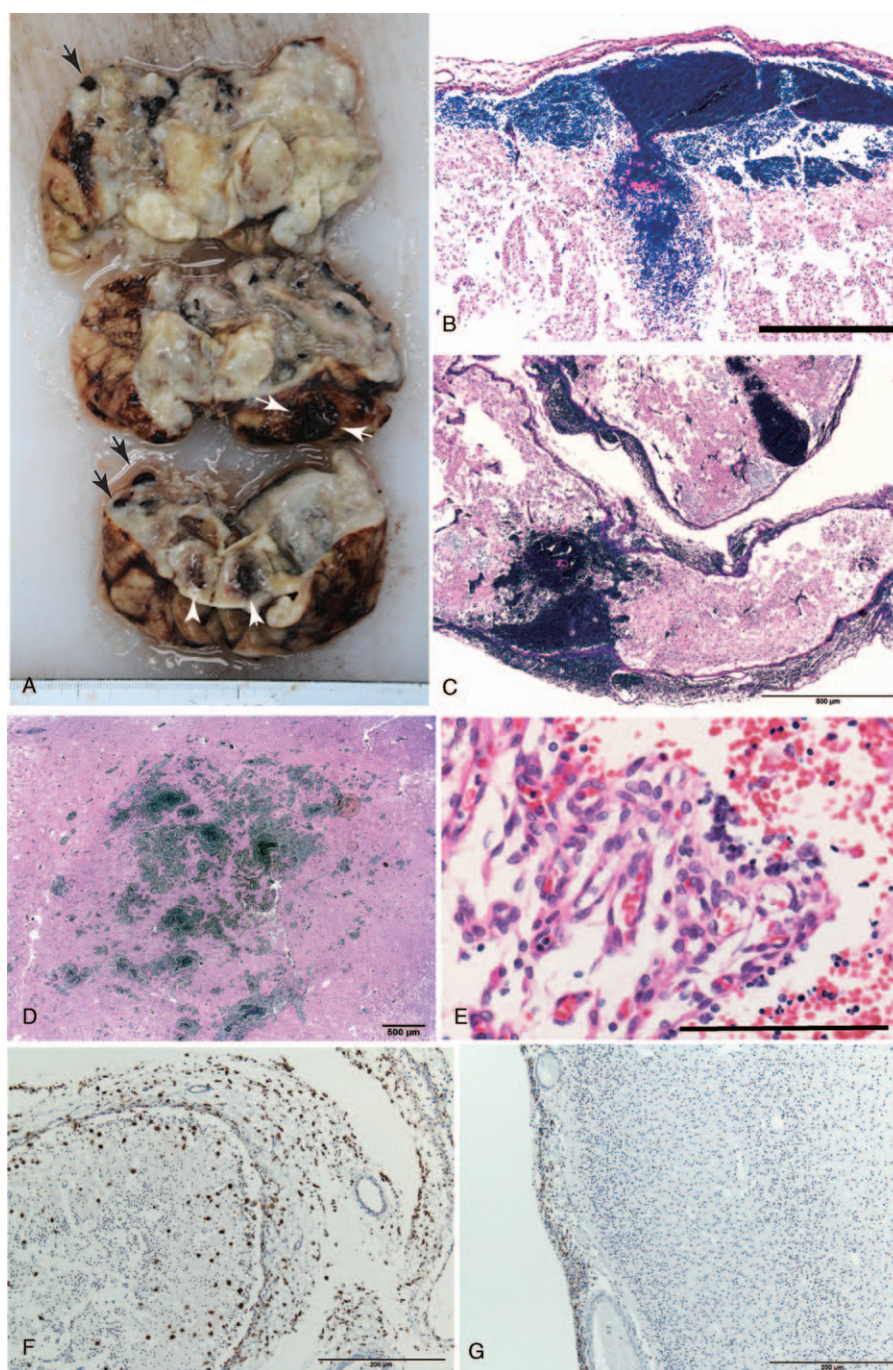


Figure 2. Pathologic appearance of the brain. (A) Coronal section of the brain. In addition to marked softening resembling jelly, fresh hemorrhage was seen in the cerebral convexity (black arrows), basal side (white arrows), and bilateral thalamus (arrowheads). (B–E) Histologic appearance of the brain using Luxol fast blue–hematoxylin/eosin. Examination revealed (B) fresh cortical hemorrhage in the convexity side of the left frontal lobe, (C) fresh cortical hemorrhage in the basal side of the temporal lobe, (D) swelling of the nuclei of endothelial cells in the hemorrhagic area of the temporal lobe, and (E) fresh hemorrhage of the right thalamus. (F, G) Immunohistochemical findings for CD68. (F) Temporal lobe of the infant and (G) an age-matched control (32 weeks’ gestation, abortive fetus due to chorioamnionitis). Markedly increased CD68-positive macrophages were seen in the injured area in contrast to the control case. Scale bar: (D) 500 μm, (B, C, F) 200 μm, and (E) 50 μm.

showed symmetric advanced edematous swelling with liquefaction of the entire cerebrum and cerebellum, subarachnoid hemorrhage with focal cortical hemorrhage in both the cerebral convexity and basal areas, and cerebellar and bilateral thalamic hemorrhage (Figs. 1D, E and 2A). On microscopic examination, myelination was limitedly found in the acoustic nerve and part of the extrapyramidal tract, which was consistent with the typical

appearance at 30 to 36 weeks of gestation.^[13] In addition to fresh cortical and thalamic hemorrhage consistent with the gross appearance (Fig. 2B–D), swelling of endothelial cells (Fig. 2E) and increased CD68-positive microglia and other inflammatory cells were found in and around the areas of cortical hemorrhage (Fig. 2F, G). Axonal fibers positive for anti-amyloid precursor protein, which is a good marker of traumatic axonal injury due to

head trauma,^[14] were limitedly found in and around the area of hemorrhage but were not evident in the white matter of the whole brain. Neither intraventricular hemorrhage nor periventricular leukomalacia was evident.

Other autopsy findings were a left-sided diaphragmatic hernia with mild collapse of the left lung and an atrial septal defect of 3 mm diameter. No significant signs of hypoxia/ischemia were evident in any thoracoabdominal organs (Supplemental Figs. 2 and 3, <http://links.lww.com/MD/C601>).

3. Discussion

Careful differentiation between traumatic injury and non-traumatic brain hemorrhage associated with a hypoxic/ischemic event is often required when evaluating neonatal intracranial hemorrhage in cases of maternal injuries. Besides the lack of intraventricular hemorrhage indicating a hypoxic/ischemic event in a fetus or infant,^[15] the combination of early manifestation of convulsions and signs of intracranial hemorrhage may suggest the occurrence of FCHI by a maternal MVA. However, the pathologic appearance of fetal head injury has not been fully elucidated, possibly because the vulnerable neonatal brain readily deteriorates during the harvesting procedure at autopsy. In the present case, focal cortical hemorrhage, a histologic hallmark of brain contusion, was shown^[16]; bilateral thalamic hemorrhage, which has not been previously reported as a finding of fetal head injury, was also found.

Injury to the uterus and/or placenta may be a predictive factor for a poor fetal outcome, mainly because of impairment of the fetal circulation.^[3,4] In the present case, continuous monitoring of laboratory parameters may suggest a good response to intensive care during the infant's clinical course. The lack of a histologic appearance suggesting an ischemic or hypoxic event in various organs may indicate that the influence of the uterine and placental injuries to the neonatal general condition was more restricted than the influence of FCHI. Progression of the tissue response in and around the hemorrhagic area, such as swelling of endothelial cells and increasing inflammatory cells, might also suggest that brain damage occurred several days before death rather than during the critical period.

Although the pathophysiology of FCHI remains unclear because of the lack of corresponding case reports, we can propose a mechanism of fetal brain contusion based on nonfetal cases. Two kinds of force are involved in the pathogenesis of brain contusion: one is shear force failure, and the other is tensile failure (also termed the contrecoup mechanism).^[16] In the present case, we assume that both shear and tensile forces may have shifted the brain enough that it collided with the skull and tentorial notch, respectively, because traumatic contusion of the brain was found in both the cerebral convexity and base. From this perspective, hypermobility of the fetal head in association with the breech position may have been a significant factor contributing to the severe brain contusion in the present case, despite the fact that a skull fracture did not occur. Although the body and brain development of the infant was within the normal range, the structural vulnerability of the fetal brain should always be considered a substantial risk factor for FCHI.

Traumatic basal ganglia hemorrhage is also uncommon in adults and is defined as a hemorrhagic lesion in the basal ganglia or a neighboring structure such as the internal capsule or thalamus. The general incidence of traumatic basal ganglia hemorrhage reportedly ranges from approximately 3% to 10%, and bilateral hemorrhage is even more rare.^[17,18] The pathophysiologic

mechanism of such hemorrhage has not been fully established; however, arterial tearing and stretching during the trauma may cause rupture of the perforators of various cerebral arteries including the anterior choroidal artery, middle cerebral artery, posterior cerebral artery, or microvasculature. Patients with traumatic basal ganglia hemorrhage have a relatively low incidence of skull fracture in contrast to the high incidence of other associated intracranial lesions.^[17,18]

Although recent studies have shown that proper employment of seat belts helps to prevent maternal and fetal injuries associated with MVAs,^[3,4] whether airbag employment in MVAs increases the risk of maternal and/or fetal injury remains unknown. In our consideration of the significance of safety equipment in the present case, we noted 2 factors: one is that the car in which the mother rode was collided by an oncoming vehicle from the right side and overturned toward the left side, and the other was an abrasion in the left iliac region of the mother. These findings may indicate that the acceleration force by the collision may have mainly been generated in the horizontal rather than longitudinal direction of the mother; if so, the airbag may not have caused harm to the mother, but the left side of her lower abdomen may have been momentarily compressed by the seatbelt, leading to increased intra-abdominal pressure. A study by Aitokallio-Tallberg and Halmesmaki^[19] showed that impact from the side and subsequent rotation toward the horizontal direction of the car might be associated with placental abruption and fetal death, although the detailed mechanism remains undetermined.

In summary, we have presented a case of complex FCHI due to a maternal closed abdominal injury associated with an MVA. Overlap of multiple factors, including the structural vulnerability of the fetal brain, the head posture of the fetus, the crash location and direction of force, and the employment of safety equipment, may have contributed to the occurrence of FCHI in the present case.

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