Virchows Arch [Pathol Anat] (1982) 398:229–235

Virchows Archiv A

Case Report

Pulmonary and Systemic Cerebellar Tissue Embolism due to Birth Injury

N. Böhm, K.M. Keller, and W.D. Kloke
Sektion Kinderpathologie, Pathologisches Institut (Ludwig-Aschoff-Haus) der Universität Freiburg, Albertstrasse 19, D-7800 Freiburg i.Br., Federal Republic of Germany

Summary. Brain tissue embolism in the coronary, leptomeningeal and pulmonary arteries was discovered microscopically following the autopsy of a female newborn. Death occurred 8 h after breech delivery, which had been complicated by both arms being turned up beside the head. The dislodged brain tissue originated from the left cerebellar hemisphere and had entered the venous blood stream through a rupture of the left sinus transversus. Both "paradoxical" systemic and pulmonary artery embolization ensued. The baby died from the combined effects of cerebral haemorrhage, pulmonary embolism, myocardial infarction and shock.

Key words: Birth injury – Breech presentation – Brain tissue embolism – Neonatal myocardial infarction – Neonatal pulmonary embolism – Shock.

Pulmonary brain tissue embolism is a rare, but well known complication of severe head injuries in adults (Ceelen 1931; Oppenheimer 1954; McMillan 1956) and children (Krakower 1936; Nunes 1955; McMillan 1956). Brain tissue embolism caused by birth injury, however, is largely unknown among obstetricians and pathologists. Nevertheless, at least 10 cases have been reported in the literature, usually as a complication of deliveries with breech presentation (Table 1).

We report here a case with extensive multiple cerebellar tissue emboli in the pulmonary, leptomeningeal and coronary arteries of a newborn, which had survived for 8 h. Myocardial infarction and coagulopathy were secondary events. We advance an explanation of the pathophysiology and of the timing of the pulmonary and systemic embolization during breech delivery, on the basis of our morphological findings.

Offprint requests to: N. Böhm at the above address

0340-1227/82/0398/0229/$01.40

Table 1. Brain tissue embolism in newborns with birth trauma

Case No.	Author	Year	Sex/weight	Presentation	Birth-procedure	Survival	Location of emboli
1.	Abrikossoff	1913	male/3,300 g	breech	?	5 min	left coronary art.
2.	Gardiner	1956	male/?	vertex	forceps	10 min	pulm. art.
3.	Tryfus	1963	?/1,800 g	footling	Veith-Smellie	10 min	pulm. art. plex. chorioideus pial veins
4.	Valdes-Dapena	1967	male/2,050 g	breech	forceps	1 h	pulm. art.
5.	Valdes-Dapena	1967	female/3,540 g	vertex	forceps	16 min	pulm. art.
6.	Gagné	1970	male/2,970 g	breech	forceps	12 h	pulm. art. pial veins coronary art.
7.	Gagné	1970	female/677 g	breech	spontaneous	1 h	pulm. art. pial veins peripancreatic and renal capsular art.
8.	Fobes	1971	male/2,750 g	footling	forceps	18 h 25 min	pulm. art.
9.	Gariépy	1973	male/3,090 g	breech	forceps	18 min	pulm. art. pial veins
10.	Moragas	1974	male/1,000 g	vertex	spontaneous	6 h	pulm. art. renal art.
11.	Present case	1982	female/2,850 g	breech	Müller and Veith-Smellie	8 h	pulm. art. left coronary art. cerebral art.

Case Report

A 24-year-old woman, para 2, gravida 2, was admitted in labour to the hospital one week prior to the expected date of delivery. Breech presentation was diagnosed by clinical and sonographic examination. Since the patient had previously had an uncomplicated vaginal delivery, and since the baby appeared to be relatively small, breech delivery seemed appropriate. Delivery was complicated, however, by both arms being turned up beside the aftercoming head, so that the baby had to be extracted by the classical manoeuvres of Müller and Veith-Smellie.

The mature girl (2850 g bodyweight = 25th percentile, 49 cm crown-toheel length, and 34 cm head circumference) was floppy and cyanotic with Apgar scores of 3/5/7 after 1/5/10 min respectively. After resuscitation the baby's respiratory and cardiovascular condition was stabilized. The baby died 8 h later from sudden and irreversible cardiac arrest.

Autopsy Findings

At autopsy, the baby showed mild cyanosis of the lips, the arms and the legs. The ano-genital region was swollen and discoloured bluish-red – a consequence of breech presentation. No malformations were discernible. The placenta was not available for examination.

No traumatic lesions of the head were visible externally. After removal of the galea, bulging of the anterior fontanel (measuring 40 × 50 mm) and widening of the sutures was noted. The skull bones were intact. After opening the skull by a circular section, massive subdural and subarachnoidal haemorrhage was found. This covered the entire surface of the

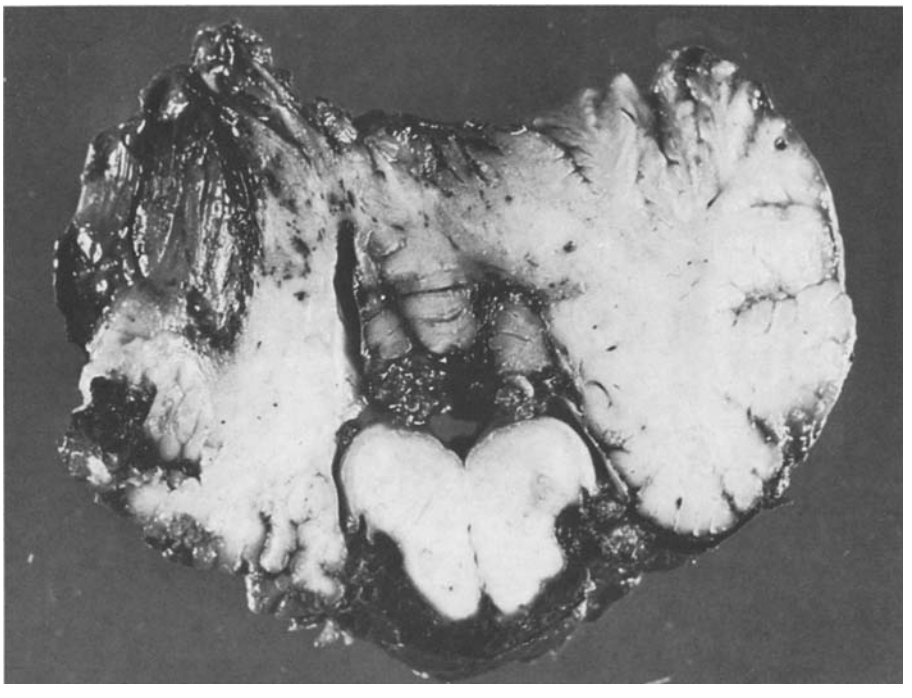

Fig. 1. Substantial defect of the left cerebellar hemisphere. Subarachnoid and focal parenchymal haemorrhage

brain and was particularly prominent in the region of the left cerebellar hemisphere. A 1 cm longitudinal tear with transverse extensions was detected in the left sinus transversus next to the confluens sinuum.

Autopsy of the brain was performed after 2 weeks formalin fixation. The weight was 410 g. Marked oedema was seen on frontal sections, and a small subependymal haemorrhage was detected in the lower horn of the right lateral ventricle. Sections of the cerebellum revealed a defect of the left cerebellar hemisphere with considerable loss of brain tissue (Fig. 1).

There were multiple petechiae of the serous membranes (pleural and abdominal) and marked interstitial pulmonary and mediastinal emphysema, but no pneumothorax.

The foramen ovale was patent, the ductus arteriosus was constricted.

Histology

Microscopically, the pulmonary alveoli were only partially aerated. There were areas of atelectasis, intraalveolar haemorrhage and amniotic fluid constituents. Many of the medium and small pulmonary arteries were obstructed by brain tissue emboli, which still revealed the typical pattern of infantile cerebellar cortex (Fig. 2). In addition, fibrin and platelet aggregates were found covering some of the embolic cerebellar tissue particles, further adding to the obstruction of the lumen.

Cerebellar cortical tissue emboli were also found in the interventricular branch (ramus descendens anterior) of the left coronary artery and in the smaller intramyocardial vessels. Recent myocardial necrosis was observed in the anterior wall of the left ventricle and in the septum. Granulocytic infiltrates were seen subendocardially and in the immediate vicinity of the myocardial infarctions (Fig. 3).

Some leptomeningeal arteries (branches of the middle cerebral artery) were also occluded by cerebellar tissue emboli (Fig. 4). The fibres of the internal elastic lamina were clearly demonstrated in the vessel walls by the elastic van-Gieson stain.

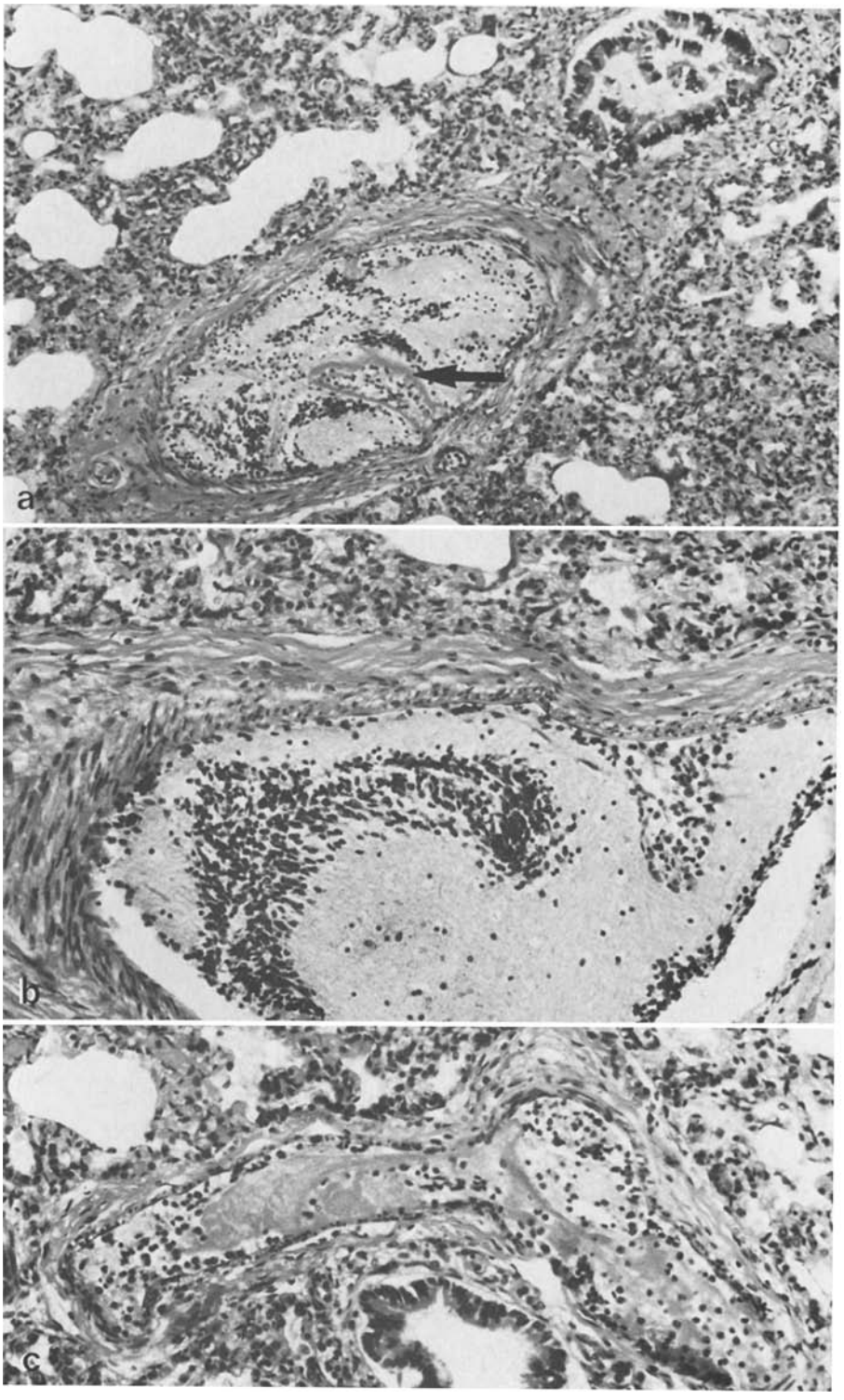

Fig. 2. Pulmonary cerebellar tissue embolism. **a** Embolic obliteration of a medium sized pulmonary artery. Laminar precipitations of fibrin and platelets (↙). HE, ×100. **b** Typical pattern of infantile cerebellar cortex in a pulmonary embolus. HE, ×160. **c** Intravascular laminar precipitations of fibrin and platelets next to the brain tissue fragments. HE, ×160

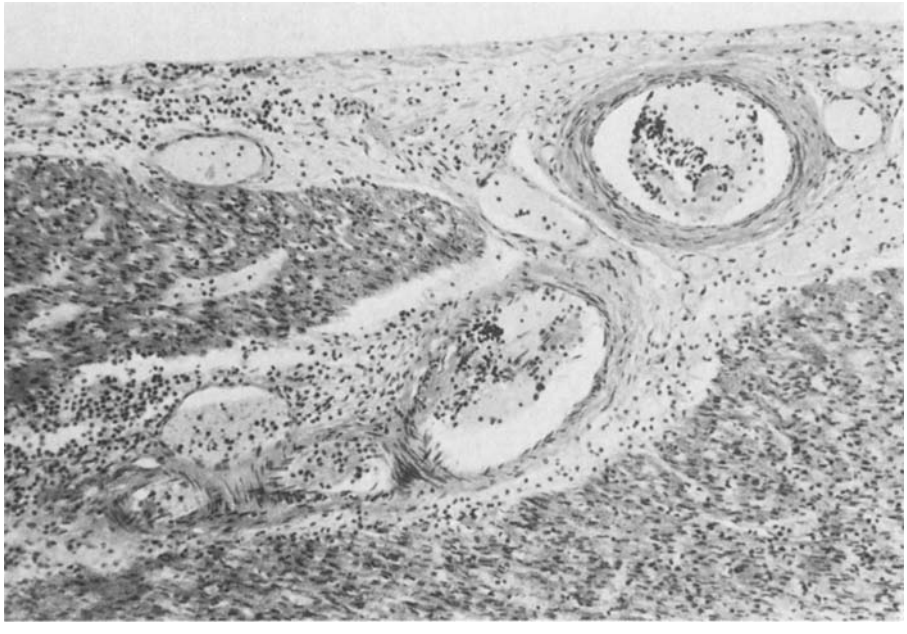

Fig. 3. Cerebellar tissue emboli in the left coronary artery branches. Recent myocardial infarction and interstitial granulocytic infiltrations. HE, ×100

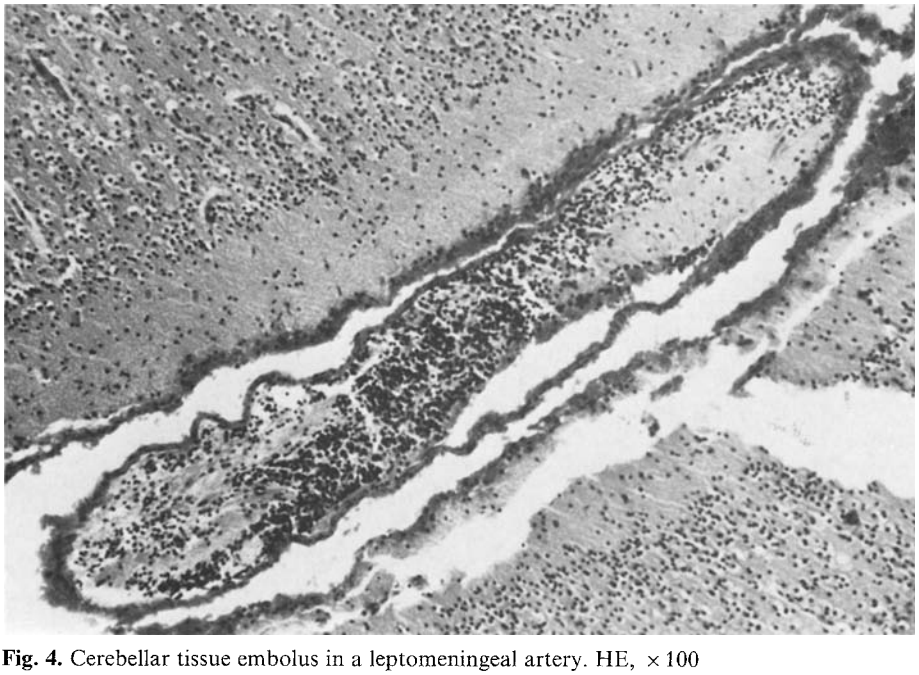

Fig. 4. Cerebellar tissue embolus in a leptomeningeal artery. HE, ×100

Brain tissue emboli were not detected in the renal, splenic, hepatic, gastrointestinal and urogenital arteries, and were also absent from the vessels of the endocrine organs. The peripheral arteries of the limbs were not investigated.

Discussion

Searching the literature, we found 10 earlier case reports of brain tissue embolism caused by birth injury (Table 1). Abrikossoff (1913) first described "paradoxical" brain tissue embolism in the coronary artery of a newborn. In 9 of the 10 cases, pulmonary embolization was found (Gardiner 1956; Tryfus 1963; Valdes-Dapena and Arey 1967; Gagné 1970; Fobes and Hirst 1971; Gariépy and Fugere 1973; Moragas et al. 1974). In addition, Moragas et al. (1974) detected brain tissue in a renal artery, and Gagné found small emboli in the coronary, parapancreatic and renal capsule arteries. Tryfus (1963), Gagné (1970) and Gariépy and Fugere (1973) described brain tissue particles in small pial veins. They proposed retrograde embolism as a possible explanation for this unusual and unexpected finding. However, we think that the extremely dilated thin-walled leptomeningeal arteries dilated by the presence of the embolus might have been mistaken for veins.

Table 1 reveals that brain tissue embolism is associated with breech or foot presentation (8/11 cases) and forceps delivery (6/11 cases). Both conditions are known to bear an increased risk of tentorial laceration and intracranial haemorrhage. Usually tentorial tears occur at or near the free anterior margin of the tentorium. Thus veins, if torn, would be too small to permit embolization of brain tissue. The rare event of brain tissue embolism is only possible if a large venous sinus has been ruptured. In both the case of Abrikossoff (1913) and in our case, it was the transverse sinus near the confluens sinuum which had been lacerated, indicating similar types of sheering force in the two cases. The occipital bone was not fractured in either case. The penetration of lacerated brain tissue into the sinus was then caused by different and intermittent pressures acting on the aftercoming fetal head during breech deliveries both, with and without forceps application.

In the case reported above the breech presentation was complicated by both arms being turned up beside the head. No forceps or vacuum was used. It may be assumed, however, that considerable force had to be applied during the final extraction of the head. The dislodged brain tissue was transported by the venous blood to the right atrium. Subsequent embolization was dependent on the type of circulation present. Initially, while fetal circulation was still maintained, "paradoxical embolization" to the systemic arteries resulted. Theoretically embolization might have been possible via the foramen ovale and/or via the ductus arteriosus. However, as only the coronary and the cephalic arteries were affected, the foramen ovale must be considered as the main (or only) route of embolization. Subsequently, while the head was still trapped and compressed in the birth canal, but the abdomen and thorax were decompressed, the lungs became increasingly perfused, and embolization to the pulmonary arteries resulted.

The baby survived for 8 h, allowing enough time for the development of histomorphological features of myocardial infarction, as well as for intravascular aggregates of fibrin and platelets induced by the brain tissue emboli. Brain tissue is well known to cause plasmatic coagulation, shock and consumption coagulopathy upon direct contact with the blood stream. Consequently, the multiple petechial haemorrhages found in the lungs and serous membranes have to be interpreted as a consequence of shock.

The baby finally succumbed due to the combined effects of cerebral haemorrhage, pulmonary embolism, myocardial infarction and shock.

Acknowledgements. The authors appreciate Dr. S.M. de Looze's kind assistance during language revision and Mrs. Annemarie Moeller's skilful typing of the manuscript and express their gratidute to the neuropathologists for autopsy of the brain.

References

Abrikossoff AJ (1913) Zur Kasuistik der Parenchymembolien: Kleinhirngewebeembolie der Arteria coronaria cordis beim Neugeborenen. Cbl Pathol Anat 24:244–246

Ceelen W (1931) Die Kreislaufstörungen der Lunge. In: Henke F, Lubarsch D (eds) Hdb d spez Pathol Anat u Histologie, Bd III/3.

Fobes CD, Hirst AE (1971) Brain embolism to the lung. A complication of breech delivery. J Am Med Ass 218:735

Gagné F (1970) Embolies pulmonaires de tissue cérébelleux chez le nouveau-né. Union Méd Can 99:275–277

Gardiner WR (1956) Massive pulmonary embolization of cerebellar cortical tissue. An unusual fetal birth injury. Stanf Med Bull 14:226–229

Gariépy G, Fugere P (1973) Pulmonary embolization of cerebellar tissue in a newborn child. Obstet Gynecol 42:118–120

Krakower C (1936) Pulmonary embolus containing cerebral tissue. Arch Pathol 22:113–115

McMillan JB (1956) Emboli of cerebral tissue in the lungs following severe head injury. Am J Pathol 32:405–415

Moragas A, Ballabriga A, Vidal MT (1974) Atlas de Histopatologia Neonatal. Salvat Editores, SA, Barcelona

Nunes MA (1955) Embolia pulmonar de tecido cerebral. Gaz Med Portuguesa 8:247–250

Oppenheimer EH (1954) Massive pulmonary embolization by cerebral cortical tissue. Bull Johns Hopk Hosp 94:86–89

Tryfus H (1963) Massive pulmonary embolism of cerebellar tissue in a newborn. Arch Dis Childh 38:292–294 (1963)

Valdes-Dapena MA, Arey JB (1967) Pulmonary emboli of cerebral origin in the newborn. Arch Pathol 84:643–646

Accepted September 30, 1982

Note Added in Proof

Following submission of the manuscript of this paper another three cases of perinatal cerebellar tissue embolism were briefly reported (Vogel M, Stoltenberg-Didinger G (1982) Geburtsh Frauenheilkunde 42:634)