Continuation of pregnancy in a woman with critical brain injury

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Abstract

Background: Critical brain injury can lead to brain death, which is medically and legally considered the death of an individual. Further therapy is discontinued, unless organ donation is possible or brain death occurs in a pregnant woman.

Case report: A 30-year-old woman at 22 weeks gestation developed a subarachnoid haemorrhage from a ruptured cerebral artery aneurysm. The patient was admitted to the Intensive Care Unit in critical condition. On treatment day 3, the symptoms of brain death occurred. Due to possible complications, the apnoea test and instrumental examinations were not performed. Therapy maintaining vital functions was carried out in order to sustain the pregnancy. The patient was ventilated, received cardiac-supportive drugs, hormone replacement therapy, enteral and parenteral feedings and systemic infections were treated as well. At the beginning of the 27th week of gestation, massive bleeding from the airways developed. A Caesarean section was performed, and a female neonate was born, birth weight 680 g, the Apgar scores 4, 6 and 6 at 1st, 5th and 10th minute, respectively. After 3.5 months, the baby was discharged from the Neonatal Intensive Care Unit. Her development at the age of 8 months is normal.

Conclusions: The case described and similar cases reported in the literature demonstrate that the maternal brain death is an interdisciplinary medical challenge. Thanks to intensive care techniques, maternal somatic functions can be maintained, and a healthy child can be delivered.

Key words: intensive therapy, pregnancy, subarachnoid haemorrhage; Caesarean section, neonate, resuscitation

Severe irreversible brain damage leading to death is a tragic and is often a sudden and abrupt event. The major causes resulting in this condition include the effects of spontaneous cerebral haemorrhages and craniocerebral trauma. The pathophysiology of brain injuries is complex and cause-dependent; however, the dominating mechanism is a critical increase in intracranial pressure exceeding the systemic pressure, which arrests the cerebral flow and results in cerebral hemisphere infarction. Brain stem destruction results from tonsillar herniation. The entire brain dies, which means the death of an individual. Any further therapy is futile, and in accordance with legally binding regulations in a given country, therapy is discontinued. Its continuation is possible in two situations – when organ donation is considered or in cases of brain-dead pregnant women [1].

During the years 1982–2010, 30 cases of pregnant patients with confirmed brain death, and several cases with irreversible cerebral coma were described. In all of the situations, intensive therapy was carried out to sustain the pregnancy and deliver a viable child, which was achieved at different times and with various outcomes. Organs for transplantation were retrieved from several of those mothers [2–4]. The aim of this report was to present the management of a pregnant patient diagnosed with massive cerebral haemorrhage and features of brain death.

CASE REPORT

A 30-year-old woman in the 22nd week of her 4th pregnancy was admitted to the ICU from the Department of Obstetrics and Gynaecology after a short-term (approximately
On ICU day 2, following a sudden increase in arterial pressure, the patient developed hypotension 60/30 mm Hg and bilateral pupil dilatation. An intravenous infusion of dopamine was started at an initial dose of 7 µg kg\(^{-1}\) min\(^{-1}\); the next hour an intravenous infusion of noradrenaline was added at a dose of 0.02 µg kg\(^{-1}\) min\(^{-1}\). During the subsequent days, infusions of both amines were continued, and haemodynamic stability was restored: SAP/DAP 140/80 mm Hg, HR 80 min\(^{-1}\).

On day 3, the patient was critical. The repeated CT of the head demonstrated massive subarachnoid haemorrhage, more intensified (compared with the previous CT), features of intracranial narrowing in the form of the compressed ventricular system and basal cisterns, smoothing of the cerebral hemisphere sulci and a diffuse hypodensity of the white matter. On the following day, the ventilation parameters deteriorated markedly due to the development of neurogenic pulmonary oedema: \(\text{SaO}_2\) 86%, \(\text{PaO}_2\) 69 mm Hg (9.2 kPa), \(\text{PaCO}_2\) 78 mm Hg (10.4 kPa) at \(\text{F}_{\text{O}}_2\) 0.45. The physical examination of the lungs did not show any abnormalities. The ventilation parameters were increased, i.e., \(f\) from 14 to 18 min\(^{-1}\), \(\text{F}_{\text{O}}_2\) from 0.45 to 0.7. After 48 hours, the arterial blood gas results were as follows: \(\text{SaO}_2\) 99%, \(\text{PaO}_2\) 96 mm Hg (12.8 kPa), \(\text{PaCO}_2\) 46.9 mm Hg (6.3 kPa).

The neurological examination found a lack of brain stem and medullary reflexes, as well as spontaneous respiration. Brain death was suspected. The apnoea test was not carried out due to the life-threatening foetal condition. The patient’s transport for the instrumental examination confirming brain death (cerebral angiography) was considered too risky.

The patient’s family was informed about the situation and poor prognosis. The circumstances were extremely difficult for family members and initially raised uncertainty and numerous doubts. The biggest doubts regarded the health of the child. The decision to continue the treatment to support the pregnancy and increase the survival chances of the foetus was taken together with the patient’s family.

Between day 3 and 7, the patient received sublingual desmopressin in a dose of 60–120 µg due to the symptoms of diabetes insipidus. From day 10 and onward, peripheral oedema started to increase. Despite therapy with loop diuretics, which was successful (diuresis — 150–200 mL h\(^{-1}\)), the oedema persisted, and on day 25 the weight gain was 20 kg.

On day 6, the intravenous infusion of dopamine was discontinued, and the infusion of noradrenaline continued (until day 17) at a dose ranging from 0.02 to 0.1 µg kg\(^{-1}\) min\(^{-1}\). Arterial pressure was stable — 140/80 mm Hg,
HR 60–80 min⁻¹. The supply of 20% magnesium sulphate and verapamil hydrochloride was continued.

Between day 3 and 7, the patient had disorders of thermoregulation with temperature fluctuations from 35 to 41°C. During the subsequent days, the body temperature was within the range of 37–39°C.

On day 7, a tracheostomy was performed. During the following four days, the arterial blood gas was: pH 7.30–7.46, PaO₂ 86–187 mm Hg (11.5–24.9 kPa), PaCO₂ 32.7–64 mm Hg (4.4–8.5 kPa), SaO₂ 83–100% (at FIO₂ 0.5–0.7). The chest X-ray performed on day 12 showed the lungs without interstitial densities, with a slight amount of fluid in the left pleural cavity.

A urinary tract infection was diagnosed in the patient and was treated with half-synthetic aminopenicillins (days 3–7) followed by second-generation cephalosporins (day 7–15). On day 13, the body temperature increased to 40°C and the C-reactive protein (CRP) was 26.8 mg L⁻¹. Blood cultures showed Candida tropicalis; the culture of the broncho-tracheal secretion revealed the presence of methicillin-susceptible Staphylococcus aureus, Klebsiella pneumoniae ESBL(-), and Enterococcus faecalis. In accordance with the antibiogram findings, the treatment was supplemented with lincosamides and antifungal preparations from the group of triazoles and imidazole derivatives.

On day 15, the physical examination revealed crepitations over the lung fields with considerably decreased respiratory murmur; the arterial gas parameters were as follows: PaO₂ 75.5 mm Hg (10.1 kPa), PaCO₂ 91.2 mm Hg (12.2 kPa), SaO₂ 92% (at FIO₂ 0.7). The laboratory tests demonstrated WBC 8.5 G L⁻¹, CRP 13.2 mg L⁻¹, procalcitonine 0.16 ng mL⁻¹. The body temperature was 38–39°C. Bronchofiberscopy was performed, during which a large amount of purulent secretion was evacuated which had caused the obstruction of the left bronchus. The bronchoalveolar lavage (BAL) was collected for quantitative microbiological culture, which showed the presence of ESBL(-) Klebsiella pneumoniae and Acinetobacter baumannii in the amount of 10⁶ CFU ml⁻¹. Based on the clinical presentation, prolonged mechanical ventilation (≥ 48 h) and characteristic quantitative BAL culture findings (≥ 10⁶ CFU mL⁻¹), ventilator-associated pneumonia (VAP) was diagnosed. Since day 18, according to the antibiogram, the patient was given carbapenems. The FIO₂ was increased to 1.0 and the PEEP to 15 cm H₂O (1.47 kPa) which resulted in a PaO₂ 90–211 mm Hg (11.9–28.1 kPa), PaCO₂ 56.7–80.9 mm Hg (7.6–10.8 kPa), SaO₂ 98–100%. In total, four bronchofiberscopes were performed during which large amounts of purulent secretions were aspirated. On day 13, during the final bronchofiberscopy, the presence of bloody secretion was additionally found in the entire bronchial tree.

The biochemical tests demonstrated electrolyte abnormalities and hypalbuminaemia, low levels of thyroid hormones with a simultaneous decrease in TSH concentration to the undetectable level (below 0.005 µU ml⁻¹ on day 28) and a low level of cortisol (29.16 mmol L⁻¹ since day 15). Since day 5, the patient developed anaemia (Hb 5.0 mmol L⁻¹), and since day 28, thrombocytopenia (PLT 83–86 G L⁻¹).

Since day 17, the patient received methylprednisolone, initially in a daily dose of 15 mg kg⁻¹, followed by 10 mg kg⁻¹. On day 18, methyldopa was re-instituted due to an elevated arterial pressure of 180/100 Hg, which reduced the pressure to 130/70 mm Hg and HR 80–100 min⁻¹. At gestational week 24, the repeated ultrasound examination revealed preterm placental maturation (grade II of maturity according to the Grannum classification). The maturation of foetal lungs was accelerated with the administration of dexamethasone.

The patient was fed enterally through a naso-intestinal tube and parenterally. The protein- and energy-rich mixtures were supplemented with multivitamin preparations. Due to persisting anaemia, iron and folic acid preparations were administered, and multiple units of red blood cells were transfused.

The foetal state was monitored during daily obstetric consultations, and ultrasound examinations were carried out every few days. Neither the inhibition of intrauterine growth nor placental pathologies were observed.

The neurological condition of the patient did not change. Throughout this period, she was unconscious, without spontaneous respiration and brain stem reflexes. Moreover, spinal reflexes were not noted.

On ICU day 31, i.e., gestational week 26 (Hbd 26.2), approximately 1 h after bronchofiberscopy, the patient developed massive haemorrhage from the bronchial tree which caused hypotension and desaturation with significantly increased cyanosis (SaO₂ 78%, PaO₂ 56 mm Hg/7.47 kPa, PaCO₂ 73 mm Hg/9.73 kPa). Considering the critical maternal condition and the life-threatening condition of the foetus, an emergency Caesarean section was performed, which was carried out in the ICU. The risk of sudden cardiac arrest in the patient was very high; therefore, she was not transported to the operating suite. The anaesthesiological care was limited to resuscitation procedures. Lung ventilation was continued, fluid therapy was administered (the red blood cell concentrate, fresh frozen plasma, colloids), the infusion of noradrenaline was instituted, yet there was no improvement.

The live child, weighing 680 g, was delivered. The Apgar scores were 4, 6 and 6 at 1, 5 and 10 minutes, respectively. During the first day, the preterm baby was intubated and transported to the neonatal intensive care unit. Due to res-
piratory distress syndrome, the child had to be mechanically ventilated. The child’s condition during the first weeks of life was serious. Apart from the respiratory failure, the new-born had features of intrauterine infection and substantial anaemia. After 3.5 months, the child was discharged in good general condition weighing 3000 g. Her development at the age of 8 months is normal.

After the procedure, the patient was in agony. Asystole developed 1 h after the Caesarean section.

**DISCUSSION**

Support of vital functions in critical conditions of brain damage leading to death is an attempt to slow down the progressing and irreversible dysfunction of all organs and systems. This is possible thanks to the advances in intensive therapy whose procedures can shift the borders of somatic irreversible disintegration of the body and delay the development of asystole [5, 6]. The longest duration (107 days) of therapy in a pregnant woman diagnosed with brain death completed with the delivery of a new-born (Caesarean section) was reported in 1989 [7].

In the case presented, brain death was suspected on ICU day 3. The examination demonstrated the lack of 7 stem reflexes and no respiratory function, evidencing the cessation of brain stem functions. The specificity of systemic disorders, including water-electrolyte, metabolic, thermoregulatory, of the cardiovascular system and hypothalamic-pituitary axis indicated brain death [2, 4, 5, 8].

In the situation described, the procedures to confirm the diagnosis of brain death were not completed. The apnoea test and instrumental examination were not performed. The priority was to ensure the best conditions for foetus survival [9]. Considering the primarily supratentorial and secondary (short-term circulatory arrest) cause of brain damage, the instrumental examination to confirm brain death is not obligatory. Finding the lack of cerebral flow would confirm brain death, yet in this case, it would not have affected further medical actions.

Despite the obvious physiological dependence, thanks to the differences between the maternal and foetal organisms, the intrauterine foetal growth was not inhibited. However, when the disease developed, the very early gestational age (week 22) made the delivery impossible. The optimal period for delivering a living and healthy child would be the gestational weeks 32–34. Prior to week 24 of foetal life, the child would have a 20−30% chance for survival, with a 40% probability of severe neurological disorders. Much better prognosis occurs in children born between the weeks 24 and 28, when the survival increases to 80%, and the risk of neurological complications is 10%. After gestational week 32, the risk for life and health of a child is the lowest: 98% survival and less than 2% of neurological complications [2, 10].

The situation presented in our report is a challenge for intensive care, obstetrics, and neonatology, as well as ethics and law. The respect for maternal and foetal autonomy when the mother cannot decide about her child requires special management involving not only the medical but also moral and legal issues. The patient’s family should be involved in all decisions and be aware of the responsibility for possible care of a child [10, 11]. The situation in question is exceptionally complex and can provoke extremely different emotions and opinions, not only of the relatives but also of society [12, 13].

The therapy supporting somatic functions in pregnant women with critically damaged brains requires uneasy and emotionally burdened decisions. In cases with the suspicion of maternal brain death, it is difficult to perform the procedure confirming brain death without threatening the life of a foetus. The proper timing for confirmation would be the post-delivery period. Yet in our case, the patient died promptly after delivery.

From the ethical point of view, the success is to give a real chance to deliver a living baby despite the mother’s death. Nevertheless, the consequences of a delivery that occurs too early and the risk of severe neurological complications associated with it can raise doubts concerning the limits of medical management. Therefore, each case should be treated prudently and individually.

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**References:**


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