Good Neurological Outcome After Hypoxic-ischemic Coma Caused by Severe Postpartum Hemorrhage: Three Case Reports and Literature Review

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Summary. Postpartum hemorrhage is a life threatening condition, which may cause a variety of disorders that may also be associated with long-lasting hypoxia. Due to progress in resuscitation, the complications of hypoxic ischemic encephalopathy are found to be various not only in post cardiac attack setting, but also in any cause of bleeding. Our objectives were to present three case reports of neurological outcome after severe postpartum hemorrhage and analyze the literature in this field including prognostication. The details of hemorrhage were collected retrospectively from the medical records. After mean follow-up time of 35 months the women were invited to neurological examination, neuropsychological assessment (Addenbrooke's Cognitive Examination – Revised adapted to Lithuanian speaking population – ACE-R^{LT}), depression and anxiety evaluation (Hospital Anxiety and Depression Scale – HADS). All three women were severely bleeding after labor with signs of disseminated intravascular coagulation and syndrome of multiple organ dysfunction. Two of them experienced asystole of 5 and 4 minutes, and the third had hemoglobin value of 15 g/L. Impairment of consciousness due to severe brain hypoperfusion evolved to hypoxic-ischemic coma in all three cases. During critical acute period and subsequently the neurological evaluations were recorded, showing a variety of neurological disorders – delirium, extrapontine myelinolysis, seizures, reversible posterior leukoencephalopathy syndrome and other disabilities, such as ataxia, hypertonus of the limbs and impaired psychosocial abilities. In general, long-term neurological outcome of these patients was found to be very good. It shows good potential adaptive possibilities and recovery of the brain after postpartum bleeding. At follow-up cognitive examination one woman reached maximal ACE-R^{LT} score, others showed the results above 89. All three women have returned to work and were able to drive a car. However, at follow-up they had complained of memory and attention disturbances, and two of them had significantly elevated depression or anxiety score on HADS. In the absence of specific prognostic measures for postpartum hemorrhage, we applied retrospectively the prognostication scores originally dedicated for predicting neurological outcome after cardiac arrest. The results allowed fairly reliably predict good neurological prognosis in patients after severe postpartum bleeding as well. These cases emphasize the importance of cooperation and multidisciplinary approach to the patient and show promising long-term results in this field of medical emergency.

Keywords: postpartum hemorrhage, hypoxic-ischemic coma, brain injury, neurological outcome.

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INTRODUCTION

According to the World Health Organization, postpartum hemorrhage (PPH) is responsible for around 25% of maternal mortality worldwide, and is also one of the leading causes of maternal death in developed countries [1]. The etiology of PPH is divided into four main potential causes, defined as four "T" – Tonus, Tissue, Trauma and Thrombosis, the first of which – uterine atony – plays the most important role. All these conditions have their own predisposing factors, however, a large number of women experiencing PPH have no risk factors at all [2]. Classically, PPH is defined as a blood loss of more than 500 ml following a vaginal delivery or more than 1000 ml following cesarean delivery [3]. In addition to death, serious morbidity may follow PPH, which could cause long term residual effects [4]. The usual sequelae include adult respiratory distress syndrome, coagulopathy, postpartum pituitary necrosis, chronic anemia, and fertility loss due to hysterectomy or fibrosis and adhesions in the cavity of the uterus. PPH dis-

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rupts the systemic circulation, cause cerebral hypoperfusion and even electrolyte disbalance, which may lead to osmotic myelinolysis. However, there are only few reports in literature concerning neurologic consequences and outcomes after PPH [5], contrary to a number of descriptions of neurological outcome after cardiac arrest. Therefore prognostic factors of neurological outcome concerning woman after PPH are neither generally analysed nor described. In this report we would like to present three cases of a good neurological outcome in women, which were in deep coma after severe PPH in intensive care unit (ICU) of Vilnius University Hospital Santariškių Klinikos (VUHSK). Details and circumstances of bleeding, general and neurological condition after PPH were evaluated retrospectively from the medical records. Long-term neurological and cognitive outcome was assessed during follow-up visit of the patients at the VUHSK Center of Neurology; the patients were contacted and invited by phone call. In order to evaluate the cognitive outcome Addenbrooke's Cognitive Examination – Revised recently adapted to Lithuanian speaking population (ACE-R^{LT}) was performed (max. score – 100; orientation-attention – 18, memory – 26, verbal fluency – 14, language – 26, and visuospatial ability – 16) [6]. The scores of Hospital Anxiety and Depression Scale (HADS) were counted for assessment of depressiveness (HADS-D) and anxiety (HADS-A) (min – 0, max – 21 on each subscale). Current subjective complaints of the patients were recorded (on 0–10 point grading scale, where 0 shows the worst, and 10 – the best ability).

CASE PRESENTATIONS

First case

A 33-year-old woman was referred to ICU of VUHSK with severe hemorrhagic shock, disseminated intravascular coagulation (DIC) syndrome and multiple organ dysfunction syndrome (MODS). These complications occurred because of bleeding after a first planned labor due to uterine atony, which resulted in primary PPH leading to hysterectomy in a private maternity hospital. During hysterectomy a 5 min asystole occurred, consequently resuscitation and defibrillation were performed. The patient was transferred to the VUHSK in critically severe condition. She had clinical signs of shock, arterial blood pressure (ABP) was immeasurable, heart beat rate (HR) – 130 b/min, Hb level – 65 g/l, APPT – 180.4 s, lactates – 11.9 mmol/l, blood glucose – 3.7 mmol/l. 2 relaparotomies due to uncontrolled bleeding were performed in VUHSK, which resulted in ligation of both internal iliac arteries. During first two days in VUHSK the patient received massive transfusions of blood products (RBC, PLT, fresh frozen plasma), adrenaline for hemodynamical support, and antibiotics for infection prophylaxis. Hemodialysis was initiated due to acute renal failure. Eventually hemodynamics and general condition was stabilized, however it remained very severe for a few weeks.

Neurological condition after PPH. The woman was admitted to ICU in deepest cerebral coma (Glasgow Coma Score (GCS) 3). Within two weeks after admission GCS improved to 11 with a maximal motor (M) and eye (E) response, but the state of consciousness remained fluctuating. The patient developed pyramidal hypertonia of the limbs and underwent an episode of clonus in the right limbs. 4 weeks after admission she gradually became conscious and started to talk in short sentences. The patient could move her legs and feet (muscle strength 2 points), however, marked pyramidal hypertonia did not improve. One week later she developed a headache, amnesia and horror dreams evolving to episodes of delirium which recurred one week. Repeated neuroimaging tests (2 CT scans and 2 MRIs) were performed during the first months. They initially showed 5 mm subdural hematoma in the left occipital area and diffuse cerebral edema, and after 1.5 months – bilateral hyperintensive signals in basal ganglia and subcortical diffuse lesions in both occipital and left frontoparietal areas. Later the patient was referred to rehabilitation department with remaining complaints of memory and difficulties to concentrate, inability to walk long distances, tiredness, limb weakness, and spasticity. In one month her Barthel index (BI) increased from 75 to 90, functional independence measure (FIM) score – from 91 to 100, and Mini Mental State Examination (MMSE) remained maximal (30 points). After overall 3.5 months treatment in different VUHSK departments and rehabilitation she was discharged home.

Neurological outcome at follow-up. The woman started to drive a car 7 months after the event. 1.5 years after the incident, she returned to the previous work. During the follow-up visit (31 months after the event) we observed significant improvement. The long-term neurological consequences included only mild slowness of movements of right limbs, mild weakness of proximal leg muscles, and increased stretch reflexes without clonus (3+) in legs. The patient complained of limited body flexibility and subjectively rated her memory and concentration/attention abilities 9 and 8 points out of 10, respectively. Cognitive examination revealed very good cognitive functions – the patient scored 97 points out of 100 in ACE-R^{LT} (1 missing point in visuospatial ability, and 2 missing points in verbal fluency). On the other hand, HADS showed significantly increased level of depressiveness (HADS-D: 14 points), contrary to borderline level of anxiety (HADS-A: 7 points).

Second case

A 32-year-old woman started bleeding due to cervical and perineal rupture and uterine hypotony after childbirth of macrosomic newborn (4050 g). Laparotomy was performed, resulting in hysterectomy and ligation of both internal iliac arteries. During the procedure, an episode of 4 min asystole occurred, resuscitation and defibrillation were performed, and the patient was transferred to ICU of VUHSK with suspicion of DIC syndrome. On admission she was already intubated and ventilated, Hb – 68 g/l,

ABP – 70/40 mmHg (maintained with adrenaline), APTT – 183 s., lactates – 3,3 mmol/l, blood glucose – 13,7 mmol/l. Due to continued bleeding a second laparatomy was performed. After two days her ABP was >180 mmHg, and sodium nitropruside was administered to treat hypertension, resulting in ABP reduction to 150/80 mmHg. Massive blood transfusions (RBC, PLT, FFP, prothrombine complex) were given during and after bleeding.

Neurological condition after PPH. The patient was admitted to the ICU with a GCS 6 (M 2, verbal (V) 2, E 2). On the next day GCS was 8 (M 4, V 1, E 3), her stretch reflexes were normal (2+), Babinski sign was positive in both sides. On the fourth day of admission the patient was extubated. She was able to communicate, however, remained disoriented. On the fifth day an hour long episode of headache occurred, with subsequent prolonged loss of vision. Motor impairment included complete paralysis of left arm and paresis of right arm and left leg. MRI was performed and showed hyperintensive signals (T2) in basal ganglia, hippocampus, frontal cortex and parietal lobe bilaterally and right insular area. These clinical and imaging findings were interpreted as hypoxic-ischemic; however, reversible posterior leukoencephalopathy syndrome was also considered (Fig. 1). Within the next five days the vision recovered, and the patient started to move her limbs, however, disorientation persisted. After 19 days in hospital the patient was sent to rehabilitation with complaints of weakness of arms; she was not able to perform finger-to-nose and heel-to-shin tests.

Neurological outcome at follow-up. The patient arrived for a follow-up 38 months after PPH. During this period she had one episode of clonic seizures. The patient returned to her previous work and resumed driving 2 months after the event. At follow-up she admitted writing difficulties and assessed her memory and attention of 6 and 8 points, respectively. On neurological examination, slowness and increased stretch reflexes (3+) in the left arm were found. HADS scores for depressiveness and anxiety were 5 (HADS-D) and 8 (HADS-A). No cognitive deficit was found (ACE-R^{LT} score 100 points).

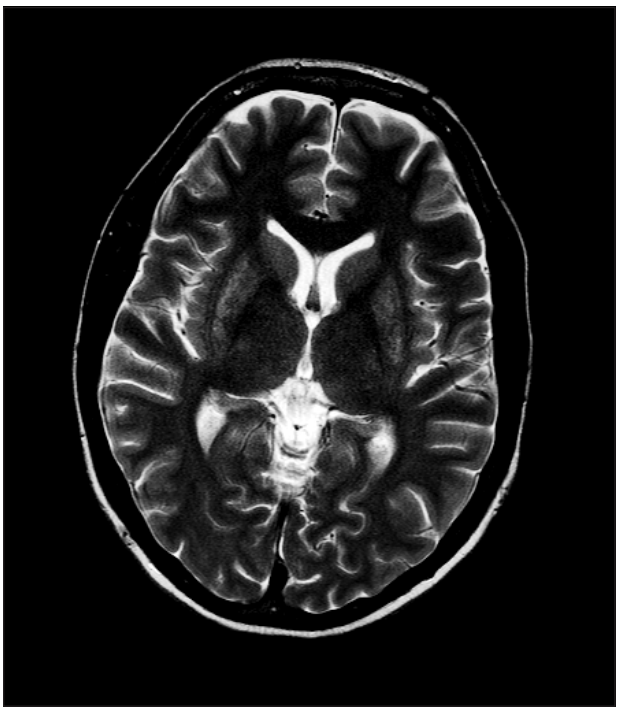

Fig. 1. **Hyperintensive signals (MRI T2) in basal ganglia and hippocampus bilaterally, and right insular area**

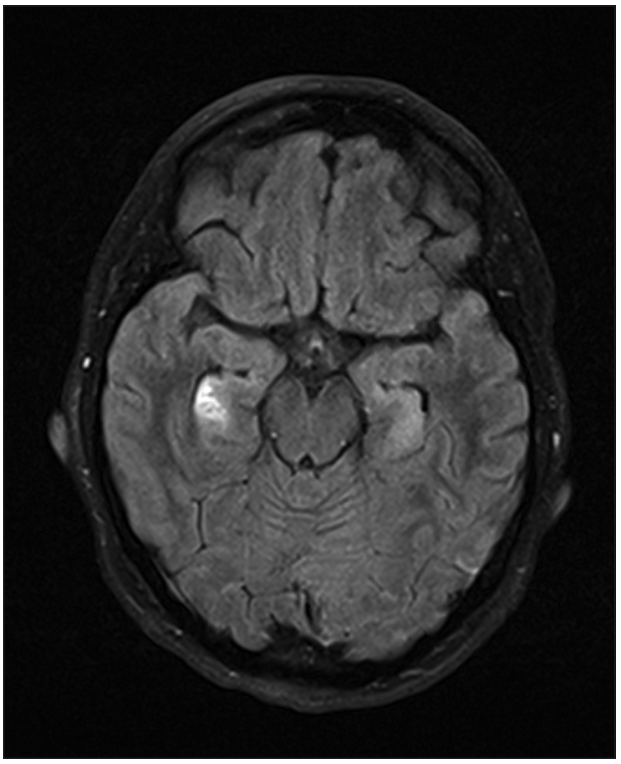

Fig. 2. **Extrapontine demyelination in corpus callosum, limbic lobes of parahippocampal gyrus and hippocampus (MRI T2 dark fluid)**

Third case

A 27-year-old woman was admitted to the ICU of VUSKH with PPH due to cervical rupture and atony of the uterus after her first labor. Laparotomic hysterectomy with ligation of both internal iliac arteries was performed. During operation her Hb level decreased to 15 g/l, she received massive blood transfusions (RBC, FFP), and was intubated. On admission she was unconscious; ABP – 112/20 mmHg (maintained with adrenaline), Hb – 90 g/L, APTT – 150 s., blood glucose – 10.3 mmol/l, lactates – 8 mmol/l. As bleeding continued, on the same day revision relaparotomy was performed, which resulted in patient's stabilization and extubation in 2 days. Next day MODS with sepsis were diagnosed. The patient was treated with antibiotics, hemodialysis, blood transfusions, and anticoagulants. Several years after this event von Willebrand disease was diagnosed.

Neurological condition after PPH. The patient was admitted to the ICU with GCS 5. On the next day she had already regained consciousness, however, headache, sleepiness and fluctuating signs of disorientation in time and place appeared. Stretch reflexes were normal (2+) and

symmetrical, Kernig sign was slightly positive. After one week her neurological status apparently worsened – the patient became very sluggish, was able to say only her name and could not respond to any commands. Initial CT scan showed hypodense lesion in brain stem, and subsequent MRI revealed signs of symmetric extrapontine demyelination in corpus callosum, limbic lobes of parahippocampal gyrus and hippocampus (Fig. 2). The patient improved in one week, however, sleepiness, amnesia, disorientation in time and place, impaired attention concentration, slowness of movements remained, and ataxia (abnormal finger-to-nose test) appeared. After 2 weeks (1 month after admission) she was transferred to rehabilitation with BI of 80, MMSE – 24, poor short-term and long-term memory, attention deficit, and slight disorientation in time and place. She had hypoesthesia in right leg (electroneurography showed right peroneal nerve damage), could not accurately perform heel-to-shin test on the left side, and had a positive Romberg sign. After 3 weeks her BI was 95, MMSE – 29, attention and orientation improved, nevertheless, tiredness persisted and she needed assistance for bathing. After 1.8 months treatment the patient was sent to out-patient department for further assistance.

Neurological outcome at follow-up. The patient resumed driving 6 months after the event and returned to her work after 1.5 years. During the follow-up visit (36 months after PPH) she assessed her own memory and attention of 5 and 4 points, respectively. The patient complained of mood fluctuations and tiredness. Neurological examination revealed slow movements and normal muscle tone in both arms. No tremor was present. HADS-D and HADS-A scores were 13 and 14, respectively. Cognitive assessment: ACE-R^{LT} 89 points (missed 1 point in orientation and attention, 4 points in memory, 2 points in language fluency, and 4 points in visuospatial ability).

DISCUSSION

GENERAL. Hypoxic-ischemic brain injury is often caused by cardiac arrest. Less common causes include respiratory arrest, severe hypotension/shock, near-drowning and chemical gas intoxication [7]. In general, patients with brief episodes of systemic hypoxia usually demonstrate clinical features of reversible "metabolic encephalopathy" – few hours of coma, short-lasting motor, sensory, intellectual deficits, transient confusion or amnesia. However, if systemic anoxia-ischemia is more severe, structural damage to specific brain areas may occur. The most vulnerable areas include cerebral cortex, cerebellum, hippocampus, the basal ganglia, and spinal cord [8], probably because these regions are the most metabolically active [9] and "watershed" territories of the brain [10]. Hemorrhagic shock is uncommon cause of neurological impairment, as the classic neuroendocrine response to hemorrhage attempts to maintain perfusion to the heart and brain via central intense vasoconstriction [11]. In addition, if severe hemorrhage occurs in a hospital, all the conditions for providing adequate fluid therapy and packed RBC for maintaining normal organ and cellular perfusion are accessible, therefore the probability of anoxic brain damage is not high. Nevertheless, these cases of PPH present the relevance and hazards of this illness, which requires the clinicians to be vigilant in daily practice.

PROGNOSIS. In literature, prognostic factors of neurological outcome and recovery of consciousness after hypoxic-ischemic coma are widely described in cardiac arrest setting. Different prognostic scales and tables are reported, which are mostly based on anamnesis, clinical and laboratory findings. GCS score alone or its separate elements connected to time are one of the most frequently used criteria [12, 13]. Normally, the pupillary light reflex is an early indicator, while oculocephalic response is a later prognostic factor, which is evaluated in two weeks [14]. Other clinical observations such as brainstem reflexes, function of cranial nerves, including corneal and oculovestibular reflexes, respiratory pattern and data such as duration of CPR, duration of anoxia, and blood glucose levels on admission are also important [15]. These clinical findings retain the best prognostic value approved by newest studies [16]. Two clinical criteria – absence of motor response and pupillary or corneal reflexes on day three – have been found to be almost 100% specific for poor prognosis [17]. Myoclonic status epilepticus is also usually associated with poor outcome but it should not be confused with multifocal myoclonus or generalized tonic-clonic seizures, which are not reliably useful for prognostication [18]. Further testing is only ancillary and is used when comorbid conditions are present. The value of N20 component in somatosensory evoked potentials as well as EEG assessment are still questionable due to high interobserver variability [19]. Two main biochemical markers – neuron-specific enolase and the glial S-100 protein – are associated with poor outcome and may help in a clinical trial setting [20]. Nowadays, due to advanced methods of resuscitation, evaluation of hypoxic-ischemic outcomes focuses on neuropsychological pattern, where the GCS is less important for prediction of good cognitive functions. C. P. Kaplan et al. found that recovery of recall memory during the first month after anoxic event may predict good cognitive functions [21]. Moreover, quantitative MRI 6 months after anoxic brain damage shows promising results in prediction of cognitive outcome after this type of injury [22, 23].

Data regarding patients with PPH who are admitted to ICU are scant, and articles usually analyze general obstetric population, so it may misrepresent the data concerning PPH. Literature presents some risk factors of bad outcome and adverse maternal morbidity after PPH, including late onset of DIC, blood transfusions, cardiac disease, other co-morbidities, etc. However, these studies are superficial and lack reports of patient outcomes subsequent to ICU admission [4, 24]. Furthermore, no prognostic factors have been reported analyzing patients' neurological outcomes after severe brain damage due to hypovolemia caused by

PPH. We presumed that this outcome could be reflected by the same prognostic factors, used for prognosis after cardiac attack, which through hypovolemia shows the similar pathogenetic pattern as PPH. According to several scales of worse or better prognosis, our observed three women had only a few factors of worse prognosis, despite the fact that the general and neurological condition of the patients was critically severe after PPH [25, 26]. Actually, A. Peskine et al. indicated that this type of injury presents no correlation between acute stage data, except the length of stay in the ICU, and the outcome [27].

OUTCOME. Improvement in resuscitation and life supporting techniques resulted in a greater number of patients with variable degree of hypoxic-ischemic brain injury determining higher morbidity. Possible neurological outcomes of anoxic-ischemic brain damage may vary ranging from brain death to relatively minor residual effects, but generally the prognosis is intermediate, compared to other causes of nontraumatic coma. This type of coma also presents a significantly increased frequency of permanent vegetative state as any other type of non-traumatic coma [28]. However, even if the impact of systemic hypoxia on the brain functions is not severe and neurological functions are not impaired dramatically, only a few patients avoid neurological dysfunction. If the structural damage of brain tissue is present, full recovery is unlikely. R. O. Hopkins et al. are one of the few who have reported a case of three individuals with sustained severe anoxia and chronic sequelae in a number of cognitive functions, where one of the patients returned to pre-injury levels of functioning [22].

Patients who survive coma make most of their improvement during the first month. The longer the coma lasts, the less are the chances of regaining independent functions [28]. Outcome of anoxic brain injury is often characterized by amnesic syndrome: severely impaired long term recall, intact short-term memory, proactive interference, and more or less depressed recognition of visual and verbal material [29, 30]. Amnesia in this case is most likely caused by bilateral hippocampus injury due to its high metabolic activity [9]. In mild cases of hypoxia, recollection (but not familiarity) is usually impaired, however, both types of recognition memory also can be affected [31]. In addition, almost all patients after anoxic brain injury present some sort of attention and self-regulation impairment (deficits in sustained attention and effort) [32]. Postanoxic axial motor disturbances (due to basal ganglia injury), extrapyramidal syndrome, dysarthria, dyspraxia, and agnosia are also mentioned in the literature [27, 33–36]. Generaly, a widespread cognitive deficit, behavioural frontal lobe and dysexecutive syndrome may appear after this type of injury, causing different levels of inability and patterns of impairments [10, 27, 36].

Obstetric patients are generally different from general population and often have peculiar outcomes after admission in ICU. Pregnant women are usually young and in good health, and possess lower mortality rate according to different scales and scores applied in ICU [37]. According to maternal morbidity outcome indicator (MMOI), PPH is the main factor predisposing maternal morbidity (28–56%) [38]. In literature, however, the outcome and morbidity usually represent a snapshot in time of the ICU admission with virtually no data on late-onset outcomes and postpartum follow-up [39]. Our findings are consistent with earlier observations of cognitive and psychosocial impairment of the patients after hypoxic-ischemic brain injury. Though the patients resumed a normal life with almost no pathological neurological findings after the event, they were found to have subjective complaints and deficits of memory and attention, which nevertheless affect the work and family relationships. Moreover, two patients showed significant signs of depression (2) or anxiety (1), and this correlated with decreased ACE-R^{LT} results.

In conclusion, although evaluating the prognosis of such patients may be challenging, our small observation shows that basic prognostication may be based on the other hypoxic-ischemic patterns. When talking about more specific and precise neuropsychological evaluation, there is no consensus, which could help outline more accurate prognosis. Prediction of neurological outcome is even more confusing due to heterogeneity of PPH. As a young part of the population, women after childbirth are important not only to relatives and their descendants, but also to the society due to socioeconomic factors. According to our observations, most PPH patients made notable improvement over an extended period of time. This may reflect the statements that women are better adapted to blood loss than men, and they may present better outcome than remaining population after hypoxic-ischemic brain injury due to their age and rare comorbidities.

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GERA NEUROLOGINĖ IŠEITIS PO POGIMDYMINIO NUKRAUJAVIMO SUKELTOS HIPOKSINĖS-IŠEMINĖS KOMOS: 3 KLINIKINIAI ATVEJAI IR LITERATŪROS APŽVALGA

Santrauka

Pogimdyminiu nukraujavimu laikomas kraujavimas, kai gimdyvė netenka 500 ml ar daugiau kraujo po natūralaus gimdymo arba daugiau nei 1000 ml po cezario pjūvio operacijos, ir dėl to sutrinka hemodinamika. Tai gali sąlygoti daugelį įvairių komplikacijų,

kurios gali turėti didelės reikšmės ne tik motinos, bet ir jos vaiko, šeimos gyvenimui, galima sociologinė ir ekonominė žala ir valstybei. Dėl pagerėjusios pradinio ir specialiojo gaivinimo kokybės svarbiausiomis ir aktualiausiomis tampa neurologinės komplikacijos, susijusios su hipoksija – hipoksinė išeminė koma ir hipoksinis smegenų pažeidimas. Šiame straipsnyje aptariame 3 moterų neurologines išeitis po sunkaus pogimdyminio nukraujavimo ir jo sukelto galvos smegenų pažeidimo. Diskusijose pateikiama literatūros, susijusios su šia problema, apžvalga. Duomenys apie buvusį nukraujavimą, gimdyvių bendrąją ir neurologinę būklę, galvos smegenų vaizdinių tyrimų radinius po jo buvo renkami retrospektyviai iš Vilniaus universiteto ligoninės (VUL) Santariškių klinikų ligos istorijų. Dabartinei būklei, subjektyviai savijautai ir galimoms atokioms pasekmėms įvertinti (praėjus vidutiniškai 35 mėn. po įvykio) moterys buvo telefonu pakviestos atvykti į VUL Santariškių klinikų neurologijos centrą. Dabartiniai nusiskundimai, neurologinė ir kognityvinė būklė, depresijos ir nerimo lygis buvo vertinami aktyviai klausiant, objektyviai tiriant, naudojant Adenbruko kognityvinio tyrimo taisytos metodikos adaptaciją lietuviškai kalbantiems gyventojams (ACE-R^{LT}), Ligoninės nerimo ir depresijos skalę (HADS). Visos trys moterys ūminiu periodu po nukraujavimo buvo kritiškai sunkios būklės, su dauginio organų nepakankamumo ir diseminuotos intravaskulinės koaguliacijos sindromų požymiais. Dviem iš jų aprašyti atitinkamai 4 ir 5 minučių trukmės asistolijos epizodai, trečios gimdyvės hemoglobino kiekis kraujyje po nukraujavimo tebuvo 15 g/l. Jų neurologinė būklė tiek kritiniu ūminiu, tiek sveikimo periodu buvo itin sunki – pasireiškė hipoksinė išeminė koma, delyras, ekstrapontinė mielinolizė, traukuliai, grįžtamasis užpakalinės leukoencefalopatijos sindromas, koordinacijos sutrikimai, galūnių raumenų hipertonija, sutriko psichosocialiniai gebėjimai. Moterų atoki neurologinė būklė buvo stebėtinai gera. Tai rodo geras potencialias galvos smegenų funkcijų atsistatymo po pogimdyminio nukraujavimo galimybes. Vienos moters ACE-R^{LT} rodiklis siekė maksimumą, kitų viršijo 89 balus iš 100. Visos grįžo į darbą, vairuoja automobilį, tačiau turi subjektyvių nusiskundimų įvairaus laipsnio atminties ir dėmesio sutrikimais. Dviem moterims buvo nustatytas reikšmingai padidėjęs depresiškumo, vienai jų – taip pat ir nerimastingumo lygis. Kadangi specialių metodų, pritaikytų išeitims po pogimdyminio nukraujavimo prognozuoti, neradome, retrospektyviai taikėme literatūroje plačiai apžvelgtus prognostinius veiksnius, naudojamus neurologinėms išeitims po širdies sustojimo numatyti. Jų rezultatai, mūsų atvejais, gana patikimai atitiko stebimas geras neurologines išeitis po išeminės hipoksinės komos dėl pogimdyminio nukraujavimo. Aprašyti atvejai pabrėžia bendradarbiavimo ir ligonių interdisciplininės priežiūros svarbą bei galimus gerus atokius rezultatus.

Raktažodžiai: pogimdyvinis nukraujavimas, hipoksinė-išeminė koma, smegenų pažeidimas, neurologinė išeitis.