CASE REPORT

RJME
Romanian Journal of
Morphology & Embryology
http://www.rjme.ro/

# Velamentous insertion of the umbilical cord vessels with *vasa praevia* – a case report

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## Abstract

Among the abnormal cord insertion pathology, velamentous cord insertion associated with rupture of *vasa praevia* is the most severe condition related to the outcome of the newborn. In velamentous cord insertion, the fetal vessels run freely through the fetal membranes without protection from Wharton's jelly, umbilical vessels diverging as they traverse the membranes. When the membranes are ruptured, complete tearing of fetal vessels through the torn membranes or partial rupture near the site of membrane rupture may occur. Velamentous insertion occurs in approximately 1% of singleton gestations, but is observed in as many as 15% of monochorionic twin gestations. The risk of perinatal death was doubled in pregnancies with velamentous cord insertion relative to normal cord insertion. This condition can be diagnosed by ultrasonography with a sensitivity of 67% and specificity of 100% in the second trimester. We report a case of a newborn who came from a velamentous cord insertion condition associated with rupture of *vasa praevia* after the spontaneously membranes rupture. After a difficult resuscitation and stabilization, the newborn survived with a good outcome after the follow-up.

**Keywords**: ruptured fetal vessel, velamentous umbilical cord insertion, fetal bleeding, newborn survival and outcome.

## Introduction

Velamentous cord insertion (VCI) is an abnormal cord insertion in which the umbilical vessels diverge as they traverse between the amnion and chorion before reaching the placenta. With a reported incidence of 1% in singleton pregnancies, it has been associated with several obstetric complications [1].

Environmental, genetic, multifactorial, and unknown causes can be involved in the genesis of this condition.

The disorder is associated with morbidity and mortality with a significantly reduced survival beyond neonatal period. Treatment is symptomatic and supportive, and requires a multidisciplinary management. Child outcome depends on the perinatal hypoxia severity and the medical and neurological complications associated.

Rarely, a *vasa praevia* is present, and is inevitably torn when the membranes rupture, leading to severe fetal blood loss. This can easily be missed because the volume of blood is relatively small in obstetric terms and it is not possible to distinguish fetal from maternal blood by eye. The incidence of *vasa praevia* is estimated to be around 1:2500 and the condition carries a high fetal mortality. This condition can be diagnosed by ultrasonography with a sensitivity of 67% and specificity of 100% in the second trimester [2]. The presence of *vasa praevia* can be detected antenatal with a color Doppler vaginal ultrasound but this is not routine practice; if

*vasa praevia* is found then the baby should be delivered by caesarean (C) section before membrane rupture [3].

Diagnosis is usually retrospective after fetal demise or fetal distress from an abnormal fetal heart rate (FHR) pattern on FHR monitoring and the condition is revealed by postpartum examination of the placenta [4].

For the newborn in this condition when hemorrhage occurs acutely during delivery, the hematocrit (Ht) will not fully reflect the degree of blood loss because there has been little hemodilution, but the neonatologist needs to manage the shock, paying close attention to the hemodynamic and cardiorespiratory parameters.

Almost invariably, newborn babies with hypovolemic shock will have experienced some degree of asphyxia, the manifestations of which will influence the assessment of shock [4].

### Aim

This report is on a rare and severe condition – velamentous cord insertion – which was not prenatally diagnosed and its impact on a newborn in respective of the perinatal and long-term outcome.

## Case report

We present a case of a 39-year-old healthy women, gravida VI, para III, of 36–37 weeks of low-risk gestation, who was admitted to our hospital for rare and unsystematic

contractions appeared in the last three days. At the admission, the vaginal examination reveals a dehiscent large cervix permitting a finger pulp with intact membranes. There were no other abnormalities found. Fetal heart rate was normal and continuously monitored. During the pregnancy, no maternal pathology was present and at the morphology test, 23 weeks revealed a healthy male fetus.

After eight hours of contractions, the vaginal examination indicated a shorted cervix, the spontaneously rupture of membranes and abundant metrorrhagia begun. Fetal heart rate begun to modify and the cardiotocography revealed atypical decelerations. Under this circumstances and considering both mother and child interests, the C-section was performed.

A 36–37 weeks premature male was delivered with cardiac arrest in "white" asphyxia. The newborn resuscitation started immediately with endotracheal intubation and positive pressure ventilation coordinated with external cardiac massage, with no response at one minute. The Apgar score was 0. The resuscitation team of neonatologists continued with umbilical vein catheterization and drugs administration: volume expanders, saline solutions, adrenaline and sodium carbonate, in parallel with cardiac massage and positive pressure ventilation. At 5 and 15 minutes, the Apgar score was still 0. At 33 minutes, the first heartbeat was audible and soon after, the first breath appeared. The baby was admitted in the neonatal intensive care unit (NICU) and received supportive treatment according to his severe status. Meantime monitoring the oxygen saturation, heart rate and capillary filling indicated $SaO_2$ (oxygen saturation) 80–86%, HR (heart rate) 109–126 beats/min., blood pressure was initially low then corrected with volume expanders. Astrup method was performed indicating a metabolic acidosis (pH 7.17), which was corrected about one hour and half; also, a marked anemia – Hb (hemoglobin) 9.1 mg/dL, Ht 27% – revealed. The emergency of OI negative Rh red blood cell transfusion was considered and started at two hours of life with 20 mL/kg/body weight.

Metabolic acidosis was revealed in the fist blood gas examination: pH 7.17, $pCO_2$ (partial pressure of carbon dioxide) 32 mmHg, $pO_2$ (partial pressure of oxygen) 86 mmHg, $HCO_3^-$ 15.4 mmol/L, BEecf (base excess in the extracellular fluid) -11.7, Hb 11.4 g/dL, Ht 32%, lactate 12 mmol/L. After two hours and correction of metabolic acidosis, the blood gas values reveals the marked anemia: pH 7.7, $pCO_2$ 35 mmHg, $pO_2$ 46 mmHg, $HCO_3^-$ 20.4 mmol/L, BEecf -2.7, Hb 9.1 g/dL, Ht 28%, lactate 6 mmol/L; when the OI Rh negative red blood cell transfusion started. After seven hours of supportive care in NICU, the blood gas was normal: pH 7.4, $pCO_2$ 35.4 mmHg, $pO_2$ 45 mmHg, $HCO_3^-$ 22.8 mmol/L, BEecf 2.7, Hb 12.4 g/dL, Ht 32%, lactate 3 mmol/L.

### Clinical examination and evolution

The physical examination after resuscitation and stabilization revealed a male late preterm with 36–37 gestational age (GA), birth date 11.07.2014, body weight (BW) 3350 g (90% percentile on growth chart), length (L) 50 cm (90% percentile on growth chart), cranial perimeter 35 cm (50% percentile), pale skin, cyanosis of extremities, HR between 118–136 beats/min., respiratory frequency 58 breaths/min., hypotonia, reduced archaic reflexes, seizures appeared at four hours from birth.

In evolution at six hours from birth, the skin became pink, without cyanosis, normal heart rate, normal blood pressure, normal breathing rate, without liver or splenomegaly, extremely irritable, the seizures persisting for 24 hours. He was stable and the neurophysical developmental curves were in normal range. He did not need any positive inotropic drug therapy. Diuresis was normal from the first day, the first stool – meconium after 24 hours. Parenteral nutrition was needed for the first two days and in the third day enteral nutrition was initiated with mother's milk bottle-fed. In the fifth day, the newborn was breastfed, being stable and adapted outside of the incubator. Treatment of seizures was performed with Phenobarbital; maternal-fetal transmitted infection was treated with antibiotics.

In the ninth day, neurological examination revealed a moderate cervical hypotonia, clinical examination showed a stable hemodynamic newborn with a good growth curve, breastfed, immunization was performed and all the discharge criteria were fulfilled. He was discharged at nine days with 3220 g weight in good condition.

### Head ultrasonography

A head ultrasonography was performed in the third and in the ninth day during the hospital stay, using a low frequency transducer, which revealed cerebral edema (Figure 1, a and b).

Performing in dynamic at one month and at three months of life, the head ultrasound (US) did not reveal any severe pathological images like leukomalacia or cysts in the cerebral structure (Figures 2 and 3).

### Neurological outcome

Neurological examinations in follow-up did not reveal any delay in neurological development.

At two months, the reflexes were normal; a clonus of menthon and right leg was discovered without pathological significance, which disappeared at four months examination. Ophthalmologic examination in follow-up did not find any pathological issues.

### Histopathological examination of the cord blood and placenta

#### *Macroscopic examination*

Macroscopic examination revealed velamentous cord insertion with ruptured *vasa praevia*, stasis and thrombosis; marginal *placenta praevia* (Figure 4).

#### *Microscopic examination*

Umbilical cord: dissecting edema in the conjunctive tissue, umbilical vessels with stasis, thrombosis and ischemia (Figure 5). Chorial membranes: hypertrophy, marked dystrophic lesions of chorion (Figure 6), edema under the amniotic epithelium here and there dissecting (Figure 7). Placenta: subchorial fibrinoid blade and hematic clots involution of decidua with hemorrhagic necrosis and necrobiosis (Figure 8). Numerous monotrophoblastic placental villi predominant with small dimensions with rare calcar deposits were present. Fibrinoid deposits inside intermediary villi; necrosis zones of villi (Figure 9).
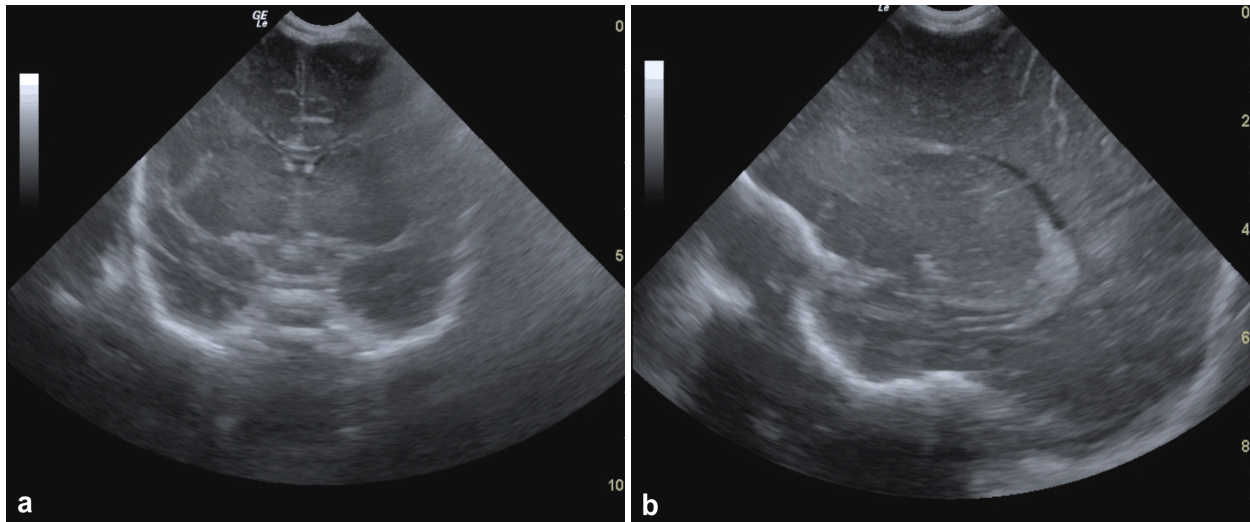
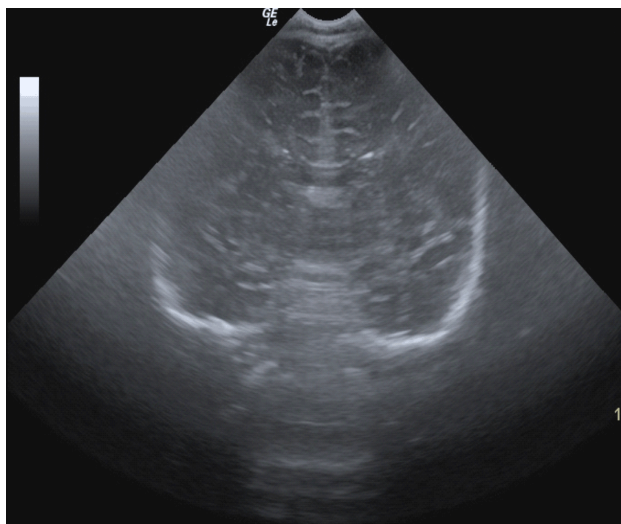**Figure 1 –** *(a and b) Head ultrasound day 3: cerebral edema.*



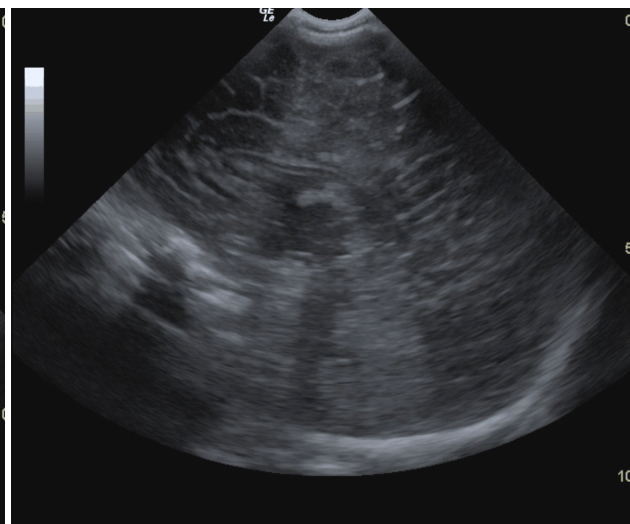**Figure 2 –** *Head US at three months. Coronal incidence: normal view.*



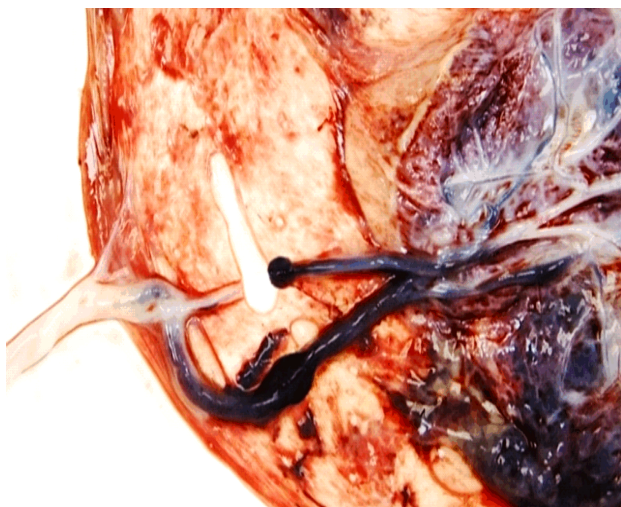**Figure 3 –** *Head US at three months. Sagital incidence: normal view.*



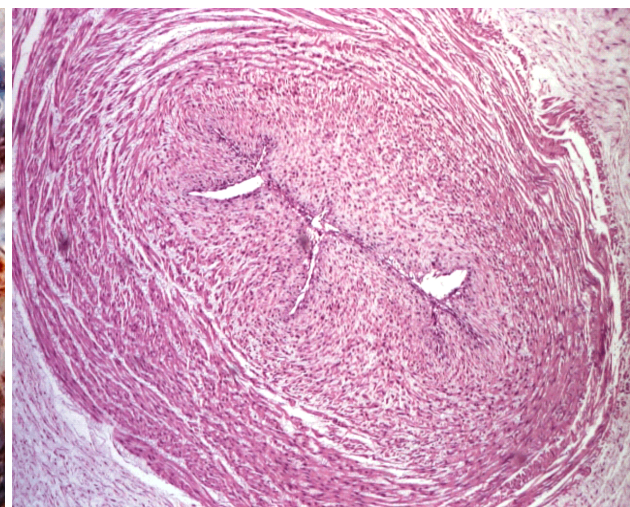**Figure 4 –** *Velamentous insertion of umbilical cord vessels.*



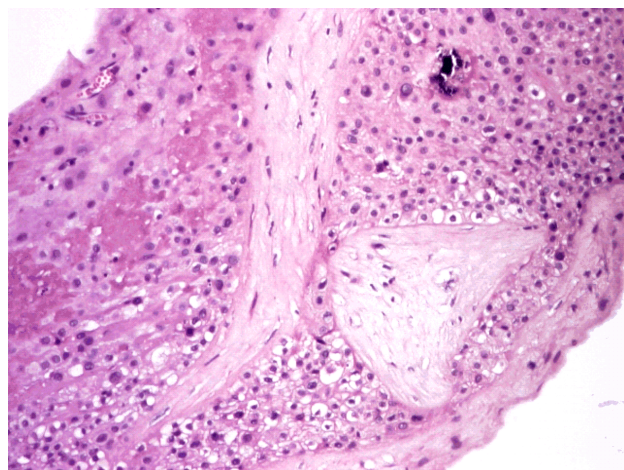**Figure 5 –** *Umbilical cord: ischemia (HE staining, ×40).*

**Figure 6 – *Hypertrophy, marked dystrophic lesions of membranes (HE staining, ×100).***
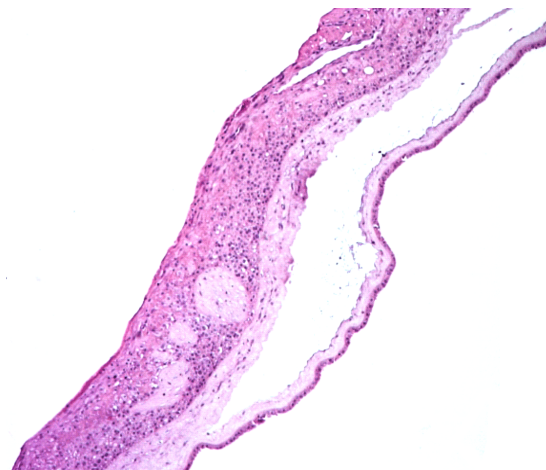


**Figure 7 – *Edema under the amniotic epithelium dissecting membranes (HE staining, ×40).***
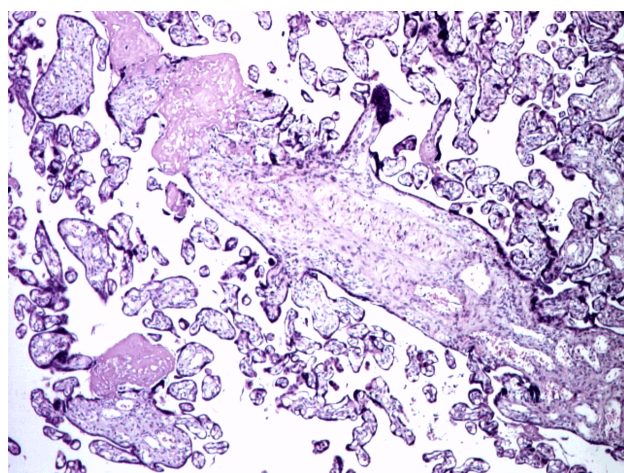


**Figure 8 – *Placenta – subchorial fibrinoid blade and hematic clots involution of decidua with hemorrhagic necrosis and necrobiosis (HE staining, ×40).***
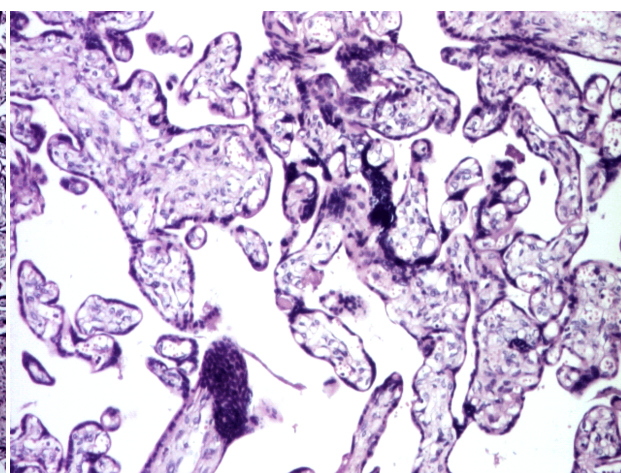


**Figure 9 – *Placenta – necrosis zones of villi (HE staining, ×100).***

## ⊡ Discussion

In a population-based study (Ebbing *et al.*, 2013) found prevalence rates of 1.5% and 6.3% for velamentous and marginal cord insertions, respectively, in singleton pregnancies, and higher rates in multiple pregnancies. The results suggest that the two conditions are closely related and have common risk factors, but in a graded fashion, with the velamentous insertion being the more-severe condition. Abnormal insertion of the cord triples the risk of perinatal death at term, which may justify an increased focus during pregnancy to identify this condition in order to better prepare for the care provided at term [5].

The study confirms the previous finding of increased risk of preterm birth in pregnancies with anomalous cord insertion, whereas the risk of pre-eclampsia was only increased in the term group. The increased risk of emergency C-delivery for pregnancies with velamentous or marginal insertion of the cord – in view of the unchanged risk of operative vaginal delivery – suggests that abnormal cord insertion is likely to lead to inter-vention before the second stage of labor.

Velamentous vessels are associated with *vasa praevia* (where the vessels traverse the internal bone of the cervix in front of the leading fetal part), a condition associated with high perinatal mortality when it is not prenatally diagnosed [6].

Because of the lack of protection from Wharton's jelly, these vessels are prone to compression, thrombosis and rupture, especially when they are located in the membranes covering the cervical ostium (*vasa praevia*). The length of the membranous vessels or the distance between the end of the normal cord and the placental insertion is highly variable [7].

In our case, the histopathological result describes the umbilical vessels with stasis, ruptures and thrombosis.

Variable deceleration without baroreceptor-mediated acceleration (VDna) frequently occurs in VCI cases. The cause of frequent and early appearance of VDna is thought to be the compression of aberrant vessels, which are not coated or thinly coated with Wharton's jelly, and the blood flow of both umbilical arteries and veins would be obstructed at the same time during uterine contractions or fetal movement [8]. Analysis of VDna occurrence in the second stage of labor shows that the rate of VDna is about three times higher in VCI cases than in controls [8].

Clark [8] reported a similar case with intact membranes, without delivery labor, where the induction of labor was initiated with oxytocin. Hasegawa *et al.* [9], in a case

study, found that there is a relation between variable deceleration and uterine contractions in labor at patients with velamentous insertion diagnosed by ultrasound exam and considered that the rising of amnion pressure during the contractions can compress simultaneously the umbilical vein and the artery causing the decelerations.

In our case report, no variable decelerations were found, the uterine contractions were rare and unorganized and the labor begun.

Velamentous insertion of the umbilical cord has been associated with an increased risk of adverse perinatal outcomes [10–12] and associated with *vasa praevia* (where the vessels traverse the internal ostium of the cervix in front of the leading fetal part), a condition that is associated with high perinatal mortality when it is not diagnosed prenatal. Despite the advances in perinatal medicine, approximately 2% of low-risk pregnant women still require an emergency cesarean section after the onset of labor. Surviving neonates born to women without prenatal diagnosis required blood transfusions [13].

### Newborn outcome

The outcome of infants sustaining cerebral hypoxia-ischemia is influenced by several factors, including the duration and severity of the insult to the brain; gestational age; presence of seizures; and associated infectious, metabolic, and traumatic derangements. Although the prognosis for any single newborn often is difficult to formulate, certain clinical and laboratory abnormalities, along with perinatal cerebral hypoxia-ischemia, are associated with a high risk of neurologic morbidity [1].

When hemorrhage occurs acutely during delivery, the hematocrit will not fully reflect the degree of blood loss because there has been little hemodilution. In this situation, one will need to aggressively manage shock, paying close attention to the hemodynamic and cardio-respiratory parameters of the baby. Measures of metabolic acidosis, capillary filling time, and both arterial and/ or central venous pressures are important to monitor, because they will guide the approach and extent of fluid resuscitation. Almost invariably, newborn babies with hypovolemic shock will have experienced some degree of asphyxia, the manifestations of which will influence the assessment of shock. Thus, one must consider this to be an important variable, and it needs rapid attention, so that the treatment of shock must not delay correction of asphyxia [4]. Acute situations may include *placenta praevia*, *vasa praevia*, abruption, or blood loss from the cut umbilical cord. In such cases, immediate volume replacement with blood is preferable because this rapidly enhances oxygen delivery to tissues, which is not the case when crystalloid solutions are used. Anticipation of the need for resuscitation is a key factor, so recognition of maternal vaginal bleeding should be a signal to anticipate for the need for transfusion. The classic approach to shock in the newborn is to transfuse 10 mL/kg of blood over five to 10 minutes and to repeat infusions until there are signs of adequate circulation [4].

Infants who were apneic for 30 or more minutes were universally and severely damaged. The data suggest that a prolonged delay in the initiation of spontaneous respirations is a reasonable indicator of irreversible brain damage due to asphyxia.

In the reported case, the period of cardiac arrest was 33 minutes and according to statistics, our newborn had a bad prognostic: highest chances of cerebral palsy.

The term asphyxia, from the Greek word for suffocation, is used to describe the interrupted supply of oxygen through the placenta and umbilical cord to the fetus. This will lead to a combined hypoxemia and hypercapnia [1].

In case of total interruption of oxygen, within minutes, anaerobic glycolysis will occur and a lactic acidosis, and thereby metabolic acidosis, will be produced. This can be measured by blood gas analysis. In addition, (fetal) bradycardia will develop, which will add ischemia to the process.

The blood gas could not be performed because we could not obtain any blood sample. After the resuscitation with saline solutions and bicarbonate solution, the values indicated a severe metabolic acidosis with very low value of pH and bicarbonate and high values of lactate. The patient was referred to neonatal intensive care unit where the process of stabilization was continued.

As the hypoxic insult becomes more severe, changes in regional cerebral blood flow occur. The brainstem is able to extract sufficient oxygen to maintain metabolism despite very low $PaO_2$ (partial pressure of oxygen) at the expense of the cerebrum. Failing myocardial function may cause a fall in cardiac output, and the watershed areas of the cerebral hemispheres are most exposed to damage [1].

An infant with acute blood loss may not be anemic if blood sampling is done soon enough after the acute event so that hemodilution has not yet occurred. Anemia usually develops within three to four hours after blood loss; repeat testing six to 12 hours after the event should reveal the true extent of the loss. In this case, anemia appeared at two hours when the Hb value was 9.1 g/dL and Ht 28% and the OI Rh negative red blood cell transfusion started.

### Resuscitation

The key to resuscitation is to store adequate oxygenation and perfusion of vital organs, particularly the brain. Systemic acidosis developing because of intrapartum asphyxia impairs cardiac contractility, but its effect on cerebral function is less clearly understood. Infants may be born in unexpectedly poor condition and require immediate resuscitation. The importance of expert resuscitation has been recognized, and every infant, wherever born, must have personnel available with expert resuscitation skills and all the appropriate equipment in good working order.

In our case, the neonatologist team is trained in NRP (Neonatal Resuscitation Program) and S.T.A.B.L.E. (Sugar, Temperature, Airway, Blood pressure, Lab work, and Emotional support); some of them are lead instructors. The coordinated teamwork resuscitation and stabilization was by far the most important point in the good neurological outcome of the baby after the delivery. Because of the lack of antenatal diagnosis of the condition, the neonatologists were in front of a difficult and prolonged. The resuscitation protocol of a newborn in cardiac arrest for 33 minutes was followed step by step.

The question was: Why the response of the procedures was delayed? After a 16 mL/kg of saline solution, 12 mL/kg of 4.2% solution of bicarbonate, adrenaline administrated by umbilical catheter, intubated and ventilated with Neo Puff in coordination with cardiac massage the fist heartbeat was present 33 minutes from delivery, and soon after the first breath. At this point, the Astrup exam still reveals metabolic acidosis (pH 7.17) and anemia (Hb 11.4 g/dL, Ht 32%).

Meantime the placenta was delivered and the velamentous cord insertion with ruptured *vasa praevia* was observed, explaining the bad condition of the newborn.

After two hours and correction of acidosis, the anemia was marked at Hb 9.1 g/dL, Ht 28%, and the emergency transfusion with 20 mL/kg, OI Rh negative, red blood cell started.

The diving reflex occurs during experimental asphyxia to maintain blood flow to vital organs such as the brain at systemic complications after a clinically significant hypoxic-ischemic insult, and the heart, kidneys, and liver are the most vulnerable organs. Almost all babies with hypoxic-ischemic encephalopathy show compromise in at least one organ system outside the brain [14, 15]. The clinical course after a hypoxic-ischemic event in labor is unpredictable, so problems should be anticipated and the infant appropriately observed by trained staff in a clinical environment. Hypoxic-ischemic insult affects the whole organism, and any organ system may be compromised [4].

### Renal system

Renal impairment is reported to occur in 23% to 70% of asphyxiated infants [15]. Acute renal failure (plasma creatinine greater than 130 μmol/L [1.2 mg/dL] for at least two consecutive days) is reported in 19% of asphyxiated infants of greater than 33 weeks' gestation.

Our renal status in the first day was with acute renal failure with creatinine 160 μmol/L (1.4 mg/dL), which normalized in the second day.

### Cardiovascular system

Myocardial contractility is impaired after hypoxic-ischemic insult, causing a significant reduction in cardiac output, hypotension, and further impairment of cerebral blood flow and perfusion of other organs. Myocardial dysfunction has been shown to occur in many asphyxiated newborn infants [14]. Other cardiac pathologic processes recognized to occur after a severe hypoxic-ischemic insult include acute cardiac dilation with functional tricuspid valve regurgitation, myocardial ischemia present on ECG assessment, and ischemic necrosis of papillary muscle. Cardiac biomarkers as a marker for perinatal myocardial damage are currently under investigation [16]. Hypotension is a common complication after severe hypoxic-ischemic insult, and volume support is often ineffective in restoring normal pressure. The inotrope dopamine (5 to 15 μg/kg/min.) has been shown to be more effective in restoring blood pressure in asphyxiated infants [4]. After stabilization of the newborn, the blood pressure was normal and being in normal range in dynamic.

### Hepatic and gastrointestinal systems

Elevated liver enzymes occur in 80% to 85% of asphyxiated full-term newborn infants during the first week of life [15]. Our patient had the liver enzymes at the highest level of normal range, without any liver damage.

### Hematological system

Coagulation impairment is relatively common after a severe asphyxial insult. The bone marrow may be transiently damaged, leading to thrombocytopenia [4]. In our case, the first level of thrombocytes was 156 000/mm$^3$, in third day decreased at 76 000/mm$^3$, in the seventh day being normal (324 000/mm$^3$).

Disseminated intravascular coagulation is also well recognized to occur after birth asphyxia, with low levels of factor XIII and elevated thrombin-antithrombin complexes; D-dimer, fibrin, and fibrinogen degradation products; and soluble fibrin monomer complexes [4]. Coagulation impairment should be anticipated and screening for hematological abnormalities undertaken in all severely asphyxiated newborn infants. Supportive treatment with platelets, vitamin K, or clotting factors may be indicated by specific abnormalities on the coagulation screen.

## Brain-oriented management

Careful attention must be paid to maintain cerebral homeostasis by anticipation of complications that may have a direct or indirect effect on brain function and recovery after a severe hypoxic-ischemic insult.

### Glucose

The answer on the clinical question of whether glucose infusion after hypoxic-ischemic insult is of benefit remains controversial.

Although routine use of high-concentration glucose is not advised after a hypoxic-ischemic event in the neonate, it is clear that hypoglycemia must be avoided. In addition, insulin levels are elevated in the hours after perinatal asphyxia, when stress hormones tend to increase and the chance of hypoglycemia is considerable [17]. Regular blood glucose testing in neonates after resuscitation is of utmost importance. Glucose levels in our patient were in a normal curve in dynamics with a lower point at 55 mg/dL at two hours after birth.

### Seizures

Seizures occur in many infants who have sustained a significant hypoxic-ischemic insult; indeed, seizure is a feature of moderate and severe hypoxic-ischemic encephalopathy. Generally, the more severe or prolonged the hypoxia-ischemia, the more seizure activity the infant presents. There has been considerable debate as to whether seizure activity after a hypoxic-ischemic event confers an additional risk factor on the infant in terms of adverse neurodevelopmental outcome. Recent studies have demonstrated adverse effects of repetitive seizures on the developing brain, and these should be treated, even if subclinical and demonstrated only using EEG techniques [18, 19]. In our case, seizures were present at four hours from birth and lasted only for one day, disappearing after Phenobarbital treatment.

### Cerebral edema

Cerebral edema occurs commonly after a severe hypoxic-ischemic insult. Studies of intracranial pressure

monitoring in human infants who have suffered a severe hypoxic-ischemic insult have shown that severely raised intracranial pressure (greater than 15 cmH$_2$O) was found in a minority of infants monitored by a median time of 26 hours. In this group of asphyxiated infants with raised intracranial pressure, successful treatment of the intracranial hypertension as judged by a significant benefit on outcome was estimated to have occurred in less than 10% of cases.

There is no evidence that routine monitoring of intracranial pressure is of benefit to the infant. Raised intracranial pressure is most probably an end result of the abnormal pathophysiological processes that occur after hypoxic-ischemic insult and in itself is a marker of damage rather than a cause of it. Consequently, the management of raised intracranial pressure becomes relatively less important.

Ultrasound examination revealed cerebral edema, which was resolved at the seventh day ultrasound examination, without any pathological images of brain hypoxic ischemic insult such as leukomalacia or cysts.

### Apgar scores and condition at birth

Several studies have demonstrated the low sensitivity of the 1- and 5-minute Apgar scores in predicting long-term neurological outcome. However, Nelson *et al.* evaluated 39 full-term infants whose 20-minute Apgar scores were 3 or less; six died in the early postnatal period, and of the 14 survivors, eight exhibited cerebral palsy when examined at seven years of age [20]. Thus, the extended Apgar score is a reliable predictor of ultimate neurological morbidity, especially when very low scores are obtained at 20 minutes [21]. The time from birth until onset of spontaneous, sustained respirations also has been correlated with long-term neurological function. Full-term infants who were apneic for 30 or more minutes were universally and severely damaged [1]. The data suggest that a prolonged delay in the initiation of spontaneous respirations is a reasonable indicator of irreversible brain damage.

In spite of 33 minutes of apneic period and cardiac arrest, the baby survived, and his condition and good acquisitions at follow-up were impressive. Neurological follow-up examinations did not reveal any delay in neurological development to date.

### Acidosis

In the study of Goldaber *et al.* [22], 2.5% of 3506 full-term newborns exhibited an umbilical artery pH of less than 7.00, 66.7% of whom had a metabolic component to their acidosis. Significantly, more of the severely acidotic newborns exhibited low (less than 3) 1- and 5-minute Apgar scores compared with infants with higher umbilical artery pH values. In addition, neonatal death was significantly more frequent in the severely acidotic group. Low *et al.* compared 59 full-term fetuses exhibiting metabolic acidosis with 59 fetuses with normal umbilical blood acid-base status and 51 fetuses exhibiting only a respiratory acidosis at birth [23]. Various complications, including encephalopathy, were apparent in the majority of newborns experiencing a metabolic acidosis, compared with very low complication rates in those infants with either respiratory acidosis or no acidosis at all. In addition, Low *et al.* had previously demonstrated a positive correlation between the severity and duration of intrapartum metabolic acidosis and neurodevelopmental outcome at one year [23].

Our patient's metabolic acidosis at birth was marked, much lower than 7.00, since the first Astrup values, following the resuscitation with saline solutions and bicarbonate solution, revealed a 7.17 pH value. After seven hours of supportive care in NICU, the blood gas was normal.

### ⮐ Conclusions

In spite of the worst prognostic of an apneic newborn for more than 30 minutes, this baby survived with a good neurodevelopmental outcome, after 33 minutes of cardiac arrest and apnea. The follow-up of this high-risk newborn revealed normal neurological, ophthalmologic and physical development at the age of five months. The ultrasound examination performed in dynamics revealed cerebral edema after birth but no other pathological images in evolution. Velamentous cord insertion is associated with morbidity and mortality with a significantly reduced survival beyond neonatal period in cases that were not prenatally diagnosed. In this case, the coordinated teamwork resuscitation and stabilization was the most important point in the good neurological outcome of the newborn.

#### Conflict of interests

The authors declare that they have no conflict of interests.

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