REVIEW

Obstetric shock and shock in obstetrics – steady obstetrical syndrome

Anis Cerovac[1,2], Dubravko Habek[3,4], Elmedina Cerovac[2,5], Jasna Čerkez Habek[3,4]

[1]General Hospital Tešanj, Department of Gynaecology and Obstetrics, Bosnia and Herzegovina, [2]University of Tuzla, School of Medicine, Bosnia and Herzegovina, [3]University Department of Gynaecology and Obstetrics, Clinical Hospital, „Sveti Duh" Zagreb, Croatia, [4]School of Medicine, Croatian Catholic University Zagreb, Croatia, [5]General Hospital Tešanj, Department of Anaesthesiology, Reanimatology and Intensive Medicine, Bosnia and Herzegovina

ABSTRACT

Obstetric shock (OS) has been defined as a life-threatening cardiovascular collapse syndrome associated with pregnancy, childbirth and puerperium (obstetrics causes), and is the most significant cause of high maternal mortality (MM) throughout human history. Shock in obstetrics (SIO) refers to indirect causes of non-obstetrics causes in pregnancy, childbirth and puerperium (polytrauma, aesthetic incidents, cardiovascular or cerebrovascular incidents, other septic syndromes). The goals of OS treatment are: to quickly detect the location or cause of bleeding / injury / inflammation, prevent the progression of shock, prevent massive transfusions, preserve the uterus (and adnexa), and preserve fertility if possible. Surgical treatment of septic shock includes exploratory laparotomy (laparoscopy), ectomy or resection of the necrotized organ, abdominal lavage with multiple drainages, continuous peritoneal drainage with lavation, extensive triple antibiosis per admission or per antibiogram and thromboprophylaxis. OS seems to remain a permanent miasma in practical clinical obstetrics, which we will not be able to influence, because we have obviously caused today's increase in MM from haemorrhagic OS by iatrogenic increase in the number of caesarean sections, especially elective ones.

Key words: Caesarean section, circulatory collapse, maternal mortality, pregnancy

Corresponding author:
Anis Cerovac
General Hospital Tešanj
Braće Pobrić 17, 74260 Tešanj,
Bosnia and Herzegovina
Phone: +387 32 650 662;
Fax: +387 32 650 605;
E-mail: cerovac.anis@gmail.com
ORCID ID: https://orcid.org/0000-0002-7209-382X

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INTRODUCTION

Obstetric shock (OS) has been defined as a life-threatening cardiovascular collapse syndrome associated with pregnancy, childbirth, and puerperium (obstetrics causes) and is the most significant cause of high maternal mortality (MM) throughout human history (1-6). It is directly related to events in the perinatal period as a biohumoral altered immune and histogenetic gestational response of the organism, while shock in obstetrics (SIO) refers to indirect non-obstetrics causes in pregnancy, childbirth and puerperium (polytrauma, anaesthetic incidents, cardiovascular or cerebrovascular incidents, other septic syndromes) (1-6).

Although in recent literature the term OS syndrome by topic is not mentioned in many articles, in the Pubmed database there are about 2000 articles in the nomenclature of various forms of SIO (5,6), which include OS in a narrower sense and thus create possible confusion between these two terms, which should be defined separately.

HISTORICAL ASPECTS OF OBSTETRICS SHOCK

The clinical state of shock and premortal condition was described by ancient physician Hippocrates (facies Hippocratica). Clowes described the severe condition of the organism in 1588, explaining it with the presence of a foreign body in the blood (4,5). In 1784 Hunter associated the difficult situation with the then surgical procedures and trauma, and in 1795 Latta introduced the term "shock" which he interpreted it as a consequence of a surgical condition and injury, and was therefore also called "surgical shock" (4,5). During the 19th century, various theories about the onset of shock based on the rejection of humoral and the acceptance of cellular theory in the onset of disease emerged. Between the two world wars the theory was developed that the shock was a condition of circulatory (cardiac or vasomotor) deficiency characterized by reduced blood volume, cardiac output (reduced volume flow) and hyperconcentration of the blood and thus lay the basis for various etiopathogenetic forms of shock, not just the resulting trauma and blood loss (4,5). This definition of shock will remain today: inadequate acute or per acute generalized tissue perfusion of the microcirculation with oxygen depletion and metabolite accumulation is generally accepted modern definition of shock (4,5).

Interestingly, the causes of OS throughout the history of obstetrics have not changed despite the development of medical biotechnology. These are obstetric haemorrhage, preeclampsia (toxemia), sepsis, embolism, and maternal comorbidity, and starvation and fear as a result of war and economic crises are cited as risk factors for poor perinatal outcome and increased MM over the centuries (1,7).

Thus Brown cites OS in 16.3% of causes of maternal deaths from 1927-1936 in Belfast Maternity Hospital and Royal Maternity Hospital (2), which is confirmed by Say et al. (7). They presented the causes of maternal deaths: obstetric haemorrhage in 21%, preeclampsia 14%, sepsis 10%, abortion 7.9%, and embolism 3% as direct causes of OS death. The MM in the UK was 10.5% from 1700-1750, 7.5% from 1750-1800 and 5.0% from 1800-1850 (8).

ETHIOLOGY OF OBSTETRIC SHOCK AND SHOCK IN OBSTETRICS

Similar data are from own research on MM in the 18th and 19th centuries in which bleeding, uterine rupture, hydrops (preeclampsia), septic or criminal abortion, and puerperal sepsis with high perinatal mortality of up to about 50% are cited as causes of MM (9). All these conditions result in a severe OS syndrome even at that time, unfortunately without adequate therapy and procedures (9). Current urban lifestyle, traffic trauma, increased incidence of non-communicable chronic diseases, older life expectancy of pregnant women and mothers with comorbidities, repeated surgical interventions in childbirth, especially the enormous increase in caesarean sections are today's factors in the development of major obstetric syndromes, and thus an elevated total MM (4). Given the etiopathogenetic features of OS and SIO and, in fact, obstetric evolutionarily unchanged causes of death caused by severe forms of OS and SIO, this review represents contribution to today's constant and unchanging obstetric problem.

ETIOPATHOGENESIS AND PATHOPHYSIOLOGICAL MECANISMS OF OBSTETRIC SHOCK

Gestational adaptive changes in the cardiovascular (haemodilution, hypercoagulability, hypervolemia), digestive and respiratory systems, changes in intermediate metabolism, and the influence of

specific new temporary biochemically aggressive tissue, trophoblasts, with numerous metabolic, haemostatic and bio humoral changes are silent risk factors for possible etiopathogenetic triggers for the development of major obstetric syndromes (2,4). As much as pregnancy was compensated by mechanisms, it is actually paradoxically the cause of the typical OS syndrome in disturbed homeostasis or sudden external events in SIO (2,4, 10-15).

Reduction of uterine circulation to less than 100 mL/kg TT/min directly correlates with decreased foetal oxygen saturation, development of anaerobic glycolysis, and lactic acidosis with the development of foetal shock syndrome (2,4, 10-15). In case of septic syndrome, a foetal inflammatory response will develop according to the severity of the mother's inflammatory response and cytokinemia, ranging from intrauterine death to survival with various consequences of systemic or focal inflammatory disease, hypoxic-ischemic encephalopathy, organopathy, to permanent disability due to neuromotor damage (2,4, 10-15).

There are four basic etiopathogenetic factors of OS or SOI: hypovolemia (loss of intravascular volume due to loss outside of the body or redistribution), cardiogenic causes, circulatory obstruction (embolisms, cardiac tamponade, tension pneumothorax) and distribution (sepsis, anaphylactic shock, neurogenic shock) (2,4, 10-15).

Given the etiopathogenesis and pathophysiological mechanisms, there is more division of OS and SIO, and some forms can be complicated by other forms of shock (Table 1) (4).

Hypovolemic and cardiogenic shock

In a broader sense, hypovolemic shock is most often due to loss of circulating blood volume (haemorrhagic shock) as a secondary complication of severe obstetric bleeding (in >90%) or non-haemorrhagic events with volume loss (in <10%). Cardiogenic shock is a condition of hypoperfusion of target organs due to heart failure, and mechanical factors involved in filling or emptying the heart or large vessels explain ob-

Table 1. Modified classification of obstetric shock and shock in obstetrics (4)

Pathophysiology shock classification	Obstetrics shock	Shock in obstetrics
Hypovolemic shock		
1. haemorrhagic shock	Hemoperitoneum with / or massive vaginal bleeding Ruptured ectopic pregnancy Antenatal, periportal and postpartum haemorrhage Uterine rupture *Placenta previa* Morbid invasive malplacentation Placental abruption Obstetrics coagulopathy	Intraabdominal non-traumatic and traumatic haemorrhage Ruptured vessels and organs (liver, spleen), ruptured aortal aneurysm Non-traumatic or traumatic haemothorax ruptured aortal aneurysm, pulmonal and cardiac penetrations injuries Limb's injuries (traumatic amputation, vessels lesion) Pre-existent coagulopathy
2. hypovolemic non-haemorrhagic shock	Gestational hyperemesis, rapid loss of amniotic fluid	Severe diarrhoea, severe burns, excessive gastric suction, ileus, peritonitis, persistent febrility with dehydration
Septic shock (endotoxic, bacteriemic, viremic shock)	Obstetric sepsis Pan metritis (amnio infections syndrome) Puerperal sepsis Toxic shock syndrome Postoperative obstetrics septic complications (post caesarean peritonitis, uterine dehiscence)	No obstetrics severe infection (meningitis, pneumonia, peritonitis, pyothorax) Postoperative non-obstetrics septic complications (peritonitis)
Cardiac and cardiogenic shock	Peripartum dilatative decompensated cardiomyopathy	Traumatic cardiogenic shock with hemopericardia and cardiac tamponade Acute myocardial infarction
Traumatic shock	Uterine rupture Uterine avulsion	Severe truncal and limb injuries, polytrauma
Anaphylactic and anaphylactoid shock	Amniotic fluid embolism (fetal debris trophoblast)	Severe alergic reaction-anaphylactic shock
Neurogenic shock	Uterine inversion, uterine rupture, uterine avulsion, severe pain	Spinal shock, Spinal cord injuries
Metabolic and endocrinologic shock	Unregulated gestational diabetes or diabetes in pregnancy (ketoacidosis comma, hypoglycaemic comma, hyperosmolar comma)	Acute adrenal cryse, severe hypoglycaemia, hyperthyreotic/ hypothyreotic comma, uraemia

structive shock (acute coronary syndrome, onset or worsening of cardiomyopathy, peracute consequences of pulmonary embolism) (2,4, 23).

Obstetrics embolisms

Obstetric embolisms are referred to as conditions that cause the so-called embolic, obstructive shock as a typical OS, and cardiac tamponade, which is the cardiac cause of cardiogenic shock, among the causes of SIO. However, amniotic fluid embolism (AFE) is considered an anaphylactic obstetric syndrome with an incidence of 1: 7000–60.000 births and a maternal mortality of as high as 86% due to the occurrence of fulminant hyperfibrinolysis (2,4,5, 16-22).

Anaphylactic and septic obstetric shock

Anaphylactic shock in pregnancy and childbirth belongs to SIO due to hyperreactivity to antigenic components in drugs, exogenous toxins, bee or wasp stings (17,24). Septic OS most often occurs in septic abortion, chorioamnionitis, puerperal infection 1-2% after vaginal birth and 30-85% after caesarean section, and SOI in other non-obstetric inflammatory conditions with bacteraemia (pneumonia, urosepsis, abscesses, peritonitis). Bacteraemia will be found in 8-10% of cases with puerperal infection, and septic shock will occur in 4-12% with proven bacteraemia (2,4,5, 16-24).

Trauma and traumatic shock in obstetrics

Trauma and traumatic SIO are the leading non-obstetric causes of maternal mortality, and in the U.S. they are experienced by 5-8% of women during pregnancy (most common traffic accidents with polytrauma and penetrating injuries, intoxications and burns, domestic and sexual violence, falls, homicides, and suicides). Uterine rupture may have the characteristics of traumatic haemorrhagic shock (2,4,17,25), and acute abdominal syndrome is the most common sign of sudden intra-abdominal events with the development of haemorrhage of obstetric or non-obstetric genesis (e.g. rupture of abdominal aneurysm) or development of OS or SIO (2,4,25).

Distributive shock

Distributive shock is referred to as a condition of secondary venous pool filling such as early septic shock, anaphylaxis, peritonitis, and neurogenic shock, whether caused by an obstetric or non-obstetric cause. Special forms of primary non-haemorrhagic OS that can be caused by amniotic fluid embolism (AFE), trophoblast or thromboembolism, uterine inversion and sepsis (neurogenic, septic, anaphylactoid shock) (2,4,11,14,15, 19-22). Older nomenclatures called non-haemorrhagic shock "obstetric shock syndrome" because of its specific clinical picture and etiology, without bleeding. Thus, distributive neurogenic OS represents a state of generalized vasodilation with relative hypovolemia due to an imbalance of sympathetic and parasympathetic regulation of vasodilation (11-13).

CLASSIFICATION OF OBSTETRIC SHOCK

Numerous old-date studies have pointed to the problems of cardiocirculatory collapse and shock in childbirth. The therapeutic approach and classification of OS and SIO have changed throughout history (1,3,5,6).

The current International Classification of Diseases (ICD) classifies OS under codes: O 75.1, shock after abortion, ectopic pregnancy and molar pregnancy, O 80.3 circulatory collapse and postoperative shock, obstetric pyemic and septic embolism O 88.3, labour sepsis O 75.3, and O 88 types of obstetric embolism, while other forms of shock, especially SIO classifies as separate entities in other groups (e.g. cardiogenic shock, traumatic shock due to polytrauma) (Table 1) (26).

Vasocentralization, coagulation disorder, precapillary vasoconstriction with consequent vasoparalysis and tissue hypoxia and cell necrosis, anaerobic glycolysis with acidosis, activation of complement mediator and kallikrein-kinin system with endothelial lesions with consequent multiorgan failure are characterized by events in shock (2-6). These events are responsible for the current condition of the pregnant woman, parturient or puerpera as well as for the condition of the foetus, which directly correlates with short-term and long-term morbidity and high mortality. Thus, according to various authors, the total MM due to OS is 30-100%, and the percentage and ratio of individual causes of MM has not actually changed for centuries compared to the total MM (2-6).

The classification of certain forms of OS has changed on the basis of modern knowledge, so

AFE has been placed in a severe anaphylactoid reaction, while earlier nomenclatures have attached it to obstructive vascular shock. But Adams et al. classified AFE in the so-called traumatic-haemorrhagic shock due to, for example, sudden placental abruption and secondary infusion of amniotic-foetal debris into the maternal circulation, while traumatic SIO may have the characteristics of hypovolemia, neurogenic, obstructive, and cardiogenic (14). Uterine inversion with primary neurogenic non-haemorrhagic shock can be complicated by early secondary haemorrhagic shock and subsequently possible septic, as well as a ruptured ectopic pregnancy that has the characteristics of primary neurogenic haemorrhagic shock (19-22).

OBSTETRICS HEMORRHAGIES

Obstetric haemorrhages (OH) in the United States and Europe account for 1-2 maternal lives / 100,000 live births, and in developing countries 600-1,500 / 100,000 live births, according to 1999 statistics, while according to a 2005 WHO report, the number reaches 529,000 per year, which according to research indicates an increase in recent years by 25%, mostly due to atony and the condition behind the unreasonable increase in the number of caesarean sections and invasive / morbid malplacentation (16). Even today, more than 90% of maternal deaths in North Carolina are caused by obstetric haemorrhage and bleeding sequelae, and the incidence of postpartum haemorrhage (PPH) and major PPH (> 2500 mL) and transfusion needs are increasing in the EU countries, with uterine atony as the most significant risk factor (16). Obstetric haemorrhage is today associated with an enormous increase in caesarean sections and consequent previal and morbid invasive malplacentation with the need for emergent peripartum hysterectomy, which increases MM by 4 - 4.5% and blood loss to 3.5 L (17, 26-32). Massive obstetric haemorrhage is defined blood loss> 2500 mL, which is complicated by OS and most often the need for emergent hysterectomies (17, 26-32). The former causes of haemorrhagic OS and consequent high MM were severe obstetric hemorrhage due to uterine atony or rupture of the uterus. There are many causes of PPH / OS, but many cases come unexpectedly. An easy way to remember the most common causes is four T: tonus (uterine atony), trauma (genital organs during childbirth), tissue (retention of conception products), thrombin (coagulopathy) (33-35). Therefore, the World Health Organization (WHO) recommends prophylactic administration of uterotonics agents that increase uterine contractility for all births. Oxytocin is recommended for the prevention and as first line treatment of PPH/OS for vaginal delivery or caesarean section (33-35).

TREATMENT OF OBSTETRIC SHOCK

The goals of OS treatment are to quickly detect the location or cause of bleeding / injury / inflammation, prevent the progression of shock (multiple-organ failure - MOF, acute respiratory distress syndrome - ARDS), prevent massive transfusions, preserve the uterus (and adnexa), and preserve fertility if possible (2,4,29,36,37). Surgical treatment of septic shock includes exploratory laparotomy (laparoscopy), ectomy or resection of the necrotized (inflamed / gangrenous / phlegmonous) organ, abdominal lavage with multiple drainages, continuous peritoneal drainage with lavation, preservation of fertilization, extensive triple antibiosis (ex juvantibus) per admission or per antibiogram and thromboprophylaxis (2,4,29,36,37). The development of the clinical picture, the degree of severity of the OS or SOI condition and the prognosis can be assessed using several measurable scales, such as shock index, obstetric shock index (OSI) or modified obstetric shock index (MOSI) (2,4,29,36,37).

Once OH / PPH is identified, monitoring, mechanical and physiological measures are initiated. Mechanical measures include manual massage and compression of the uterus as well external aortic compression (38). Conservative treatment of OH / PPH consists of the use of pharmacological uterotonic agents: ergometrine (0.2 mg IM or IV), oxytocin (5 IU IV, then 10-20 IU/2 hours infusion), misoprostol (1000 mcg rectally) and injectable prostaglandins (carboprost and sulproston 100-500 mcg/hour IV infusion). Then fluid replacement: NaCl (IV in 10 minutes), HES (hydroxyethyl starch solution 6%, 20 ml/kg), Haemaccel (polygelin, sodium chloride, potassium chloride, calcium chloride), transfusion of blood products; two units of red blood cells 0 negative as required (38,39).

Commonly used haemostatic agents during hemorrhage are tranexamic acid for reducing clot dissolution, desmopressin acetate for platelets dysfunction (0.3 mcg/kg), recombinant activator factor VII, fibrinogen concentrates for improving clot strength (early use to maintain fibrinogen level above 200 mg/dL, initial dose commonly used between 2 and 3 g), prothrombin complex concentrates for reducing the time for initial clot formation (consisted of concentrates of human-derived vitamin K-dependent factors (II, VII, IX, X) (39,40).

The repeated introduction of tranexamic acid (TXA) into clinical obstetric practice has resulted in a significant reduction in blood loss and the development of hyperfibrinolysis, a reduction in maternal morbidity and mortality, and WHO has placed TXA in essential drugs (41,42). TXA is a safe drug for the prevention and treatment of obstetric bleeding. Use of TXA prevents acute complications of OH, including obstetric shock, development of disseminated intravascular coagulopathy and maternal mortality. TXA has no adverse effects during pregnancy, lactation and in the early neonatal period (43).

Prophylactic use is recommended in anaemic women before childbirth and caesarean section, as well as haematological diseases prone to coagulopathies and with expected significant PPH (distended uterus, polyhydramnios, multiparity, myomatous uterus, multiple pregnancy, previal and / or invasive malplacentation). Prophylactic dose is 1 g TXA intravenously 10-20 minutes before skin incision at caesarean section with uterotonic oxytocin in infusion (43,44).

The recommended maximum dose within 24 hours is 2 g. TXA therapy can be repeated for repeated or prolonged bleeding > 1500 mL, after half an hour, 1 g iv.

Therapeutically, TXA is administered when diagnosed with PPH in an intravenous bolus of 0.5-2 g for 10 minutes, or by continuous intravenous infusion of 1-2 mg / kg / hour with other bleeding treatment measures. There is no evidence of an elevated thrombogenic effect at the recommended doses (43- 44).

TXA is prescribed in all forms of obstetric shock in which hyperfibrinolysis has started or is at risk of developing disseminated intravascular coagulation, such as septic shock.

TXA can be safely administered in pregnancy with severe antenatal bleeding, placenta previa, or placental abruption, as well as polytrauma and bleeding from the respiratory or digestive tract (43-45).

Due to its pronounced antifibrinolytic effect, TXA is contradictory for the prophylaxis or treatment of PPH with acute thrombotic comorbidity such as deep vein thrombosis, cerebral thrombosis, or acute pulmonary embolism (43-45).

Surgical measures of OH/PPH are instrumental revision of uterine cavity, surgical repair of genital tract trauma, uterine artery embolization, uterine tamponade: surgical or by balloon catheter, laparotomy, compression sutures (B-Lynch, Hayman, Cho), ligation uterine arteries or internal iliac artery and emergency hysterectomy (33-35, 38).

The three most commonly used compression suture techniques for PPH are B- Lynch, Hayman, and Pereira, while other less often used compression sutures are Cho, Ouahba, Hackethal, and Massuba (33-35,).

In cases of severe OS or SOI during maternal resuscitation in viable pregnancy (≥ 23 weeks), prehospital or hospital perimortem caesarean section (PMCS) should be performed no later than 4 minutes (when possible) after maternal cardiac arrest to save the foetus and aided maternal resuscitation by burdening the pregnant uterus by increasing cardiac output and decreasing aortocaval compression and the possibility of better ventilation (47,48).

There are numerous reports in the literature of frequent and bizarre cases of various forms of OS or SIO that are all worthy of clinical attention and presentation as life-threatening conditions (49,50). Properly made early diagnosis (identification) of OS or SOI syndrome of any etiology, assessment of blood loss, assessment of clinical and laboratory condition, experience, training and multidisciplinary of the obstetric / midwifery, neonatology and anaesthesia team directly correlate with perinatal survival and early and late maternal and child morbidity (30-50).

FUTURE DIRECTIONS

Further standardization of the PPH nomenclature and OS development based on the need for therapeutic procedures, as well as the distinction

between OS and SIO forms is necessary, although the therapeutic approach will be according to the shock treatment guidelines outlined (16).

We need more experts and investigators who are interested in the OS and SIO challenges faced by women at the time of childbirth. We need more experts who are willing to study the causes, diagnostic and therapeutic approach of OS and SIO (51).

In conclusion OS seems to remain a permanent miasma in practical clinical obstetrics, which we will obviously not be able to influence, because we have led to the causes of today's increase in MM from haemorrhagic OS by iatrogenic increase in the number of caesarean sections, especially elective ones.

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