

The Golden Hour: Early Interventions for Medical Emergencies during Pregnancy

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Abstract

Maternal mortality has increased in the last decades in the United States as a result of increased prevalence of coexisting medical diseases such as hypertension, diabetes, and both acquired and congenital heart diseases. Obstetricians and maternal–fetal medicine physicians should have the basic medical knowledge to initiate appropriate diagnostic and early therapeutic interventions since they may be the only provider available at the time of presentation. The goal of this article is not to extensively discuss the management of complex medical diseases during pregnancy, rather we provide a concise review of key early medical interventions that will likely result in improved clinical outcomes.

Keywords

- pregnancy
- medical diseases
- medical emergencies

Key Points

- Obstetricians and maternal–fetal medicine physicians must be familiar with initial basic management of common medical emergencies.
- Management of these complex cases is ideally multidisciplinary.
- Residency/fellowship programs should include common disease management to improve maternal outcomes.

Contrary to other developed nations, maternal mortality has not decreased and may have even increased over time in the United States.¹ The rise in the prevalence of coexisting cardiovascular disease and obesity, among others, has changed the field of obstetrics and maternal–fetal medicine (MFM) dramatically. Unfortunately, most obstetrical programs do not provide adequate training in management of acute maternal care; as a result, obstetricians (OB) and MFM specialists must usually rely on multiple consultants in managing common medical emergencies during pregnancy. While multidisciplinary management of complex medical conditions is optimal, it often falls on the obstetrical team of providers to make the correct diagnosis and manage the patient before the arrival of consultant services. This initial interaction with the obstetrical service, including arriving at a differential diagnosis that will inform further evaluation and management, is critical for

determining outcomes. The obstetrical service is therefore the first line of defense when patients present with life-threatening conditions. As such, OB and MFM physicians should have basic medical knowledge to initiate such evaluation and interventions since they may be the only provider available initially. The goal of this article is not to extensively discuss management of complex medical diseases during pregnancy, rather we provide a concise review of key early medical interventions that will likely result in improved clinical outcomes. The goal for the obstetrician is to arrive at the appropriate differential diagnosis and institute the initial phase of management until the appropriate consultants arrive.

Sepsis

Sepsis is defined as the presence of an infectious process leading to a dysregulated inflammatory response resulting in

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acute organ dysfunction.² Sepsis should be suspected in any patient with an infectious process and evidence of any organ dysfunction (e.g., confusion, hypotension, oliguria, dyspnea and hypoxemia, ileus, abnormal coagulation studies, and elevated liver enzymes). Early source control is fundamental and achievable in many cases (e.g., delivery for chorioamnionitis, dilation and curettage for retained infected placental products, and wound debridement as indicated). Together with early source control, other vital interventions include obtaining cultures as indicated (e.g., blood, urine, wounds, and vaginal) and serum lactate.³ Lactate levels should be followed during early resuscitation; a progressive decline in lactate correlates with decreased mortality.³ Early administration of broad-spectrum antibiotics (in most cases providing coverage against anaerobic and gram-positive and gram-negative bacteria), together with fluid resuscitation with 1 to 2 L of crystalloid (preferably lactated Ringer's solution or plasma-lyte A) aiming for a mean arterial blood pressure (MAP) of 65 mm Hg, should not be delayed. If hypotension and hypoperfusion persist, norepinephrine (0.05 µg/kg/minute as a starting dose) should be initiated targeting a MAP of 65 mm Hg.^{3,4} During the midtrimester, physiologic vasodilation may result in MAP below 65 mm Hg; the latter should not be the sole indication for aggressive fluid resuscitation and vasopressor support in the absence of other markers of hypoperfusion such as oliguria, confusion, tachycardia, cold extremities, and elevated serum lactate.

Ischemic Stroke

Ischemic stroke should be suspected with the acute onset of neurologic deficits, such as paresthesia, hemiparesis or hemiplegia, seizures, dysarthria, facial droop, or confusion/obtundation, among others. Once suspected, stroke protocols should be activated immediately. Most hospitals have such a protocol or have a process for emergency consultation of a stroke team. Obstetrical care providers should be familiar with their particular institutional approaches. Imaging with computed tomography (without contrast, followed by angiography in many centers) should proceed without delay, as this will identify optimal candidates for reperfusion therapy.⁵ Early measures to prevent secondary brain injury must be undertaken as soon as possible. If the airway cannot be protected, the patient should be immediately intubated, aiming for a normal arterial partial pressure of oxygen (above 80 mm Hg) and carbon dioxide (35–45 mm Hg in nonpregnant patients, 30 mm Hg in pregnancy). Hypotonic fluids (e.g., the lactated Ringer's solution or plasma-lyte A) should be avoided, as they will result in hyponatremia, potentially worsening cerebral edema.⁵ Normal saline should be the initial fluid utilized. Hyperglycemia and hypoglycemia should be avoided, targeting glucose levels between 140 and 180 mg/dL.⁵ Any temperature elevation should be managed as soon as possible since hyperthermia worsens neuronal injury. Cooling blankets targeting normothermia are indicated.

If the patient is a candidate for reperfusion with systemic tissue plasminogen activator (tPA; window between presentation and onset of symptoms of 4.5 hours or less), blood

pressure control is important. In nonpregnant individuals, treatment is required if the blood pressure is above 185/110 mm Hg prior to administering tPA.⁶ For tPA recipients during pregnancy, we recommend targeting blood pressures between 140 and 160/90 and 110 mm Hg with the use of labetalol (sequential boluses of 10, 20, 40, and 80 mg at every 10 minutes with a maximum cumulative dose of 300 mg) or nicardipine (continuous infusion starting at 2.5 mg/h with increments of 2.5 mg every 5–10 minutes to a maximum of 15 mg/h).⁵ The window for endovascular intervention with local thrombolysis and clot stent retrieval of proximal thrombi is up to 24 hours after onset of symptoms.⁷ For this procedure, we recommend targeting the same blood pressures described for systemic tPA. If tPA is administered, aspirin and deep vein thrombosis (DVT) prophylaxis should not be started until 24 hours later and after documenting the absence of hemorrhagic transformation post tPA.

If no tPA or thrombectomy is planned, blood pressure should also be maintained between 140 and 160/90 and 110 mm Hg. In these patients, aspirin 160 to 300 mg/day, together with DVT prophylaxis, should be started as soon as possible.⁸

Hemorrhagic Stroke

Hemorrhagic strokes from uncontrolled hypertension commonly affect the basal ganglia. The clinical presentation of a hemorrhagic stroke may be similar to the ischemic counterpart; suspect it with any acute onset of neurologic dysfunction including seizures, paresthesia, hemiparesis or hemiplegia, dysarthria, facial droop, or confusion/obtundation. A hypertensive response usually follows the stroke event in an attempt to increase cerebral perfusion pressure. Current guidelines recommend blood pressure treatment with values above 220/120 mm Hg.⁶ Treatment aiming for lower blood pressures (systolic blood pressure < 140 mm Hg) does not improve neurologic outcomes and may increase the risk of end organ damage (acute kidney injury).⁹ During pregnancy, we recommend maintaining blood pressure between 140 and 160/90 and 110 mm Hg with labetalol or nicardipine as described above.⁵ As with ischemic strokes, prevention of secondary brain injury is paramount: avoid hypotonic fluids, fever, and hypo/hyperglycemia (target glucose: 140–180 mg/dL). If the patient is on low-dose aspirin, discontinuation is appropriate; transfusion of platelets, however, is not indicated unless a neurosurgical procedure is planned.¹⁰ Thrombocytopenia may result in hematoma expansion. Most experts recommend maintaining a platelet count above 100,000/mm³ with platelet transfusions as needed.¹¹ Reversal of anticoagulants, such as warfarin (with intravenous [IV] vitamin K and prothrombin complex concentrates), unfractionated heparin (with protamine sulfate), and the new direct oral anticoagulants (with idarucizumab or andexanet α) may be required.¹⁰ Intubation with mechanical ventilation may be necessary if the patient is unable to protect her airway or if hypoxemia and/or hypercapnia develop (as the latter two result in cerebral vasodilation with worsening intracranial hypertension). If preeclampsia is suspected in the differential diagnosis, magnesium sulfate should be administered.

Aortic Dissection

The clinical presentation of aortic dissection typically includes sudden onset of severe chest pain which radiates to the interscapular region. Risk factors for aortic dissection include Ehlers-Danlos, Marfan's, and Turner's syndromes; fibromuscular dysplasia; and congenital bicuspid aortic valve. If suspected, the most important early medical management goal is rapid blood pressure control, as uncontrolled hypertension will propagate the dissection. Treatment with antihypertensives should begin as soon as possible, even before imaging, if clinical suspicion is high. Vasodilators should not be administered prior to adequate β blockade since the acute decrease in afterload will increase shear stress with further dissection.¹² Instead, treatment is started with IV esmolol (25–300 $\mu\text{g/kg/minute}$) followed by an IV vasodilator, such as nicardipine (2.5–15 mg/h) or nitroprusside (0.2–2 $\mu\text{g/kg/min}$), aiming for a systolic blood pressure below 120 mm Hg.⁶ Alternatively, IV labetalol monotherapy may be utilized, as it is a combined α - and β -blockers. Proximal aortic dissection (Stanford's A) requires surgical repair. Most distal dissections (Stanford's B) are managed expectantly.

Myocardial Infarction

Myocardial infarction (MI) usually presents with sudden onset of chest pain which radiates to the upper extremities and/or jaw. Other symptoms include diaphoresis, dyspnea, nausea, vomiting, and epigastric pain. Women are prone to atypical presentations including lack of significant chest pain. Grossly, MI may be divided into ST MI (elevation of the ST segment) and non-ST MI (without elevation of the ST segment). Elevation of serum troponins reflects myocardial necrosis. Unlike in nonpregnant individuals, most MI during pregnancy are due to coronary dissection (especially during the third trimester and postpartum period).¹³ Most coronary dissections affect the left descending coronary artery.

Initial management of a suspected MI includes pain relief with nitroglycerin (0.4-mg sublingual in every 5 minutes up to three doses) or morphine (2–4 mg IV). Oxygen should be administered if peripheral oxygen saturation (SpO_2) is below 90%.¹⁴ The cornerstone of management is administration of concomitant anticoagulation and antiplatelet therapy. IV unfractionated heparin (UFH) should be started (bolus of 60 units/kg, maximum 4,000 units, followed by an initial infusion of 12 units/kg/h, maximum 1,000 units/h, aiming for an activated partial thromboplastin time of 50–70 seconds) together with chewed noncoated aspirin (162–325 mg, followed by 81 mg daily).¹⁵ Dual antiplatelet therapy is standard and consists of adding a P2Y₁₂ blocker, such as clopidogrel, ticagrelor, or prasugrel, to aspirin. Available limited data suggest that clopidogrel (300 mg oral load followed by 75 mg a day) is safe in pregnancy.¹⁶ No data exist on the safety of ticagrelor or prasugrel during pregnancy. Recent studies suggest that ticagrelor is the preferred agent for patients treated conservatively, while prasugrel is optimal among patients undergoing percutaneous coronary intervention (PCI).¹⁷ If in doubt, addition of a P2Y₁₂ blocker may be delayed until the coronary angiography is performed

(if no PCI is indicated, then clopidogrel or ticagrelor may be added; if PCI is indicated, then prasugrel will be ideal).¹⁷ If hypertension and tachycardia are present, IV β -blockers are indicated (metoprolol 2.5–5 mg at every 5 minutes to a maximum of 15 mg or esmolol 25–300 $\mu\text{g/kg/min}$). Persistent hypertension may be treated with IV nitroglycerin 5 to 100 $\mu\text{g/min}$ (avoid if right ventricular MI is suspected). If coronary angiography reveals a coronary dissection, patients are commonly managed conservatively without PCI, as coronary interventions may worsen the dissection.¹⁸ UFH should be stopped after the diagnosis of coronary dissection, and continuation of aspirin and β -blockers is recommended.¹⁸ Methergine is contraindicated in patients with acute MI.

Seizures (Noneclamptic)

Most generalized tonic-clonic epileptic seizures are self-limited. During the episode, administration of oxygen and positioning the patient in the left lateral decubitus is recommended. First-line medication is IV lorazepam (0.1 mg/kg, usually starting with 4 mg).¹⁹ If IV access is not available, intramuscular (IM) midazolam (10 mg) is equally effective.²⁰ Most patients will require a long acting antiepileptic drug (AED) concomitantly. Commonly used agents include IV phenytoin or fosphenytoin (15–20 mg/kg) or levetiracetam (500–2,000 mg IV). In patients previously receiving a specific AED, a loading dose of the maintenance agent is commonly indicated. Refractory cases may require endotracheal intubation followed by infusions of midazolam (preferred) or propofol.²¹

Acute Right Ventricular Failure

Acute right ventricular failure (RV failure) may occur with any condition that increases right ventricular afterload (e.g., pulmonary embolism, air embolism, amniotic fluid embolism, or exacerbation of existing pulmonary hypertension by triggers such as hypoxemia, acidosis, or hypercarbia). Clinically, patients may present with systemic hypotension, dyspnea, oxygen desaturation, peripheral edema, hepatic congestion, and distended jugular veins. The diagnosis may be easily confirmed with limited bedside transthoracic echocardiography (TTE; [Fig. 1](#)). The most important aspect of acute treatment is to avoid fluids. Overzealous fluid administration worsens hypotension by further dilating the RV, with left-sided displacement of the interventricular septum resulting in left ventricular obliteration and decreased cardiac output.²² Instead, hypotension should be treated with vasopressors (IV norepinephrine starting at 0.05 $\mu\text{g/kg/min}$ and titrated to a MAP of 65 mm Hg). Oxygen should be administered if hypoxemia is present since the latter worsens RV failure by causing pulmonary vasoconstriction.²² Contractility of the RV may be improved by starting an inotropic agent, such as dobutamine (2.5–5 $\mu\text{g/kg/min}$) or milrinone (0.25–0.75 $\mu\text{g/kg/min}$). If pulmonary embolism (PE) is the underlying cause, immediate therapeutic anticoagulation with UFH should be started (80 U/kg bolus, followed by 18 U/kg/h, aiming for an activated partial thromboplastin time of 60–85 seconds or an anti-Xa value of 0.3–0.7 U/mL).

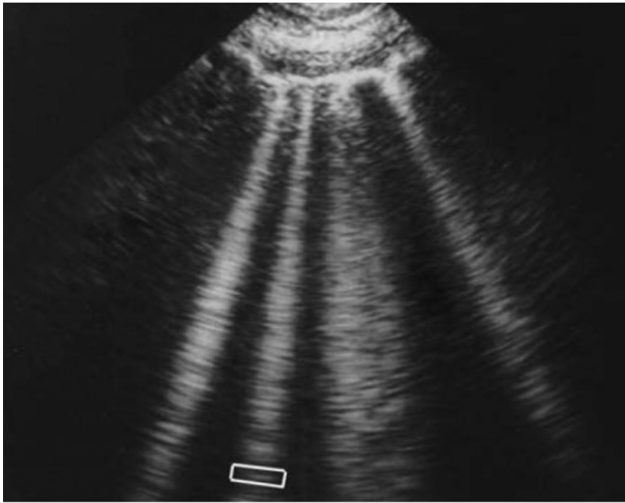


Fig. 1 Acute right ventricular failure. Please note the massively dilated right ventricle compressing the left ventricle. Reproduced with permission under Creative Commons license type BY 2.0 from Jochmann et al.³¹

The presence of hypotension (systolic blood pressure below 90 mm Hg) in the setting of PE warrants the use of thrombolytic therapy, such as tPA.²³ For massive PE, the recommended dose of tPA is 100 mg IV over 2 hours.²³ Thrombolysis should not be delayed if indicated during pregnancy.²⁴ Recent surgery (e.g., cesarean section) is a relative contraindication to using tPA, but its use in this life-threatening condition may be indicated even in the setting of a recent surgical intervention.

Acute Left Ventricular Failure

Acute left ventricular failure commonly presents with new onset dyspnea, hypoxemia, tachypnea, and orthopnea. Clinical examination reveals bilateral pulmonary crackles. Bed-side point-of-care ultrasonography will show B lines (rocket lines, ▶Fig. 2).²⁵ In severe cases, decreased systolic function may result in hypotension, oliguria, confusion, and cold clammy extremities. The immediate management of acute cardiogenic pulmonary edema centers on preload and afterload reduction combined with both respiratory and inotropic support. All fluids should be discontinued. Diuresis with

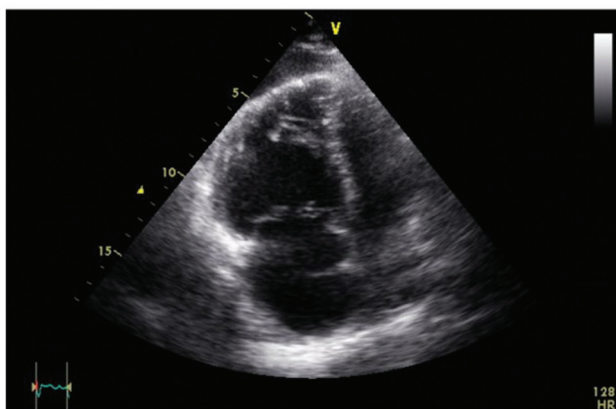


Fig. 2 Hyperechoic B lines extending to the bottom of the screen indicating pulmonary edema. Reproduced with permission under Creative Commons license type BY 4.0 from Macias.³²

furosemide (intermittent boluses or a continuous infusion) is indicated.²⁶ Common doses include 20- to 80-mg IV boluses at every 6 to 8 hours or an infusion of 1 to 10 mg/h. Besides its diuretic effect, furosemide induces venodilation with subsequent decreased preload. The use of morphine sulfate (2–4 mg IV) also results in venodilation and preload reduction. If systemic hypertension is present, afterload reduction is indicated. Commonly used agents in this setting include IV nitroglycerin (5–300 µg/min), nitroprusside (0.2–2 µg/kg/min), nicardipine (2.5–15 mg/h), and hydralazine (5–10 mg every 20 minutes). Nitroglycerin may be particularly effective, as it will improve pulmonary edema by inducing systemic venodilation with decreased preload. Beta-blockers should be avoided in acute decompensated left-ventricular failure. Respiratory support with noninvasive mechanical ventilation (e.g., CPAP) commonly results in improved oxygenation and improvement in pulmonary edema (a continuous positive airway pressure of 8–10 cm H₂O is a reasonable starting point). In patients with cardiogenic shock (systolic blood pressure below 90 mm Hg) and evidence of end-organ hypoperfusion, blood pressure support with norepinephrine (starting at 0.05 µg/kg/min and titrated to a MAP of 65 mm Hg) is usually indicated. Addition of inotropic agents (e.g., dobutamine or milrinone) is commonly required, as norepinephrine may further decrease cardiac output after increasing systemic vascular resistances.²⁶ Refractory cases may require mechanical support with left ventricular assist devices.

Diabetic Ketoacidosis

Diabetic ketoacidosis (DKA) usually presents with nonspecific symptoms including nausea, vomiting, abdominal pain, polyuria, and polydipsia leading to dehydration together with a gap metabolic acidosis and hyperglycemia. Other findings include deep fast breathing (Kussmaul's respiration) in an attempt to clear carbon dioxide. A fruity breath odor indicates acetone clearance. The diagnosis is usually confirmed by identifying β-hydroxy butyric acid in blood with a wide anion gap. The anion gap is calculated as (Sodium – [chloride + bicarbonate]), and it is considered increased when above 12 mmol/L. Early management of DKA includes IV fluids, insulin administration, and potassium replacement. Patients with DKA are usually volume depleted secondary to osmotic diuresis and require IV fluid resuscitation. This may be accomplished with 1 to 2 L of crystalloid (normal saline, lactated Ringer's solution, or plasma-lyte A) in the first 1 to 2 hours, depending on the severity of hypovolemia, followed by an infusion of 150 to 250 cc/h. Once a glucose value of 200 to 250 mg/dL is reached, 5% dextrose may be added to each bag of crystalloid to prevent iatrogenic hypoglycemia. Normal saline has been associated with a higher risk of acute kidney injury secondary to hyperchloremia in critically ill patients.²⁷ Although this has not been described in patients with DKA, if serum chloride is elevated following use of normal saline, we recommend switching to a balanced crystalloid solution, such as lactated Ringer's solution or plasma-lyte A. IV insulin should be started at a rate of 0.1 U/kg/h.²⁸ If blood glucose does not decrease by at least 50 to 70 mg/dL in the first hour, the infusion rate should be

Table 1 Key points on early acute management of medical emergencies in pregnancy

Medical emergency	Management
Sepsis	<ul style="list-style-type: none"> • Suspect with any infectious process and evidence of any organ dysfunction (e.g., confusion, hypotension, oliguria, dyspnea and hypoxemia, ileus, abnormal coagulation studies, and elevated liver enzymes) • Early volume administration (1–2 L crystalloid); add norepinephrine if no response to fluids • Obtain cultures and serum lactate • Start broad spectrum antibiotics • Achieve source control
Ischemic stroke	<ul style="list-style-type: none"> • Suspect with acute onset of neurologic deficits such as paresthesia, hemiparesis or hemiplegia, seizures, dysarthria, facial droop, and confusion/obtundation • Early head imaging and stroke protocol activation • Secure airway if needed • Blood pressure control (<160/110 mm Hg in pregnancy) • Normal saline as maintenance fluid • Avoid hyponatremia, fever, or hyperglycemia
Hemorrhagic stroke	<ul style="list-style-type: none"> • Suspect with acute onset of neurologic deficits, such as paresthesia, hemiparesis or hemiplegia, seizures, dysarthria, facial droop, and confusion/obtundation • Early head imaging • Secure airway if needed • Blood pressure control (<160/110 mm Hg during pregnancy) • Normal saline as maintenance fluid • Avoid hyponatremia, fever, or hyperglycemia • Magnesium sulfate if preeclampsia suspected
Aortic dissection	<ul style="list-style-type: none"> • Usually presents with sudden onset of severe chest pain which radiates to the interscapular region. Risk factors for aortic dissection include Ehlers–Danlos, Marfan's, and Turner's syndromes; fibromuscular dysplasia; and congenital bicuspid aortic valve • Early blood pressure control, aim for systolic <120 mm Hg • Avoid pure vasodilators in absence of early β-blockade • Avoid misdiagnosis with acute coronary syndrome
Myocardial infarction	<ul style="list-style-type: none"> • Sudden onset of chest pain which radiates to the upper extremities and/or jaw. Other symptoms include diaphoresis, dyspnea, nausea, vomiting, and epigastric pain • Pain control with morphine sulfate or sublingual nitroglycerin • Oxygen if saturations below 90% • Start aspirin • Start intravenous unfractionated heparin • Intravenous β-blockade if hypertension and tachycardia
Non eclamptic seizures	<ul style="list-style-type: none"> • Acute onset of generalized tonic clonic movements • Oxygen administration • Left lateral decubitus • Early benzodiazepine administration • Load with long acting antiepileptic drug
Acute right ventricular failure	<ul style="list-style-type: none"> • Systemic hypotension, dyspnea, oxygenation desaturation, peripheral edema, hepatic congestion, and distended jugular veins. Confirm with bedside transthoracic echocardiography if available • Bedside transthoracic echocardiography if available • Treat hypotension with vasopressors/inotropes in lieu of fluids • Avoid hypoxemia, acidosis, or hypercarbia • If pulmonary embolism, start intravenous unfractionated heparin. If hypotension, administer systemic tissue plasminogen activator

Table 1 (Continued)

Medical emergency	Management
Acute left ventricular failure with pulmonary edema	<ul style="list-style-type: none"> • Discontinue all fluids • Administer furosemide • Positive pressure ventilation as needed (invasive or noninvasive) • Treat hypertension with vasodilators • Vasopressors and/or inotropes if systemic hypotension
Diabetic ketoacidosis	<ul style="list-style-type: none"> • Start intravenous fluids • Check potassium level prior to starting insulin infusion • Insulin infusion • Identify potentially treatable triggering factors (e.g., infection)
Thyroid storm	<ul style="list-style-type: none"> • Early propylthiouracil (PTU) • Iodide following PTU • Corticosteroids • Beta blockers as needed

doubled. Importantly, in patients with significant hypokalemia (serum potassium <3.3 mEq/L), potassium replacement should be started prior to insulin to avoid the potential for insulin-induced life-threatening hypokalemia.^{28,29} Usually, replacement of KCl 20 mEq IV per hour for 2 hours before insulin administration will suffice. In patients with normal serum potassium, maintenance fluids should contain potassium (20–40 mEq/L). In women presenting with potassium levels above 5.3 mEq/L, initial fluids are administered without potassium; however, serial potassium measurements are indicated since hypokalemia will be “unmasked” as IV fluids and insulin are administered. Clinicians should actively seek out any treatable precipitating factor, such as infection.

Fetal heart rate anomalies, including minimal or absent variability, are frequently seen with maternal acidosis and should not lead to a rush for cesarean delivery. The latter should be reserved for prolonged bradycardia that does not resolve with maternal resuscitation. Beta mimetics should be avoided in patients with DKA.

Thyroid Storm

Thyroid storm usually presents with systolic hypertension (wide pulse pressure), tachycardia, hyperthermia, diaphoresis, tremor, nausea, vomiting, diarrhea, and agitation. Acute management is directed at blocking synthesis, release, and peripheral action of thyroid hormones. Propylthiouracil (PTU) should be started immediately at a dose of 1 g orally (nasogastric or orogastric tube if unable to swallow), even before the laboratory testing results are available. Once the diagnosis is confirmed, the loading dose is followed by 200 mg at every 6 hours.³⁰ One hour after PTU is started and iodide is administered. Iodide may be given as potassium iodide (KI; 5 drops orally every 8 hours) or Lugol’s solution (10 drops every 8 hours). IV sodium iodide is another option (1 g every 8 hours).³⁰ Dexamethasone 2 mg IV every 6 hours for four doses also will decrease thyroid hormone production and secretion. Hypertension and tachycardia usually require management with β -blocking agents (propranolol, labetalol, or esmolol). IV esmolol (25–300 μ g/kg/min) is particularly useful in patients at risk of high-output cardiac failure (e.g., severe tachycardia with mild pulmonary edema secondary to decreased ventricular filling time) since iatrogenic worsening of cardiac output following β -blockade is easily reversible after stopping the infusion (esmolol half-life is only a few minutes long). ▶Table 1 provides a summary of our recommendations.

Conclusion

Obstetrical care providers are often the first to respond to the above emergencies. In all the conditions described here, timeliness of the intervention affects outcome. OBs are therefore required to arrive at the diagnosis and initiate management in a timely manner and before any help arrives. We recognize that many of these recommendations are extrapolated from nonpregnant individuals. Future studies should ideally include pregnant women to test the efficacy in obstetric patients. ▶Table 1 provides a summary of our recommendations.

Conflict of Interest

None declared.

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