

Stroke and Pregnancy: An Integrative Review With Implications for Neuroscience Nurses



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ABSTRACT

Stroke in association with pregnancy is an infrequent occurrence, but there is evidence that the incidence is rising. The physiological changes of pregnancy are thought to increase stroke risk, and several conditions specific to pregnancy further increase risk. The provision of optimal care to pregnant and postpartum women who experience stroke requires awareness of how the physiological changes of pregnancy may affect the course of stroke and nursing actions. This article provides an overview of current knowledge about pregnancy-related stroke including underlying pathophysiology, risk factors unique to pregnancy, and treatment issues when stroke is a complication of pregnancy. Implications for the nursing care of women with pregnancy-related stroke and maternal child considerations are discussed.

Keywords: cerebrovascular disease, pregnancy, review, stroke

Pregnancy-related stroke (PRS) is an infrequent but potentially catastrophic vascular event. For this article, PRS is defined as a stroke of any type occurring while a woman is pregnant (antenatal stroke), during labor and delivery including the time spent in the hospital after giving birth (delivery stroke), and within 6 weeks of delivery (postpartum stroke; Kuklinka, Tong, Bansil, George, & Callaghan, 2011). Data from the Nationwide Inpatient Sample (NIS), an Agency for Healthcare Research and Quality database, indicate that the prevalence of stroke in 2006/2007 was 0.71 per 1,000 antenatal, delivery, and postpartum hospitalizations (Kuklinka et al., 2011). Between 1994/1995 and 2006/2007, the number of PRS hospitalizations rose by 54%, from 4,085 to 6,293 (Kuklinka et al., 2011).

The increase in PRS mirrors a rise in stroke hospitalizations among younger individuals. Hospitalizations for acute ischemic stroke (AIS), the most common type of stroke, significantly increased for both female and male persons aged 15–44 years between 1995/1996 and 2007/2008 (George, Tong, Kuklinka, & Labarthe, 2011). The rise in AIS hospitalizations for younger adults may reflect an increase in identified risk factors for stroke that include hypertension, diabetes, obesity, arrhythmia, migraine, and tobacco use (George et al., 2011). Among adolescent and young women aged 15–34 years hospitalized for ischemic stroke in

2007/2008, 32.2% had hypertension compared with 19.2% with hypertension in 1995/1996 ($p < .001$), and diabetes increased from 7.4% to 17.1% ($p < .001$) during the same period (George et al., 2011).

Stroke is a leading cause of death and disability, affecting more than 6 million individuals worldwide each year (http://www.who.int/cardiovascular_diseases/resources/atlas/en/). The reported rise in stroke among younger individuals and women of childbearing age is of concern because these individuals often are actively engaged in the workforce and starting and caring for families. Nurses in critical care units, stroke units, and medical units may be called upon to provide care for women who have experienced PRS. This article provides an overview of current knowledge about PRS including underlying pathophysiology, risk factors unique to pregnancy, and treatment issues when stroke is a complication of pregnancy. Implications for the nursing care of women with PRS and maternal child considerations are discussed.

Pathophysiology of Stroke Stroke Classifications

Strokes are broadly classified as ischemic or hemorrhagic. Eighty-seven percent of all strokes occurring annually in the United States are ischemic (Mozaffarian et al., 2014). AIS occurs when an artery in the brain is occluded by atherosclerotic plaque and/or thrombi, depriving the surrounding area of oxygenated blood and causing brain tissue injury (infarction). Thrombi may form outside the brain and travel through the circulatory system until becoming lodged in an intracerebral vessel. Infarction is the result of a series of metabolic processes called ischemic cascade, during which glucose and oxygen deprivation causes acidosis, depolarization of the cell membrane, and

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disturbances in intracellular calcium and sodium in brain cells (Durukan & Tatsumaka, 2007; Siesjo, 1992a, 1992b; Smith, 2004). Major risk factors for AIS identified by the American Heart Association/American Stroke Association (AHA/ASA) include hypertension, diabetes, atrial fibrillation, history of parental AIS before the age of 65 years, and cigarette smoking (Mozaffarian et al., 2014).

Cerebral venous thrombosis (CVT), another form of ischemic stroke, occurs when thrombi form in the cerebral or dural sinus veins that drain the brain. CVT is estimated to account for less than 1% of all strokes (Bousser & Ferro, 2007). The pathophysiology of CVT is similar to that underlying the development of deep vein thrombosis (DVT) and consists of “Virchow’s triad” of venous stasis, blood vessel wall changes, and hypercoagulability (de Freitas & Bogousslavsky, 2008). Conditions associated with elevated CVT risk include deficiencies of antithrombin III, protein C and protein S, antiphospholipid syndrome, pregnancy and the postpartum period, and oral contraceptives (Saposnik et al., 2011).

Hemorrhagic stroke is because of bleeding within the brain or the subarachnoid space, and 10% of all strokes are because of intracerebral bleeding, and 3% of all strokes are because of subarachnoid bleeding (Mozaffarian et al., 2014). Subarachnoid hemorrhage (SAH) results from a ruptured aneurysm or an arteriovenous malformation. Intracerebral hemorrhage (ICH) usually originates from small blood vessels that have undergone damage from chronic hypertension (Qureshi et al., 2001). During ICH, brain tissue is damaged by reduced perfusion, the mechanical force of leaking blood, edema, and inflammatory response (Testai & Aiyagari, 2008).

Physiologic Changes of Pregnancy and Stroke

Pregnancy is characterized by complex changes in virtually every system of the body. Many of these changes are the result of the two main steroidal hormones of pregnancy, estrogens and progesterone, which, by the end of pregnancy, are many times higher than in nonpregnant women (Cunningham et al., 2010). Hormone-mediated hemodynamic and hemostatic changes may predispose pregnant and postpartum women to stroke, although the extent to which these changes affect stroke risk has not been clearly delineated (Del Zotto, Giossi, Volonghi, Padovani, & Pezzini, 2011).

Serum concentrations of clotting factors VII, VIII, and X and von Willebrand factor increase during pregnancy, and coagulation inhibitor protein S begins falling in the first trimester and remains low throughout pregnancy (Brenner, 2004). Fibrinogen, the protein converted to fibrin during the blood clotting process, increases during pregnancy, slowing the breakdown of

Hormonal and hemodynamic changes that occur normally during pregnancy, and that meet the physiologic needs of the fetus and the demands of delivery, may play a significant role in the development of pregnancy-related stroke.

fibrin (Brenner, 2004). These alternations in the coagulation profile support placental functioning and reduce bleeding at delivery (Cunningham et al., 2010) but may contribute to the risk for AIS and CVT by predisposing pregnant and postpartum women to thrombus formation (Tate & Bushnell, 2011; Treadwell, Thanvi, & Robinson, 2008). Volume depletion because of the exertion of labor and delivery, blood loss after delivery, venous stasis because of compression of the iliac vein by the gravid uterus, and reduced mobility as delivery nears and during labor are proposed as contributors to AIS and CVT (Lanska & Kryscio, 2000; Treadwell et al., 2008).

Hemodynamic changes that meet the increased circulatory needs of the uterus, placenta, developing fetus, and the demands of delivery may play a role in PRS. Blood volume begins to rise in early pregnancy, and by delivery, it is 30%–40% greater than in nonpregnant women (Cunningham et al., 2010). Pressure on the vasculature because of the increased blood volume is proposed as a cause of bleeding from an arteriovenous malformation or aneurysm (Treadwell et al., 2008). However, the risk for SAH because of aneurysm during pregnancy through 6 weeks after birth did not differ from rates expected for nonpregnant women (Tiel Groenestege, Rinkel, van der Bom, Algra, & Klijn, 2009), and an increased rate of hemorrhagic stroke because of vascular malformations was not found in a case control study (Bateman et al., 2006).

Timing of PRS

Approximately two thirds of stroke hospitalizations in the 2006/2007 NIS data were during the delivery and postpartum periods (Kuklinka et al., 2011). This pattern is similar to an earlier analysis from 46 hospitals participating in the Baltimore–Washington Cooperative Young Stroke Study in which 62% of PRS occurred within 24 hours of delivery through 6 weeks after delivery (Kittner et al., 1996).

In the 2006/2007 NIS data, CVT was more common (30.6%) in the antenatal period than either ischemic

(21.4%) or hemorrhagic (15.7%) stroke (Kuklinka et al., 2011). The type of stroke occurring most often during delivery hospitalizations was CVT (43%), followed in frequency by hemorrhagic stroke (10.7%) and ischemic stroke (8.1%). Hemorrhagic stroke accounted for 35.6% of strokes in the postpartum period, compared with strokes because of CVT (24%) and AIS (16.5%; Kuklinka et al., 2011). It is notable that the rate of postpartum hemorrhagic stroke increased fourfold between 1994/1995 and 2006/2007 (Kuklinka et al., 2011).

Outcomes of PRS

Mortality was 15.8% among 183 women who had a stroke while hospitalized for delivery (Lanska & Kryscio, 2000). Scott et al. (2012) reported an overall case fatality rate of 20% among 30 cases of antepartum stroke, with all mortality attributed to either ICH or SAH. Similarly, Jaigobin and Silver (2000) reported in a series of 34 women with PRS that all 21 women with ischemic stroke or CVT survived, and the three fatalities were among 13 women with hemorrhagic stroke. These studies implicate hemorrhagic stroke as an important cause of maternal mortality. The authors of an analysis of 423 pregnant and postpartum women with ICH in which 20% died estimated that 7.1% of all pregnancy-related maternal deaths were attributed to ICH (Bateman et al., 2006).

There are few studies reporting functional outcomes among women with PRS. Ten percent of the women in the Lanska and Kryscio (2000) study required care at a skilled nursing or other facility after discharge from the hospital. In an earlier analysis from France, 5 of 15 (33.3%) of women with nonhemorrhagic stroke had mild-to-moderate neurological deficits at the time of hospital discharge, and 8 of 12 (66.6%) of women who survived hemorrhagic stroke had some degree of neurological deficit (Sharshar, Lamy, & Mas, 1995). Among the 423 women with ICH in the Bateman et al. (2006) analysis, fewer than half (44.2%) were discharged routinely, and 35.2% were either transferred to other medical facilities or required home health services.

Risk Factors for PRS

General stroke risk factors when present in a pregnant or postpartum woman may contribute to PRS. Preexisting hypertension increases the odds for PRS threefold (Bateman et al., 2006) to ninefold (James, Bushnell, Jamison, & Myers, 2005). Migraine headache in pregnancy, even after adjustment for other factors, was associated with significant risk for both hemorrhagic and nonhemorrhagic PRS (Bushnell, Jamison, & James, 2009; Scott et al., 2012). Cardiac disease including preexisting atrial fibrillation, congenital heart disease including

atrioventricular malformations, rheumatic heart disease with or without valve replacement, and left ventricular hypertrophy are risk factors for AIS in pregnancy (Jeng, Tang, & Yip, 2004).

In a national cohort study, preexisting coagulopathy increased the odds of PRS, particularly ICH, twentyfold, with 95% confidence interval (CI) of [13.67, 31.23] (Bateman et al., 2006). Preexisting coagulopathy is frequently related to other diseases such as systemic lupus, which is associated with idiopathic thrombocytopenic purpura. James et al. (2005) reported that lupus significantly increased the odds for PRS, (odds ratio [OR] = 10.5, 95% CI [7.4, 31.2]). Primary antiphospholipid syndrome, with or without an association with another disease such as lupus, is another cause of preexisting coagulopathy that increases risk for PRS. In another cohort study, the most common risk factor for PRS-CVT when comparing stroke related and unrelated to pregnancy was coagulopathy, accounting for 7 of the 11 (64%) women with PRS-CVT (Jeng et al., 2004).

Age and race are associated with PRS. In a population-based cohort in the United Kingdom, 33% of women experiencing an antenatal stroke were aged 35 years or older compared with 15% of the women in the control group ($p = .03$; Scott et al., 2012). Results from analyses within the NIS also cited advanced maternal age as an independent risk factor for stroke in pregnancy (OR = 2.11, 95% CI [1.69, 2.64]; Bateman et al., 2006), and women aged 35 years and older were particularly at risk (James et al., 2005). African American race was associated with increased risk for PRS (OR = 1.83, 95% CI [1.39, 2.41]; Bateman et al., 2006), and the risk increased even further among African American women aged 35 years and older (OR = 4.5, 95% CI [2.9, 6.8]; James et al., 2005). Hispanic race was associated with increased risk for death subsequent to stroke in pregnancy (Bateman et al., 2006).

Pregnancy-Related Conditions

Several conditions unique to pregnancy contribute to PRS including gestational hypertension with or without preeclampsia and/or eclampsia, cardiomyopathy, amniotic fluid embolism (AFE), postpartum angiopathy, and postpartum hemorrhage (PPH). The data on risk for PRS and cesarean birth are conflicting (Lanska & Kryscio, 2000). One thought is that cesarean birth is more likely in women with other risk factors for stroke, leading to confusion about the nature of the association between cesarean and stroke. Another postulate is that cesarean birth increases the risk for postpartum CVT (Tate & Bushnell, 2011).

Hypertensive Conditions

Preeclampsia and eclampsia, systematic conditions that typically involve increases in blood pressure, proteinuria,

and hemodynamic changes, have been associated with both AIS- and ICH-PRS (Jeng et al., 2004; Kittner et al., 1996; Scott et al., 2012; Wasay et al., 2010). The endothelial dysfunction associated with this disease may cause a synergistic effect with the increased clotting of pregnancy that promotes risk for stroke (Jeng et al., 2004). The hypertension of preeclampsia and eclampsia may lead to segmental vasoconstriction, increasing risk for ICH (Tate & Bushnell, 2011). Pregnant women with hypertension, and in particular, women with eclampsia or preeclampsia, had a significant risk for stroke because of cerebral hemorrhage (Jeng et al., 2004).

Results from a UK study indicate that the odds of antenatal stroke were increased among women with preeclampsia (OR = 7.7, 95% CI [1.3, 55.7]; Scott et al., 2012). In this study, the risk for PRS increased by 8% for every millimeter-of-mercury increase above the highest recorded diastolic blood pressure and 3% for every millimeter-of-mercury increase above the highest recorded systolic blood pressure (adjusted OR = 1.08, 95% CI [1.03, 1.13] and adjusted OR = 1.03, 95% CI [1.00, 1.05], respectively). Women at high risk for preeclampsia and eclampsia frequently have conditions that are independent risk factors for PRS (e.g., obesity, preexisting cardiac disease, lupus, diabetes) that potentiate risk when women develop these hypertensive conditions in pregnancy. Moreover, both preeclampsia and eclampsia can evolve into more severe complications that magnify the proclotting state of pregnancy and exacerbate the risk for PRS.

Cardiac Myopathy

Cardiac myopathy is a rare disorder of pregnancy that develops in the last trimester of pregnancy or in the months immediately after delivery. The reported incidence is between 1 in 3,000 and 1 in 4,000 live births (Abboud, Murad, Chen-Scarabelli, Saravolatz, & Scarabelli, 2007). Although the cause is mostly unknown and the condition frequently presents in the absence of preexisting heart disease or hypertension, advanced maternal age, obesity, multiparity, and multiple gestations are risk factors. Dilated cardiac arteries have been observed in women who subsequently experience heart failure. The cerebral hypoperfusion associated with heart failure has been implicated in AIS. Cardiac myopathy also is associated with systemic and pulmonary embolism. Mortality is high, and the need for heart transplantation is about 11% (Del Zotto et al., 2011). Women with a history of cardiac myopathy in a prior pregnancy are more likely to experience it in a subsequent pregnancy (Del Zotto et al., 2011).

Amniotic Fluid Embolism

AFE is rare, and a disruption in the barrier between the amniotic sac and the maternal circulation is the proposed

etiology. Women with advanced maternal age and multiparity are at increased risk, but the reason why AFE occurs is not well understood. The presentation is often during labor among women with rupture of the amniotic sac who develop sudden-onset dyspnea, acute hypotension, and cardiac arrest followed by coagulopathy. The mortality rate is high, ranging from 61% to 86% (Del Zotto et al., 2011).

Postpartum Angiopathy

Postpartum angiopathy is a condition associated with reversible cerebral vasoconstriction that is not necessarily associated with preeclampsia or eclampsia (Tate & Bushnell, 2011). Narrowing of the cerebral arteries is seen on angiography among women with this condition (Calabrese, Dodick, Schwedt, & Singhal, 2007). Most women who present with this condition have a history of uncomplicated pregnancy and birth. Women typically present within the first few days postpartally with a thunder-clap headache, described as the worst headache of their life (Singhal & Bernstein, 2005). Other symptoms include nausea and vomiting and central nervous system involvement including impairment of mental status (Tate & Bushnell, 2011).

Postpartum Hemorrhage

PPH may be a precursor for PRS. Although the exact etiology is unknown, vessel infarct because of extreme blood loss has been postulated as the underlying etiology of stroke because of PPH (Tate & Bushnell, 2011). Of the PRS reported in the postpartum period, 58.4% occurred in association with PPH (Bateman et al., 2006). Data from the NIS for 2000–2001 found that PPH was associated with almost a twofold increase risk in PRS (95% CI [1.2, 2.8]; James et al., 2005). In this same cohort, blood transfusion was a significant risk factor for PRS (OR = 10.3, 95% CI [7.1, 15.1]), as was postpartum infection (OR = 25, 95% CI [18.3, 34.0]).

PRS Treatment Issues

The AHA/ASA has delineated two phases of stroke care: the hyperacute phase that includes the emergency department and the acute phase occurring after patients with stroke are admitted to a stroke unit or other hospital unit (Summers et al., 2009). Issues salient to the pregnant patient during the hyperacute phase of AIS include the appropriateness of fibrinolytic therapy and/or endovascular interventions to achieve arterial recanalization. In the acute phase of stroke, the physiologic changes of pregnancy have implications for the clinical course of the patient with PRS and planning and administering nursing care.

Fibrinolytic Therapy

Reperfusion therapy with intravenous (IV) tissue plasminogen activator (t-PA) can, to varying degrees, restore blood flow to the area around the infarction, called the ischemic penumbra, that has undergone a lesser degree of ischemia because of collateral circulation (Smith, 2004). In clinical trials, t-PA was associated with better functional outcomes compared with placebo (Wardlaw et al., 2012). Current guidelines indicate that IV t-PA can be administered to eligible patients within 4.5 hours of symptom onset (Jauch et al., 2013).

Pregnancy is a relative exclusion criterion for IV t-PA (Jauch et al., 2013). Concerns about the use of t-PA in pregnant women center on the possibility of bleeding from the highly vascular placenta with the attendant risk for maternal and fetal harm (Selim & Molina, 2013) and fetal hemorrhage (Broderick, 2013). Teratogenic effects on the fetus are another hypothesized risk. However, drugs with a molecular weight greater than 400 kDa are unlikely to cross the placenta (Hockman & Kelsey, 2010), and the molecular weight of t-PA is 7200 kDa, making it an unlikely cause of teratogenicity or direct fetal effects (Leonhardt, Gaul, Nietsch, Buerke, & Schleussner, 2006).

No controlled trials of IV t-PA in pregnant women have been conducted. In a review of 28 cases in which t-PA was used in pregnancy for a variety of conditions including stroke, the authors concluded that the rates of maternal complications were similar to those seen in clinical trials with nonpregnant individuals, but two cases of fetal death may have been attributable to thrombolysis (Leonhardt et al., 2006). Del Zotto et al. (2011) summarized the results of 11 published case reports and concluded that serious maternal or fetal complications were infrequent with t-PA. Selim and Molina (2013) state that the decision to use IV t-PA during pregnancy should be individualized and made in consultation with obstetrical care givers, the patient, and her family.

Endovascular Treatment

Endovascular treatments for AIS include intra-arterial fibrinolysis (IA) and mechanical clot removal. IA may be a treatment option for AIS when the occlusion is in a large artery, exclusion criteria for IV t-PA exist, and the patient is outside the window for IV t-PA treatment because IA fibrinolysis may be administered within 6 hours of symptom onset (Jauch et al., 2013). Data about IA fibrinolysis during pregnancy come mostly from individual case studies, including a report by Johnson et al. (2005) of the successful treatment of a woman at 37-week gestation with an occlusion of the middle cerebral artery.

Mechanical removal (thrombectomy) with one of several devices approved by the U.S. Food and Drug

Administration is a treatment option for patients who do not meet the criteria for t-PA (Jauch et al., 2013). There are little data on the use of thrombectomy during pregnancy. A concern with interventional radiology procedures is possible fetal adverse effects because of radiation exposure, especially during the first half of pregnancy when the fetus is most vulnerable to such effects (Selmin & Molina, 2103).

Nursing Care for Patients With PRS

Nurses may feel unprepared to provide care for pregnant women with an acute condition such as stroke (Perozzi & Englert, 2004). It is not within the scope of this article to provide an in-depth description of nursing care of the patient with acute stroke, which has been described in an AHA/ASA guideline (Summers et al., 2009). The primary aim of such care is to assess for and prevent deterioration of the patient's condition through the use of validated stroke scales to detect changes in the patient's neurological status, blood pressure and temperature management, cardiac monitoring to detect arrhythmias, and blood glucose measurement to prevent complications related to hyperglycemia (Summers et al., 2009). Awareness of how physiological changes of pregnancy may affect the clinical course of a patient with PRS will assist nurses to anticipate care needs. In addition, nurses must consider both the mother and fetus/baby when planning and providing care.

Antepartum

The upper airway of the pregnant woman often is edematous because of capillary engorgement secondary to the increased blood volume of pregnancy (Munnur, de Boisblanc, & Suresh, 2004). An edematous airway may present difficulties with intubation should the need for mechanical ventilation arise. In addition, hormonally mediated lower esophageal sphincter relaxation coupled with alterations in the position of the gastroesophageal junction by the uterus place pregnant women at increased risk for aspiration (Munnur et al., 2004). Patients with stroke with dysphagia often are at increased risk for aspiration, and nurses should assess swallowing capability by closely observing for delay initiating swallowing, coughing, or choking during ingestion of food or drink (Summers et al., 2009).

Several pregnancy-related pulmonary changes are relevant to the care of women with acute medical conditions. Tidal volume, the amount of air inhaled and exhaled in one respiratory cycle, increases significantly to meet the increased oxygen demands of pregnancy (Cunningham et al., 2010). As pregnancy progresses, functional residual capacity and residual volumes are decreased as a consequence of elevation of the diaphragm by the enlarging uterus. These physiologic changes result in a mild degree of respiratory alkalosis

(Fujitani & Baldisseri, 2005). Mild dyspnea is common in pregnant women and may be more pronounced when women are in the supine position (Brokl & Baldisseri, 2010), and slight elevation of the head of the bed may ease breathing. It is important for nurses to be alert for sudden onset or worsening of dyspnea, which may indicate potentially serious conditions including postpartum cardiomyopathy, embolic disorders, and pulmonary edema (Soubra & Guntupalli, 2005). Because of decreased functional residual capacity and increased oxygen consumption, oxygen reserves of both mother and fetus are reduced, placing both mother and fetus at increased risk for hypoxia (Fujitani & Baldisseri, 2005). An arterial oxygen saturation of 94% or greater is recommended for pregnant patients (Brokl & Baldisseri, 2010).

After 24 weeks of gestation, compression of the inferior vena cava by the gravid uterus when women are in the supine position reduces venous return to the heart with a subsequent decrease in cardiac output, causing episodes of hypotension and bradycardia (Fujitani & Baldisseri, 2005; Yeomans & Gilstrap, 2005). Pregnant women, especially those in the third trimester when the uterus is large, should be positioned on their left side to facilitate venous return, prevent supine hypotension, and lessen aspiration risk. Pregnant women also may be at increased risk for fluid overload and pulmonary edema because of decreased colloid osmotic pressure and a decrease in the colloid osmotic pressure–pulmonary capillary occlusion pressure gradient (Yeomans & Gilstrap, 2005).

Patients with stroke with impaired mobility are at risk for venous thromboembolism (VTE), a condition that includes DVT and pulmonary embolism (Anderson & Spencer, 2003). Pregnant and postpartum women already are at an elevated risk for VTE, with a relative risk of 4.29 (95% CI [3.49, 5.22]), and the incidence of VTE is five times greater in the postpartum period than during pregnancy (Heit et al., 2005). Unfortunately, DVT may be asymptomatic or mistaken for pregnancy-related physiologic changes such as edema of the legs (Bates & Ginsberg, 2001). Nurses have an important role in the recognition and prevention of VTE by regularly assessing for pain and swelling of an extremity and calf asymmetry, promptly reporting suspected abnormalities to facilitate timely diagnostic testing and encouraging mobility as soon as patients are stable (Flanders & Gunn, 2011).

Low-molecular-weight heparin (LMWH) at doses lower than used to achieve anticoagulation and intermittent pneumatic compression devices are recommended as VTE prophylaxis for AIS patients with restricted mobility (Lansberg et al., 2012). LMWH is preferred over unfractionated heparin for VTE prevention and treatment because it is associated with less

bleeding and can be administered less frequently and by the subcutaneous route (Greer & Nelson-Piercy, 2005). It may be used for VTE prophylaxis in patients with hemorrhagic stroke once there is evidence that bleeding has stopped (Morgenstern et al., 2010). At therapeutic doses, LMWH is used to treat strokes because of CVT (Saposnik et al., 2011) but has not been shown to improve outcomes or prevent neurological deterioration after AIS (Jauch et al., 2013).

A systematic review of LMWH use in pregnancy found it effective for VTE prevention and treatment, and the 2% risk for severe bleeding found in the review was considered by the authors within an acceptable range of reported rates of pregnancy-associated hemorrhage (Greer & Nelson-Piercy, 2005). Unlike vitamin K antagonists such as warfarin that are associated with fetal bleeding and anomalies, LMWH is unlikely to cause direct fetal effects because of its large molecular weight (Bates, Greer, Pabinger, Sofaer, & Hirsh, 2008). Nurses should routinely assess patients on anticoagulant therapy for signs of bleeding, which may be manifested by epistaxis, bleeding gums, hemoptysis, hematuria, or melena (Moore, Hendrix, Conti, & Guzman, 2011).

Nurses providing care for a pregnant woman with stroke have two patients. Depending on the woman's condition, fetal heart rate assessment should be done every 4 hours at a minimum using intermittent Doppler auscultation or intermittent or continuous electronic fetal heart rate monitoring (EFM). The more critical the condition of the mother, the more frequent fetal heart assessments should be done. A normal baseline fetal heart rate is between 110 and 160 beats per minute. It is unlikely that nurses outside the obstetrical unit will assume primary responsibility for EFM or the interpretation of EFM data, and obstetrical nurses and obstetricians ideally are part of the interdisciplinary team providing care to pregnant women in acute care settings (Simpson, 2004). Each seriously ill pregnant patient should have a delivery plan that balances the risks and benefits of continuing the pregnancy and anticipates the possibility of delivery by cesarean section should the condition of the mother or fetus deteriorate (Brokl & Baldisseri, 2010).

Postpartum

The obstetrical care needs of a postpartum woman consist of the prevention of infection and excessive uterine bleeding and attending to lactation. The upper limit for blood loss associated with a vaginal delivery is 500 cc; uterine bleeding should be regularly observed, and the amount recorded and consultation should be sought with the obstetrical care provider if bleeding is excessive. A full bladder may be a cause of excessive uterine bleeding, and nurses should monitor voiding in patients without catheters. Massage of the upper part of the

uterus to reduce excessive bleeding may be necessary if there is evidence that the uterine muscle is not contracting properly (Perry, Hockenberry, Lowermilk, & Wilson, 2010). Because nurses outside the obstetrical unit may be unfamiliar with the acceptable limits of postpartum blood loss and the correct technique for uterine massage, obstetrical nurses may assume responsibility for these aspects of care.

Oxytocin (Pitocin) may be administered after delivery to control excess bleeding. Oxytocin usually is given as an IV infusion in which 20–40 units of Pitocin are added to 1000 cc of normal saline or Ringer's lactate solution and run continuously (King & Brucker, 2011). Intramuscular methylergonovine maleate (Methergine) is sometimes used to treat uterine atony but should not be used in women with hypertension because of vasoconstrictive properties (King & Brucker, 2011). Uterine cramping is common after birth, possibly because of the release of prostaglandins, and is effectively treated with nonsteroidal anti-inflammatory medications.

If a woman was breastfeeding at the time of stroke, it is likely that lactation will be disrupted. Depending on the clinical status of the mother, support should be provided for the continuation of breastfeeding or the maintenance of the milk supply through breast pumping. Most postpartum units have breast pumps, and nurses can request assistance with breast pumping from maternal-child nurses to maintain milk supply. If lactation consultants are employed at the hospital, they are an ideal resource to assist with the continuance of breastfeeding. If a postpartum woman was breastfeeding before her stroke, nurses should advocate for the infant to be brought to the hospital for breastfeeding if the mother's condition allows.

Medications used to treat patients with stroke may be secreted in the breast milk, prompting concerns about the effect on the infant. Molecular weight, plasma protein binding, and dissolvability in lipids are some of the factors that affect drug excretion (Ito & Lee, 2003). According to the American Academy of Pediatrics Section on Breastfeeding (2012), few medications are contraindicated during breastfeeding with the exception of amphetamines, chemotherapy agents, statins, and ergotamines. Although data on the use of anticoagulants among breastfeeding women are limited, guidelines from the American College of Chest Physicians indicate that breastfeeding does not need to cease during treatment with unfractionated heparin, LMWH, and warfarin (Bates et al., 2008).

If neither breastfeeding nor breast pumping is desired or feasible, a supportive bra worn for 72–96 hours after delivery and cold packs applied to the breasts can be used to suppress lactation and minimize discomfort associated with breast engorgement (Perry et al., 2010). Analgesics may be prescribed as necessary. Medications

to suppress lactation, including estrogen and estrogen–testosterone preparations, are no longer in use because of adverse side effects that included an increased risk for VTE (King & Brucker, 2011).

The psychosocial implications of postpartum stroke center on minimizing the disruption of maternal–infant bonding and addressing anxiety on the part of the mother and/or her family about the implications of the stroke for the woman's future health and functioning. When serious illness occurs in a pregnant or postpartum woman, the nurse should provide accurate and timely information to the woman and her family in a caring manner and facilitate family access to the woman (Perozzi & Englert, 2004). As the mother's condition allows, nurses should support and encourage maternal and family contact with the infant.

Although most women with PRS survive, the mortality rate for hemorrhagic stroke is not inconsiderable, and in this unfortunate situation, nurses should be prepared to respond with acceptance and understanding to the emotional reactions exhibited by grieving family members (Perozzi & Englert, 2004). Among survivors of PRS, neurological deficits may include motor weakness, dysphagia, speech and language disorders, memory problems, altered behavior, and confusion. In anticipation of hospital discharge, the coordinated efforts of the nursing and medical staff, social services, speech and language pathologists, and rehabilitation specialists are needed to assess patient needs and devise a plan to address those needs. Nursing strategies to facilitate effective discharge planning include educating patients and their families about secondary stroke prevention and stroke warning signs, encouraging family participation in rehabilitation sessions, and involving families in the decision-making process about posthospital care (Summers et al., 2009).

Conclusion

Although PRS remains rare, the incidence has increased during the last several decades, possibly because of a rise in stroke risk factors among young women. The physiological changes of pregnancy place pregnant and postpartum women at increased risk for stroke, and several conditions specific to pregnancy further increase risk. The provision of optimal care to this unique population of individuals requires awareness of how physiological changes of pregnancy may affect the course of stroke and nursing actions.

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