Intracranial Hemorrhage in the Neonate

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The purpose of this article is to review the pathophysiology, presentation, and management of intracranial hemorrhage in the neonate.

Abstract

Intracranial hemorrhage is a serious cause of morbidity and mortality in the neonate. Subgaleal, subdural, subarachnoid, and intraventricular hemorrhage have varying pathophysiology, but each can have serious long-term consequences. This article reviews the pathophysiology, presentation, and outcomes for intracranial hemorrhage in the newborn, as well as potential therapeutic interventions.

Keywords: neonate; intracranial hemorrhage; subgaleal hemorrhage; subdural hemorrhage; intraventricular hemorrhage

The neonatal period and early infancy constitute a critical window of brain development. These times are also periods of risk for intracranial bleeding, which can range in severity from common and benign to potentially devastating. Understanding the pathology and location of different types of bleeds in the neonate facilitates predicting associated morbidity and mortality. It is crucial that nurses and clinicians recognize signs and symptoms of intracranial hemorrhage to facilitate appropriate management. We discuss specific categories of intracranial hemorrhage as classified by location, beginning with extra-axial blood collections and proceeding inward (Table 1).

SUBGALEAL HEMORRHAGE

Pathophysiology

Subgaleal hemorrhage occurs when the emissary veins between the skull and the intracranial venous sinuses are sheared or torn, and blood collects in between the aponeurosis and the periosteum of the skull (Figure 1).

Incidence

The described incidence of subgaleal hemorrhage (also called *subgaleal hematoma*) ranges from 1.5 to 30.0 per 10,000, with higher rates for vacuum-assisted or forceps extractions.[1–4] Subgaleal hemorrhage is more common after mechanically assisted delivery because of the external shearing pressures placed on the veins as the compliant neonatal skull is pulled from the birth canal. Subgaleal hemorrhage is also more common in term infants than preterm infants, partially because most preterm infants have smaller heads, with less skull deformation and less resulting shearing pressures during delivery.

Diagnosis and Management

Subgaleal hemorrhage may present as a large, boggy fluid collection palpable on the head's surface. Characteristic of a subgaleal hemorrhage is that it is not restricted by suture lines and may shift with movement. This is in contrast to the much more common cephalohematoma, a superficial collection of blood restricted to the space between the periosteum and skull, which is contained along suture lines. Neonates with subgaleal hemorrhage are at high risk for rapid decompensation; the subgaleal space can expand to collect a newborn's entire intravascular blood volume if bleeding continues unrecognized. Nurses or clinicians may observe swelling of the ears or increasing head circumference as bleeding expands into this space. As a subgaleal hemorrhage progresses, neonates can compensate up to a point and then quickly decompensate with hypovolemic shock, including tachycardia, hypotension,

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TABLE 1 ■ Categories of Neonatal Intracranial Hemorrhage

Type of Hemorrhage	Location	Risk Factors	Clinical Management
Subgaleal	Between the galeal aponeurosis and the periosteum (just outside the skull)	Vacuum- or forceps-assisted delivery Coagulopathy	Early identification Monitor for signs of hypovolemia and shock May require emergent volume repletion or transfusion
Subdural	Between the dura mater and the arachnoid mater (within the skull, outside the brain)	Vacuum- or forceps-assisted delivery Coagulopathy	Monitor for clinical evidence of expansion In severe cases, secondary seizures or encephalopathy may be present and require management. Neurosurgical drainage rarely indicated
Subarachnoid	Below the arachnoid mater (on the surface of the brain)	Vacuum- or forceps-assisted delivery Coagulopathy	Supportive management Monitor for development of secondary hydrocephalus
Intraventricular	Originates in the germinal matrix (adjacent to the ventricles)	Prematurity Chorioamnionitis Hypotension Acidosis Respiratory distress Bicarbonate therapy Coagulopathy	Monitor for anemia and hypovolemia In severe cases, monitor for posthemorrhagic hydrocephalus

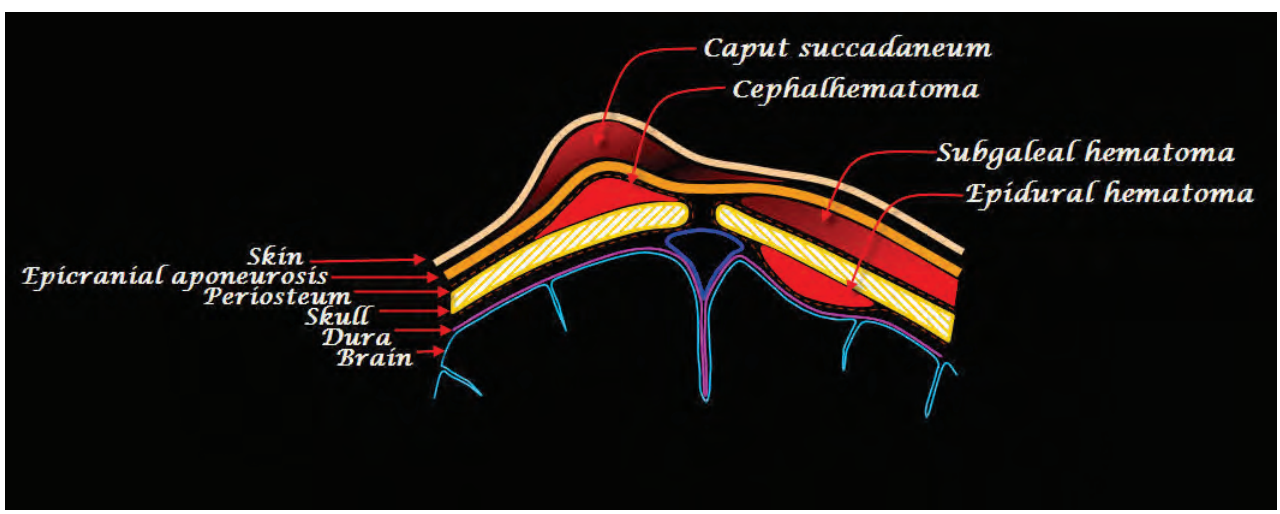
and acidosis. Thus, when these signs are identified, they should be brought to the attention of the treating clinician immediately. Any neonate with a suspected subgaleal hemorrhage should be transferred to a NICU with the ability to perform resuscitation, place central lines, and transfuse blood emergently (see Table 1).

For the asymptomatic, stable neonate with a subgaleal hemorrhage, observation of vital signs and serial hematocrits is essential. Close nursing observation for at least 24–48 hours prior to discharge is necessary, as well as documentation of a normal neurologic exam, observation of good feeding, and a plan for close follow-up.

Prognosis

When subgaleal hemorrhage does not impact overall hemodynamics, outcomes are typically very good. If there is hemodynamic instability, the risk for subsequent neurodevelopmental impairment largely depends on the extent of hypovolemic shock. In one study, mortality with severe subgaleal hemorrhage was 12 percent, whereas the mortality was

FIGURE 1 ■ A schematic diagram illustrating the location of intracranial hemorrhage by subtype.



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0 percent with mild or moderate subgaleal hemorrhage. Of those with mild or moderate hemorrhage, most had a normal neurologic exam prior to discharge.[5]

SUBDURAL HEMORRHAGE

Pathophysiology

Subdural hemorrhages are the most common type of intracranial bleeding in neonates, although they are often so small that the majority are asymptomatic. A subdural hemorrhage occurs when bridging veins carrying blood through the dura mater to the arachnoid mater of the meninges are torn. This causes bleeding, with blood collecting below the dura and superior to the subarachnoid villi.

Incidence

As with subgaleal hemorrhage, the prevalence of subdural hemorrhage is higher with vaginal compared with cesarean deliveries.[6,7] The use of instrumentation, such as vacuum or forceps, increases the rate of subdural hemorrhages. The most current published data suggest that clinically identified subdural hemorrhages occurred in 2.9 per 10,000 spontaneous deliveries, as compared with 8.0 and 9.8 per 10,000 in vacuum-assisted and forceps-assisted deliveries, respectively. When both vacuum and forceps are used in delivery, the rate goes up to 21.3 per 10,000.[8] These data reflect a population-based study from 1999. More recent studies suggest that the prevalence of asymptomatic subdural hemorrhages in newborns varies from 8 to 45 percent.[7,9] The most common location for these small hemorrhages is along the tentorium or near the falx (see Table 1).

Diagnosis and Management

Because subdural bleeds are located within the skull, there is often no physical sign on the scalp that reflects injury. Instead, the presence of hemorrhage may initially be unrecognized. For most neonates, subdural hemorrhage remains asymptomatic and resolves without consequence. Clinical problems can arise in the case of large-volume hemorrhage or if bleeding slowly continues over hours or even days, as in cases of bleeding disorders. Symptomatic neonates often present 24–48 hours after birth with nonspecific signs such as apnea, respiratory distress, altered neurologic state, or seizures. Neuroimaging is required for diagnosis. Computed tomography (CT) scan is highly accurate for diagnosing subdural hemorrhage and in some cases can be obtained more quickly than magnetic resonance imaging (MRI).[9] However, CT scan requires exposure to radiation. MRI is also highly accurate and does not require radiation; MRI is therefore preferred when feasible. Head ultrasound sometimes allows visualization of subdural blood; in those cases, serial cranial ultrasound may be used to follow evolution of hemorrhage. However, ultrasound cannot always identify collections close to the surface of the head and thus is not reliable as a sole means of excluding the diagnosis of subdural hemorrhage.

Most neonates with subdural hemorrhage can be managed symptomatically by identifying and addressing any secondary symptoms that may be present. Serial hematocrits and vital signs should be monitored frequently until stable. In most cases, blood collections will gradually resorb over the weeks and months following the initial hemorrhage. Rarely, in the case of large subdural hemorrhage causing increased intracranial pressure or mass effect, neurosurgical drainage may be required.

Prognosis

In most neonates with subdural hematomas, outcomes are thought to be generally good, with one study showing up to 80 percent of infants with subdural hemorrhages with no disability.[10]

SUBARACHNOID HEMORRHAGE

Pathophysiology

A subarachnoid hemorrhage (SAH) occurs when the veins of the subarachnoid villi are torn, leading to a collection of blood in the subarachnoid space (see Table 1).

Incidence

The prevalence of SAH is 1.3 per 10,000 spontaneous vaginal deliveries, with a higher prevalence associated with vacuum-assisted or forceps deliveries.[8]

Diagnosis and Management

Neonates with SAH may present similarly to those with subdural hemorrhage. In addition, neonates with subarachnoid hemorrhage may also present with seizures, as the blood from the SAH may irritate the meninges and adjacent cortex.[9] As with all neonatal seizures, these seizures may manifest as posturing, subtle movements, or apnea and require EEG for accurate diagnosis. Treatment of seizures is with antiseizure medications.

In addition, in some cases with large SAH, irritation of the meninges can result in secondary impairment of cerebrospinal fluid (CSF) resorption.[9] If this happens, hydrocephalus can develop. As such, following SAH, neonates should receive at least serial head circumference measurements, and in some cases, serial head ultrasounds, to screen for hydrocephalus.

Prognosis

The outcomes for neonates with SAH are thought to be generally good, although the location and extent of the hemorrhage may play a role. For example, hemorrhage in the frontal lobe or hemorrhage in multiple areas was found to be associated with higher rates of disability.[11]

INTRAVENTRICULAR HEMORRHAGE

Pathophysiology

Preterm neonates are especially vulnerable to intraventricular hemorrhage (IVH) primarily because of the vulnerability of the germinal matrix. The germinal matrix is located adjacent to the ventricles and in the growing fetus is responsible for producing neurons and glial cells.[12] The germinal matrix normally has a rich blood supply until the fetus matures, at which point blood flow redistributes. In the premature neonate, the blood vessels of the germinal matrix lack significant structural support, which increases the risk of rupture, especially in the setting of altered cerebral blood flow autoregulation.[13]

Many studies have looked at risk factors associated with IVH. Chorioamnionitis, or inflammation of the amniotic sac, has been associated with a 1.6-fold increased risk of IVH.[14] Other factors associated with IVH include hypotension, acidosis, respiratory distress associated with hypocapnia, hypercapnia or hypoxemia, mechanical ventilation, and bicarbonate therapy.

Incidence

Although prior studies have estimated the prevalence of IVH in preterm neonates as high as 50 percent,[15,16] more recent studies have estimated the prevalence closer to 17.5 percent in neonates weighing less than 1,500 g.[17] Risk factors associated with the development of IVH include low birth weight, male sex, short gestation, respiratory distress syndrome (RDS),[16] hypercarbia,[16] fluctuating blood pressure, and hypotension.[18]

There are several prenatal and postnatal factors that may have contributed to the decreased prevalence of IVH. The administration of antenatal steroids has had a large effect in reduction of IVH.[19] In the Cochrane Review, the relative risk of cerebroventricular hemorrhage was 0.54 for those neonates who had received antenatal steroids, and the relative risk of severe cerebroventricular hemorrhage was 0.28.[20] Antenatal steroids are thought to work by (1) preventing RDS and the need for prolonged ventilation and (2) constricting fetal vessels that balance the vasodilation that occurs with hypercarbia.

Immediately after birth, delayed cord clamping is thought to help prevent IVH by increasing the blood volume and reducing hemodynamic instability. A randomized controlled trial showed the IVH rate in neonates with immediate cord clamping was three times that of those who received delayed cord clamping.[21] Similarly, several studies have shown that indomethacin reduces the frequency and severity of IVH. Indomethacin is a cyclooxygenase inhibitor of prostaglandin synthesis that in animal models promotes maturation of the germinal matrix and modulates cerebral blood flow. In a multicenter randomized controlled trial, 12 percent of neonates treated with indomethacin beginning at 6–12 hours of life had IVH, in comparison with 18 percent of neonates treated with placebo ($p = .03$).[22] Subsequent analysis done by Ment and colleagues showed a statistically significant reduction in IVH in boys, without the same effects on girls (relative risk of 0.34 for boys vs 1.16 for girls).[23] However, indomethacin is not routinely used in NICUs for IVH prevention, in part because of its side effect profile.

Postnatally, clinical practices have been shown to reduce the incidence of IVH. Maintaining a midline head position for premature neonates can be beneficial because it allows for unobstructed drainage of the jugular veins, which prevents venous congestion that may make neonates prone to IVH.[24] In addition, maintenance of adequate blood pressure also reduces the incidence of IVH.[25]

Diagnosis

The diagnosis of IVH remains problematic for clinicians. There are very few clinical symptoms of IVH. When present, signs may include an acute drop in hematocrit, new-onset hypotension, and lethargy. However, these symptoms are often present in extremely low birth weight and premature neonates because of other common morbidities and are not pathognomonic of IVH. Thus, signs may not be readily recognized because of IVH. Most clinicians rely on routine ultrasound screening to detect the presence of IVH. Because most IVH occurs within the first three days, most institutions will perform an ultrasound within the first week of life. The American Academy of Neurology recommends that every neonate <30 weeks gestational age (GA) at birth get a routine head ultrasound by 7–14 days and ideally again at 36–40 weeks postmenstrual age.[26]

Newer technologies also support understanding of IVH. Near-infrared spectroscopy (NIRS) is a bedside monitor that measures real-time brain tissue oxygenation, which can reflect cerebral blood flow and loss of autoregulation.[27] Studies have correlated changes on the NIRS monitor with increased IVH in preterm neonates.[28,29] Similarly, amplitude-integrated electroencephalogram (aEEG) is another bedside tool that measures brain electrical activity. Neonates with severe IVH have depressed background activity on aEEG, and the evolution of IVH to posthemorrhagic hydrocephalus may sometimes be seen on aEEG.[30] In this way, new modalities may be complementary in understanding changes in physiology that accompany IVH, although imaging remains the mainstay of diagnosis.

Traditionally, the Papile grading system has been used to describe the severity of IVH[15] (Table 2). However, this classification does not take into account the severity of the involvement of the parenchyma (the extent of the Grade IV bleed) nor does it take into account bilaterality of IVH, which can both significantly change prognosis. Although there is a need for an improved scoring system to describe severity of IVH, there is no consensus on the use of alternate grading systems.[31]

TABLE 2 ■ Papile Grading System of Intraventricular Hemorrhage[15]

Grade	Description
I	Blood in the periventricular germinal matrix regions or germinal matrix hemorrhage
II	Blood within the lateral ventricular system without ventricular dilation
III	Blood acutely distending the lateral ventricles
IV	Blood within the ventricular system and intraparenchymal hemorrhage

Management

Once IVH has occurred, clinicians provide supportive care to help reduce further damage by correcting acidosis, repleting intravascular volume and maintaining hemodynamic stability. Blood is eventually resorbed over the course of weeks.

During the period following larger (Grade III or IV) IVH, it is important to monitor the neonate via ultrasound for posthemorrhagic hydrocephalus. Posthemorrhagic hydrocephalus occurs in approximately 29 percent of neonates with IVH and typically develops 2–6 weeks after the initial hemorrhage.[32] When progressive, it is associated with worse outcomes and may require neurosurgical intervention. Posthemorrhagic hydrocephalus is thought to be caused by inflammation and obstruction of the subarachnoid villi by blood, leading to impaired CSF resorption and subsequent hydrocephalus. Various treatments have been studied in attempts to decrease the risk of posthemorrhagic hydrocephalus after IVH; these include use of acetazolamide to decrease CSF production, thrombolysis with intraventricular fibrinolytics, and CSF irrigation.[32] Unfortunately, none of these have been proven effective in improving long-term outcomes for neonates at risk for posthemorrhagic hydrocephalus.[32]

Prognosis

Prognosis following IVH depends in large part on the severity of the bleed. Historically, severe bleeds (Grade III–IV) were associated with up to 20 percent mortality and a higher risk of neurodevelopmental impairment including cerebral palsy (CP), blindness, and motor and cognitive delays.[33] More recent data suggest that multiple factors contribute to predicting death and neurodevelopmental impairment, including the severity, location (bilateral vs lateral), and presence of hemorrhagic parenchymal infarction.[34] Risk of subsequent CP is clearly associated with severity of IVH; in one study, CP occurred in up to 50 percent of neonates with Grade IV IVH, as compared with 8 percent of neonates with Grade I IVH.[35]

For low-grade IVH (Grade I–II), there are mixed data on the outcomes. In a study by the Neonatal Research Network, extremely low birth weight neonates with Grade I or II IVH had no significant difference in rates of CP or gross motor, cognitive, and language scores at 18–22 months when compared with neonates with no IVH.[36] Other studies, however, have showed that a Grade I or II IVH was associated with slightly higher rates of CP and total neurodevelopmental impairment compared with those neonates without IVH.[37,38] Further research is needed to determine if these differences remain at school age.

CONCLUSION

Intracranial hemorrhage is a common problem in the neonate. Extra-axial bleeds are associated with instrumental deliveries, whereas IVH most often occurs because of prematurity. The impact on morbidity and mortality ranges widely. Early diagnosis by the neonatal team can facilitate interventions that may improve neurodevelopmental outcomes.

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