Newborn Birth Injuries

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Introduction

Birth injuries, although declining due to improvements in obstetric care and prenatal diagnosis, remain a significant cause of neonatal morbidity and mortality and are a source of great concern for the parents, obstetricians, pediatricians, and other healthcare providers.

There is a wide spectrum of birth injuries that range from minor and self-limited to severe. Often injuries occur due to risk factors such as macrosomia, prematurity, forceps delivery, vacuum extraction, abnormal fetal presentation, prolonged labor, and precipitous delivery [1, 2], but damage can also occur in utero before initiation of birth process and in the absence of any identifiable risk factors.

At times, signs and symptoms may not be apparent immediately after birth due to the presence of other associated clinical problems. Some injuries may become more evident at the time of or after discharge. In order to initiate appropriate treatment, it is important for clinicians to remain alert to the possibility that birth injuries may become apparent even after newborns are discharged from the hospital.

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In most cases, management of soft tissue injuries requires only careful observation and follow-up. However, in other instances such as subgaleal hemorrhage, early recognition and immediate intervention is required for survival.

It is important that clinicians are able to recognize and manage birth injuries and provide appropriate counseling to parents regarding prognosis. Misdiagnosis and/or mistreatment can have significant impact on both short-term and long-term well-being of a child. Counseling of parents regarding the severity of birth injuries and associated prognosis helps in establishing expectations regarding the outcome and avoiding misunderstandings.

Case Presentation

You are evaluating a 12-h-old male neonate with estimated gestational age of 38 weeks, birth weight 3.8 kg with a scalp swelling, weakness, and pallor of the body. The mother G1P0 received antenatal care in a private hospital, kept all appointments, and used only the prescribed medications. She delivered vaginally by vacuum extraction; Apgar score was 7/8. Physical examination revealed a hypotonic, pale infant with diminished peripheral pulses and a weak cry. The anterior fontanelle was full, and a fluctuant mass with bruised skin on the posterior aspect of the head was noted.

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At this time, you think the swelling on the scalp is:

1. Caput succedaneum
2. Cephalohematoma
3. Subgaleal hemorrhage
4. Subarachnoid hemorrhage

Extracranial injuries (Fig. 2.1) occur above the parietal layer of the skull. They include caput succedaneum, cephalohematoma, subgaleal hemorrhage, and skull fractures.

Caput Succedaneum

Caput succedaneum (Fig. 2.2) is characterized by a vaguely demarcated area of edema over the scalp that was the presenting part during a vertex delivery. The edema is due to a serosanguinous fluid collection a few millimeters thick above the periosteum that presents as a soft tissue swelling of the scalp. Because of the location external to the periosteum, it may extend across the suture lines and can be confused with subgaleal hemorrhage, the more serious form of extracranial injury. Careful assessment is necessary to avoid misdiagnosis, which can have potentially catastrophic results. Firm, constant pressure in one spot is the easiest way to elicit the characteristic pitting edema of caput succedaneum.

Caput succedaneum is easily differentiated from a cephalohematoma as the swelling is above the periosteum and crosses the suture lines although occasionally a bilateral cephalohematoma can be difficult to distinguish from a caput succedaneum.

Cephalohematoma

A cephalohematoma is subperiosteal collection of blood caused by rupture of diploic blood vessels that traverse from skull to periosteum. Repeated buffeting of the fetal skull against the maternal pelvis during a prolonged or difficult labor and mechanical trauma from forceps or vacuum devices during delivery have been implicated [1]. Cephalohematomas may not be apparent at birth and may develop incrementally during

Fig. 2.2 Newborn at 10 min of life with caput succedaneum

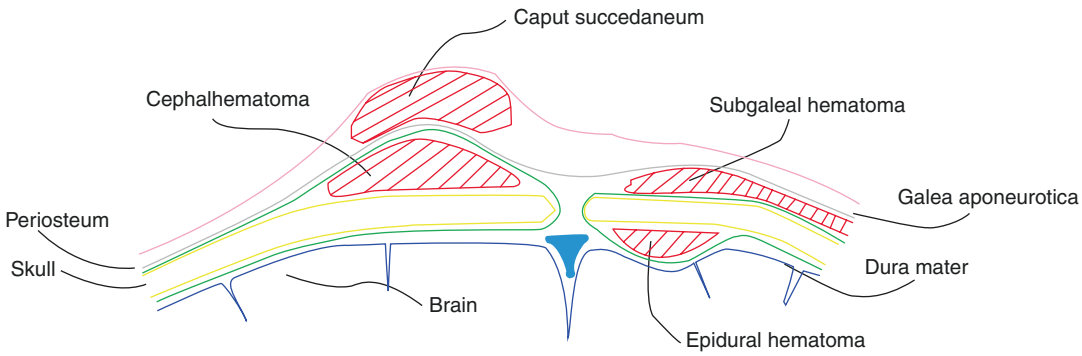

Fig. 2.1 Extracranial injuries in the planes of the scalp

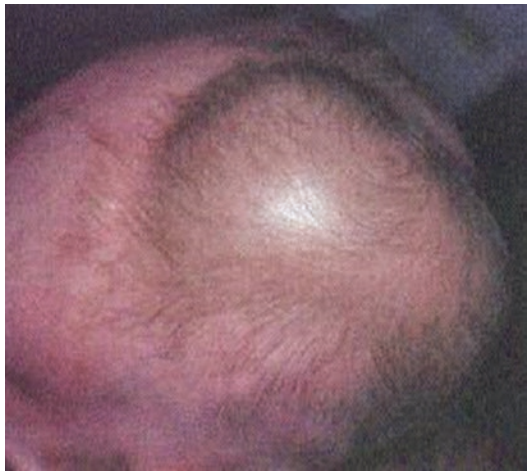

Fig. 2.3 Unilateral left parietal cephalohematoma

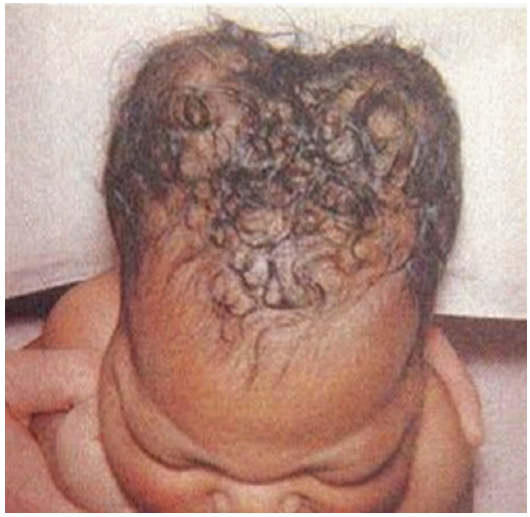

Fig. 2.4 Bilateral parietal cephalohematomas

the first 24 h of life and present as either a unilateral (Fig. 2.3) or bilateral (Fig. 2.4) palpable mass, often over a parietal or occipital bone.

Because the blood is subperiosteal, the swelling does not extend across the suture lines and is the distinguishing feature of cephalohematoma.

Subgaleal Hemorrhage

A subgaleal hemorrhage (SGH) is a potentially fatal lesion that results from bleeding under the epicranial aponeurosis. The epicranial aponeurosis is a sheet of fibrous tissue that extends from the orbital ridges anteriorly to the nape of the neck posteriorly and to the level of the ears laterally, creating a subgaleal or subaponeurotic space. Thus, SGH can spread across the entire calvarium. SGH is most often associated with vacuum-assisted deliveries which may produce a shearing force to the scalp, thereby tearing large emissary veins. This risk increases further in deliveries in which both forceps and vacuum extraction are used [1]. Infants with SGH present initially with pallor, poor tone, and fluctuant swelling on the scalp that can rapidly result in shock [3]. As the hemorrhage progresses, it may displace the ears anteriorly, and periorbital edema may develop (Fig. 2.5). Other symptoms may include signs of pain, particularly when the head or scalp is manipulated.

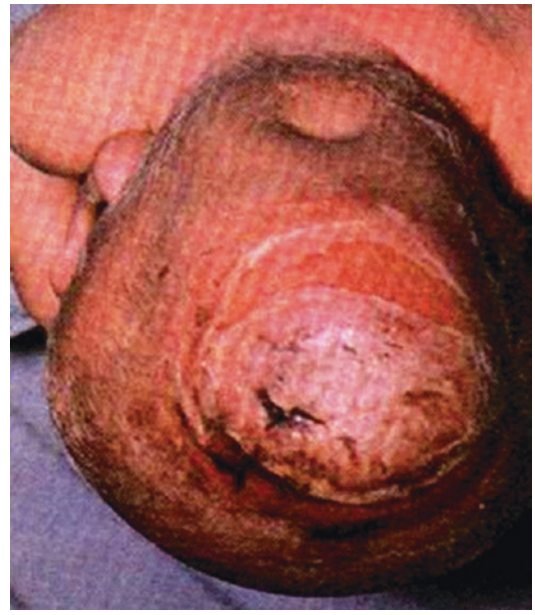

Fig. 2.5 Severe scalp edema and abrasion of the scalp in an infant with subgaleal hemorrhage delivered by vacuum extraction

In any extensive SGH, an underlying bleeding disorder such as thrombocytopenia, vitamin K deficiency, hemophilia, and disseminated intravascular coagulation must be considered in the etiology. Clotting abnormalities not only predispose the infant to bleeding but also may contribute to the extension of a relatively insignificant

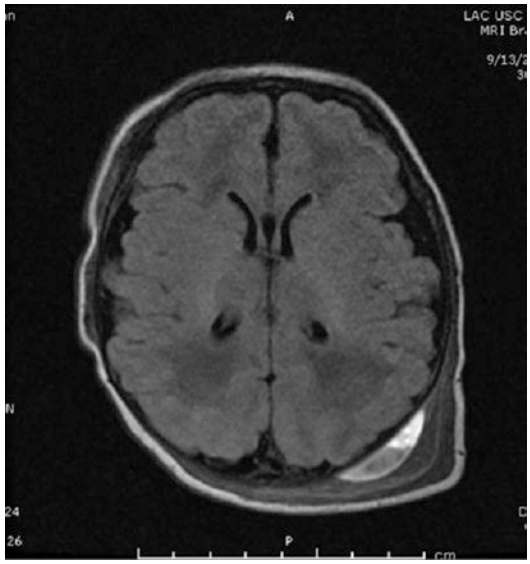

Fig. 2.6 MRI of the brain showing cephalohematoma beneath subgaleal hematoma

hemorrhage into a larger, more serious loss of blood. A coagulopathy may be related to consumption of clotting factors and platelets by the hematoma [4].

The infant in the vignette was found to have subgaleal hemorrhage and a cephalohematoma on MRI scan as seen in (Fig. 2.6).

Answer: 3

At this time you are concerned about:

1. Anemia
2. Jaundice
3. Infection
4. Shock from blood loss

It is rare to have significant blood loss or jaundice with caput. Blood loss in cephalohematoma is limited by periosteal attachment to the scalp bone.

Jaundice in infants with cephalohematoma and SGH is common but not in the first 24 h, usually occurs later than classic physiologic hyperbilirubinemia following the breakdown of the red blood cells (RBCs) in the cephalohematoma and in the SGH.

Fig. 2.7 Infant with scalp abrasion and infection following vacuum delivery

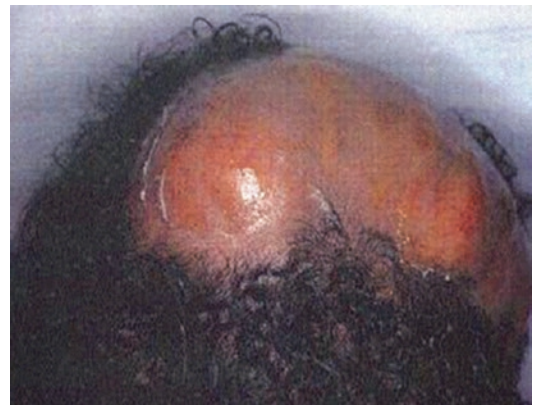

Fig. 2.8 Infant with infected cephalohematoma

Physicians should be aware that caput, cephalohematoma, and SGH are rare but potential sites for infection which may be caused either by direct traumatic scalp lesions (Fig. 2.7) or hematogenous extension [5]. Infected cephalohematoma presents as erythematous, fluctuant, painful mass that may have expanded from its baseline size (Fig. 2.8). Imaging with computed tomography (CT) or magnetic resonance imaging (MRI) may be helpful in making the diagnosis. Untreated infected cephalohematomas and SGH may lead to osteomyelitis, epidural abscess, or subdural empyema, and hence appropriate antibiotic treatment and surgical debridement are necessary when an infection occurs.

The subgaleal area is not limited by sutures, and therefore there are no barriers to prevent extension of bleeding, and hence a massive

hemorrhage can quickly occur. The subaponeurotic space can hold up to 260 ml of blood (newborn's estimated blood volume is 80–100 ml/kg). Most subgaleal hemorrhages develop slowly over several hours to days. Mean age at onset of symptoms is 9 h but may occur sooner if hemorrhage is severe. Extracranial swelling with associated tachycardia and pallor should be a concern for blood loss and hypovolemic shock. Early recognition and management of this injury are crucial for good outcomes [6].

Answer: 4

How would you manage this patient?

1. Observation
2. Blood transfusion
3. Normal saline transfusion
4. Skull radiograph
5. Immediate CT/MRI scan

Management differs for each of the extracranial lesion. In cases of caput, the swelling subsides over the next few days, and observation is all that is necessary. In cases of uncomplicated cephalohematomas, no specific treatment is indicated, but anemia and hyperbilirubinemia may be treated as needed. The lesion gets absorbed within 2 weeks to 3 months. If neurologic symptoms develop or concerns regarding the possibility of a depressed skull fracture exist, a CT scan or MRI may be indicated to rule out intracranial pathology [7]. The skull radiographs below (Fig. 2.9a, b) show diffuse soft tissue swelling and do not offer lot of information unless there is underlying skull fracture.

Treatment of SGH includes carefully monitoring for the classic triad of clinical findings including tachycardia, a falling hematocrit, and increasing head circumference in the first 24–48 h after birth. These factors are particularly important in those infants who are considered stable enough to allow admission to the normal newborn nursery [6].

For infants presenting with shock, volume resuscitation is required. This may be achieved with normal saline, packed RBCs, fresh frozen plasma, and coagulation factors (if indicated). Treatment should not be delayed awaiting CT or

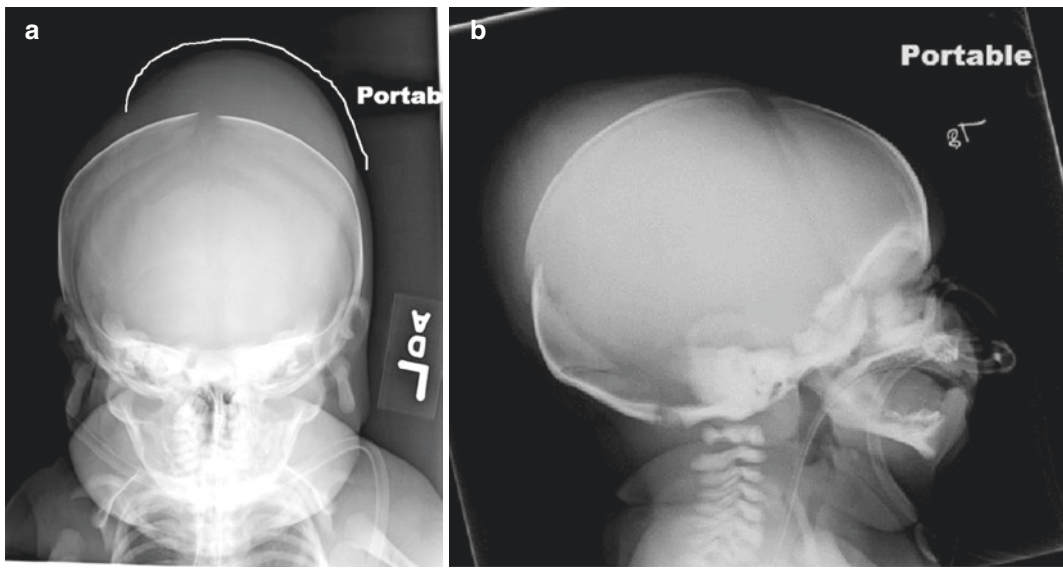

Fig. 2.9 (**a**) AP radiograph of the skull showing diffuse soft tissue swelling of the scalp that crosses sutures and that is especially prominent over the left frontoparietal region. The findings were felt to be most compatible with a subgaleal hematoma. (**b**) Lateral radiographs of the skull demonstrate diffuse soft tissue swelling of the scalp that crosses sutures and that is especially prominent over the left frontoparietal region. The findings were felt to be most compatible with a subgaleal hematoma

MRI confirmation. Stabilization of the infant is the immediate priority. Unrecognized hypovolemic shock can greatly diminish the chances of survival for infant with SGH [6]. This infant was effectively treated with two normal saline boluses and a blood transfusion for hypovolemic shock from blood loss. When discharged careful follow-up is necessary.

Answers: 2 and 3

What are some of the long-term complications you may observe during follow-up in this infant?

1. Alopecia
2. Calcification

Rarely in infants with caput succedaneum, a non-scarring distinct pattern of annular hair loss, commonly referred to as halo scalp ring, has been reported [8]. This is thought to be due to prolonged pressure on the scalp by the cervical os during or before the delivery.

Cephalohematoma may become calcified and cause bony deformities of the skull [9]. Persistent calcification that is not resolved by 6 months may need surgical excision.

Alopecia and calcification need to be evaluated during follow-up and may not be apparent during discharge from nursery.

Answers: 1 and 2

The above case presentations and questions provide information on various aspects of neonatal extracranial hemorrhage. Their distinguishing features are summarized in Table 2.1 below. (Table 2.1).

Case Presentation

A nurse from normal nursery calls you because she noted a large depression over the right parietal bone in a 4-h-old 3500 g infant born to a primiparous woman following a vertex vaginal delivery at 37 weeks of estimated GA. The obstetric history is significant for prolonged labor. The delivery was assisted with midforceps and vacuum extraction. Physical examination is remarkable for a 4 × 5 cm depression of the skull in the right temporoparietal region and a full anterior fontanelle. Neurological examination was normal.

At this time, you:

1. Order skull radiograph
2. Order ultrasound of the head
3. Order CT scan

Physical exam in this infant is consistent with depressed skull fracture. Depressed fractures are visible, palpable indentations in the smooth contour of the skull, like dents in a Ping-Pong ball. Depressed skull fractures are due to the inward buckling of the skull bones and are often associated with forceps-assisted deliveries. While the diagnosis is made by a plain radiograph of the head (Fig. 2.10), imaging with CT (Fig. 2.11) is required to determine the presence or absence of bone fragments in the cerebrum or associated intracranial injury [10, 11].

Linear skull fractures usually affect the parietal bones. They often are associated with cephalohematomas (Fig. 2.12). Linear skull fractures

Table 2.1 Distinguishing features of neonatal extracranial hemorrhage

Features	Caput succedaneum	Cephalohematoma	SGH
Location	At point of presentation, crosses suture lines	Over parietal bones Limited by sutures	Beneath epicranial aponeurosis Extends from orbit to nape of the neck
Findings	Vaguely demarcated Pitting edema	Distinct margins Initially firm Fluctuant after 48 h	Firm to fluctuant Crepitus, fluid waves
Timing	Max size at birth Resolves 48–72 h	Increases after birth for 12–24 h Resolves 2–3 weeks	Increases after birth, resolves 2–3 weeks
Blood loss	Minimal	Rarely severe	May be massive
Complications	Alopecia	Anemia, jaundice, infection, calcification	Hypovolemic shock, anemia, jaundice, infection

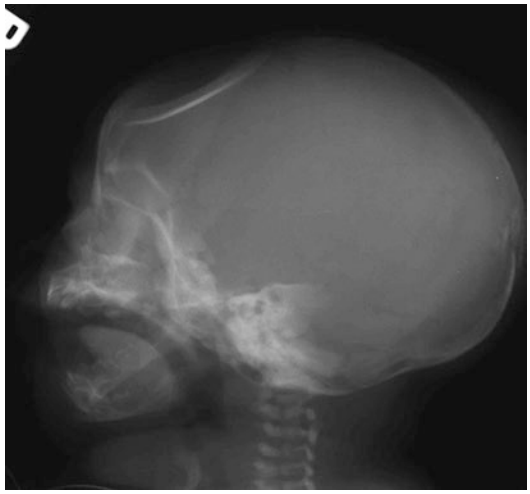

Fig. 2.10 Lateral skull radiograph showing the right parietal depressed skull fracture

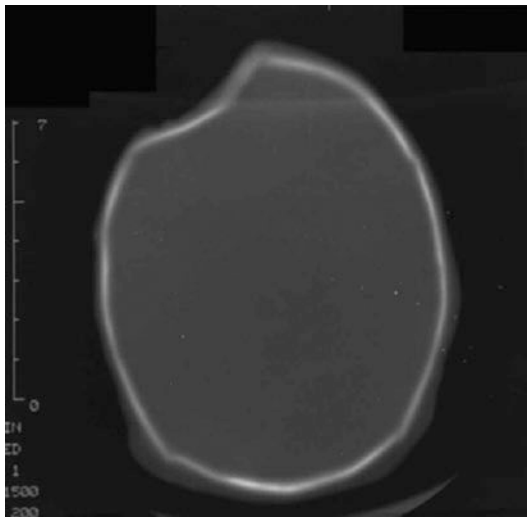

Fig. 2.11 CT of the infant with depressed skull fracture

are clinically not apparent and require no treatment [6, 10, 11].

Answers: 1 and 3

How would you manage the infant in this case?

1. Neurosurgical consultation
2. Observation only

Management depends on presence or absence of neurologic sequelae. Neurologic findings

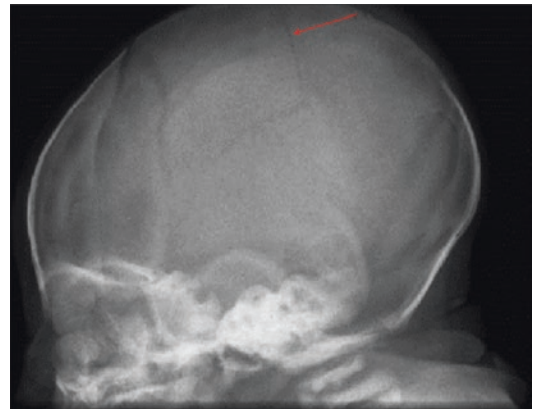

Fig. 2.12 Skull X-ray lateral view of infant with linear skull fracture (arrows)

due to skull fractures in infants born via normal spontaneous vaginal delivery are rare. These cases resolve spontaneously as in this case. Neurosurgical consultation was obtained, but no intervention was done as baby had no neurologic symptoms. Fracture reduction is done when the depth of buckling is more than 1 cm or baby has neurologic signs [6, 10, 11]. Parental reassurance and repeat skull radiographs for follow-up were done in this case.

Rarely, in an infant with linear skull fracture, a leptomeningeal cyst from a dural tear may occur [12]. Abnormally rapid head growth should raise suspicion for a leptomeningeal cyst in baby who had linear skull fracture. Infants with leptomeningeal cyst need to be referred to neurosurgery.

In this case neurosurgical consultation was obtained, and infant was observed closely for neurologic sequelae.

Answer: 1

Case Presentation

You are asked to evaluate a term male newborn with asymmetric facial movements when crying. The neonate was born to a 35-year-old, G1p0 mother with a birth weight of 3700 g and an Apgar score of 8/9 at 1 and 5 min. respectively. Baby was delivered vaginally after a prolonged

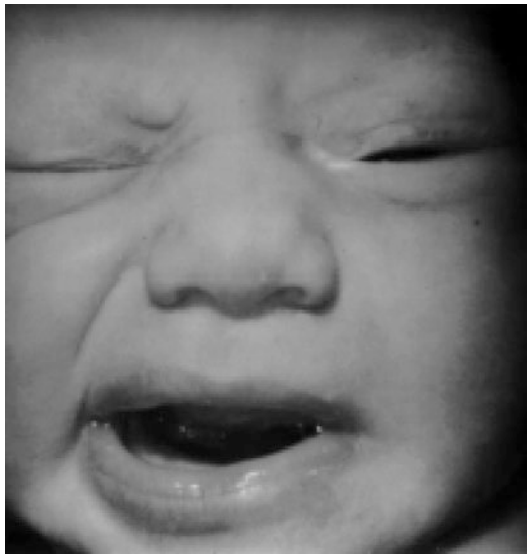

Fig. 2.13 Infant with left facial nerve palsy. (Adapted from Physical assessment of the newborn: a comprehensive approach to the art of physical examination, fifth edition, chapter 5. Springer Publishing Company)

labor. At birth, the infant was noted to have left palpebral fissure wider than the right and absence of nasolabial fold on the left side. You notice that when the infant cries there is deviation of angle of mouth to right and the left eye did not close completely. There is no forehead wrinkling and complete absence of facial movements on the left side (Fig. 2.13).

You examine the baby and conclude he has:

1. Right central facial palsy
2. Left central facial palsy
3. Right peripheral facial palsy
4. Left peripheral facial palsy

Peripheral facial nerve damage produces total facial paralysis on the involved side of the face. A central lesion will affect only the lower half of the face due to the fact that the corticobulbar fibers to the forehead and upper half of the face are distributed bilaterally. This patient has no movement of the forehead or periorbital eye muscles on the left. In addition there is flattening of the left palpebral fissure and movement of the mouth to the right (which may seem to be a drooping of the right side of the mouth). The entire left side of the face is involved, and so this baby has a left peripheral facial nerve palsy.

Answer: 4

At this time, you would:

1. Call a neurologist
2. Transfer the baby to neonatal intensive care unit

There is no emergency to call a neurologist. Congenital facial nerve paralysis should be differentiated from traumatic facial nerve paralysis as early as possible as this determines the course of the disease process and treatment plan, and therefore transfer of the baby to the NICU would be appropriate.

Answer: 2

How would you manage this patient?

1. Attention to eye care
2. Attention to feeding
3. Consult an ophthalmologist

Immediate medical treatment of facial paralysis requires attention to eye care. Infant should be managed with artificial tears to prevent dryness of the affected eye and eye padding to protect corneas [2].

This infant roomed in with the mother. Artificial tears and eye padding was provided to protect the eye from corneal abrasions. Baby was observed for feeding difficulties, as the ability to suck may be impaired due to inability to contract the lower facial muscles on the affected side. Infant was discharged on day 3 of life with significant improvement in facial nerve weakness. Parents were informed about this benign self-limiting condition and need for adequate eye care. Infant gradually improved in the follow-up clinic.

Answers: 1 and 2

Case Presentation

You are evaluating a 4200 g newborn infant with decreased movement of the right arm. The mother is 35 years old, G2P1, and her perinatal history includes difficult prolonged labor, shoulder dystocia, vertex presentation, vacuum extraction, and traction on the neck during delivery. Physical examination of the right arm revealed an adducted shoulder, internally rotated upper arm, extended elbow, pronated forearm, and flexed wrist. The infant had mild respiratory distress.

At this time, most likely cause for the decreased movement of the right arm is:

1. Erb's palsy
2. Klumpke's palsy
3. Pseudoparalysis from clavicle fractures
4. Pseudoparalysis from humerus fractures
5. Phrenic nerve palsy

When an infant is born with brachial plexus palsy (BPP), the condition generally is apparent from birth. Physical finding of the arm hanging limply from the shoulder is typical for BPP. In a common scenario as described in this case, the baby is large for gestational age and is the product of a difficult delivery to a multiparous woman, requiring the use of vacuum or forceps [13].

Clinical presentation of BPP can be classified according to the site of the nerve injury. Injury to the upper trunk involves nerve roots (C5–C6), middle trunk (C7), and lower trunk (C8–T1). Total BPP affects nerves at all levels (C5–T1).

Erb-Duchenne Palsy refers to an injury of the upper brachial plexus nerve roots (C5, C6, and C7) leading to loss of motion around the shoulder and ability to flex the elbow. Isolated lesion of upper trunk (C5–C6) is known as Erb's palsy. Infants with Erb's palsy classically have a "waiter's tip" limb posture characterized by adduction and internal rotation of the affected arm with extended elbow, pronated forearm and flexed wrist (Fig. 2.14).

Klumpke's palsy refers to an injury of the lower brachial plexus nerve roots (C8–T1). The infant with Klumpke's palsy holds the arm supinated, with the elbow bent and the wrist extended, often described as "beggar's hand" [14].

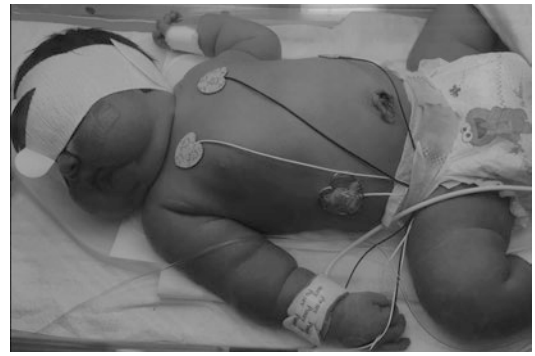

Fig. 2.14 Infant with right-sided upper plexus injury (Erb's palsy)

The infant with complete BPP (C5–T1) typically lies with the arm held limply at his/her side. In infants with Klumpke's palsy and total plexus lesions (C5–T1), careful examination of the child's eye should be performed for presence of Horner's syndrome (i.e., miosis, ptosis, anhidrosis), which suggests injury to the stellate ganglion.

The Moro, grasp, asymmetric tonic neck, and biceps reflexes should be evaluated, and any sensory loss in corresponding dermatomes should be noted. Deep tendon reflexes (DTRs) in the affected arm are absent, and the Moro response is asymmetrical, with no active abduction of the ipsilateral arm.

Injury to phrenic nerve with ipsilateral diaphragmatic paralysis should be considered in a newborn with BPP that has tachypnea and requires oxygen. Diagnosis of phrenic nerve injury is made by a chest radiograph (Fig. 2.15), which shows elevated diaphragm on the affected side. Real-time ultrasonography at the bedside can reveal abnormal motion of the affected hemidiaphragm and confirm the diagnosis [15]. Infant with phrenic nerve injury may require continuous positive airway pressure (CPAP) or mechanical ventilation and may even need surgical plication of diaphragm.

The most common symptom associated with a clavicle fracture in a newborn is fussiness or crying with movement of the affected arm due to pain in the clavicle. Decreased movements of the

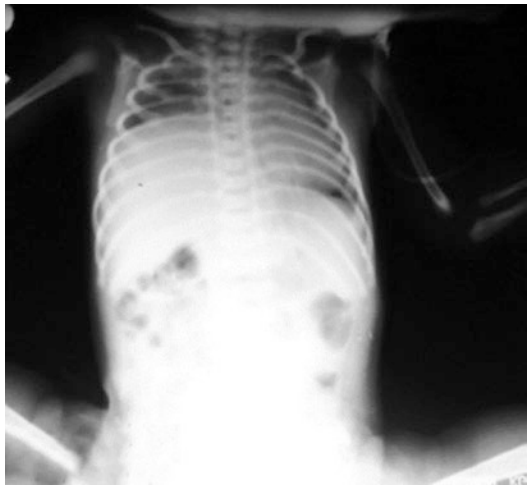

Fig. 2.15 CXR of infant with elevated right hemidiaphragm from phrenic nerve injury. (Reproduced with unrestricted permit from J Pediatr Neurosci. 2012 Sep–Dec; 7(3): 225–227)

affected upper limb due to pain (pseudoparalysis) and asymmetric Moro reflex is the most common indicator of the injury. This can be differentiated from BPP, because tendon reflexes remain intact following isolated clavicular fractures.

Infants with humeral fractures (Fig. 2.16) also present with decreased movement of the affected arm, decreased Moro reflex, localized swelling and crepitation, and an increased pain response with palpation and movement of the arm. Any infant with a humeral fracture should be evaluated for brachial plexus injury, as this is a common associated finding.

The infant in the vignette was diagnosed to have Erb's palsy. On physical examination, it was noted that baby was fussy and cried with movement of the affected limb. There was crepitus and swelling over the right clavicle as well.

Answers: 1 and 3

At this time, you look for:

1. Fracture or injury to the humerus
2. Fracture of the clavicle

The clavicle is the most commonly fractured bone in the neonate. Although, fractured clavicles

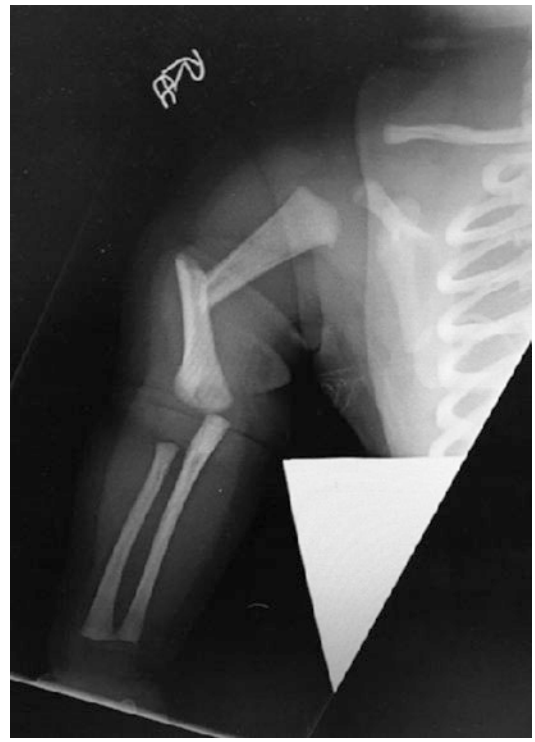

Fig. 2.16 Radiograph showing mid-shaft diaphyseal oblique fracture of right humerus

are often associated with difficult vaginal delivery, they also occur in infants who are products of a normal spontaneous vaginal or cesarean delivery.

Clavicle fractures should be considered in any infant with BPP or infant presenting with decreased upper extremity movement. Infants with a clavicle fracture may be asymptomatic or be crying with passive movements of the affected extremity. Presence of crepitation, swelling, palpable bony irregularity, and/or bruising over the affected clavicle may be present in some infants.

Displaced fracture of the clavicle (Fig. 2.17) is accompanied by physical findings mentioned above in the immediate post-delivery period, whereas nondisplaced clavicle fracture (Fig. 2.18) may remain asymptomatic and diagnosis may be missed or delayed until there is a formation of a visible or palpable callous.

Diagnosis is confirmed with radiography (Fig. 2.17), where a clear fracture is usually readily identified.

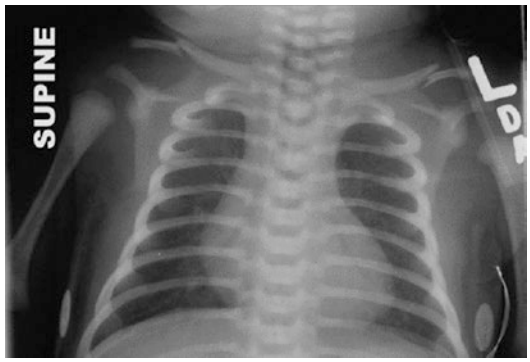

Fig. 2.17 CXR of infant with bilateral displaced clavicle fracture

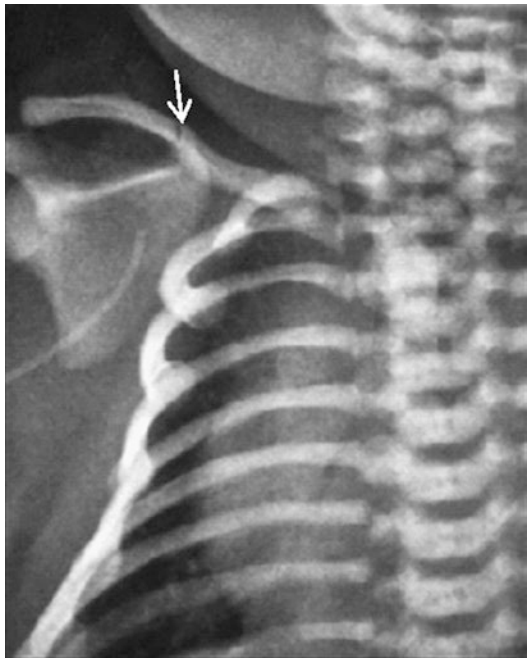

Fig. 2.18 CXR of infant with nondisplaced clavicle fracture (arrow)

Clavicle fractures in infants heal spontaneously with no long-term sequelae. In most cases, especially the asymptomatic ones, parental reassurance and gentle handling are all that are required [6, 10]. Analgesics may be given to decrease the pain. In infants with pseudoparalysis, the affected side can be placed in a long-sleeved garment and pinned to the chest with the elbow at 90 ° of flexion for 2 weeks. In infant with lack of tenderness and callus formation detected on physical examination, a repeat radiograph at 2 weeks of age is not necessary.

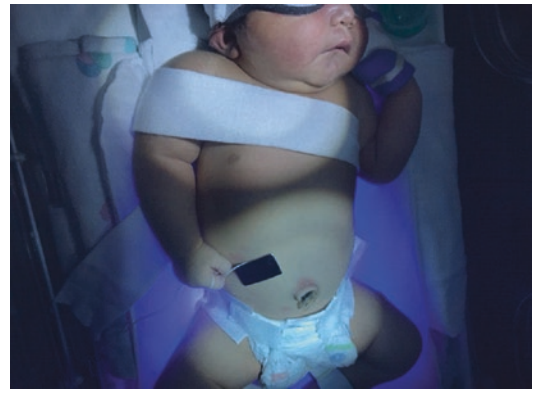

Fig. 2.19 Infant with a sling for right humeral fracture

Humeral fracture is also associated with pain response with palpation and movement of the arm, localized swelling, crepitation, and decreased Moro reflex. The diagnosis is generally made by a plain radiograph of the arm (Fig. 2.16). Because proximal epiphysis in the newborn is entirely cartilaginous, it is radiolucent; thus ultrasound evaluation may be more useful in proximal epiphyseal fracture of the humerus. Immobilization of the affected arm (Fig. 2.19) with the elbow in 90 ° flexion to prevent rotational deformities is the best treatment [6, 10]. Callus formation usually occurs in 7–10 days. A repeat radiograph can be performed at 3–4 weeks post-injury to confirm healing of humeral fracture. Reassurance to the parents that angulation will remodel as the infant grows should be provided.

Infants with decreased upper extremity movement should be evaluated for BPP, clavicle, and humeral fracture, as these lesions sometimes accompany each other.

Answer: 2

How would you manage the brachial plexus injury in this patient?

1. Discharge with follow-up in 1 week
2. MRI with follow-up with neurology
3. Follow-up with referral to physical therapy

Initial management of infant with BPP includes careful evaluation as the diagnosis of BPP is made from clinical findings. Observe the newborn for respiratory distress due to possible diaphragmatic de-innervation from phrenic nerve injury. Evaluate for signs of Horner's syndrome if the diagnosis is Klumpke's palsy. The neurologic examination should include observation of spontaneous movements, passive and active range of motion, stimulated motor and sensory responses, and assessment of Moro, grasp, and asymmetric tonic neck reflexes.

Since spontaneous recovery occurs in almost 90% of patients with Erb's palsy, outpatient follow-up within a week is appropriate. Physical therapy with passive range of motion exercises should be considered if no improvement is noted in 7–10 days.

Neuroimaging with high-resolution MRI can be considered in infants with no improvement. Follow-up with a neurologist is recommended to establish baseline loss of function and to monitor the improvement.

Full, spontaneous recovery is expected for infants showing some improvement within the first 2 weeks; partial recovery can be expected if initial improvement takes 4–6 weeks, and if no improvement is appreciated by 3 months of age, surgery may be attempted to improve the prognosis [16]. Surgical intervention may include nerve reconstruction or the transfer of other nerves to the affected area.

Answer: 1

Case Presentation

You are examining a 48-hour-old baby girl whose parents are anxious to go home. The baby is 8 lb., 6 oz., 38 weeks gestational age. The mother is 30 years old, G2P1. Her obstetric history and pregnancy are uneventful. Due to maternal preference for "natural" child birth, she delivered vaginally despite a breech presentation. On physical examination, you note that her feet are still bruised and she has new findings of erythematous, indurated plaques on her right posterior shoulder (Fig. 2.20) which were not there on her first physical exam soon after birth.

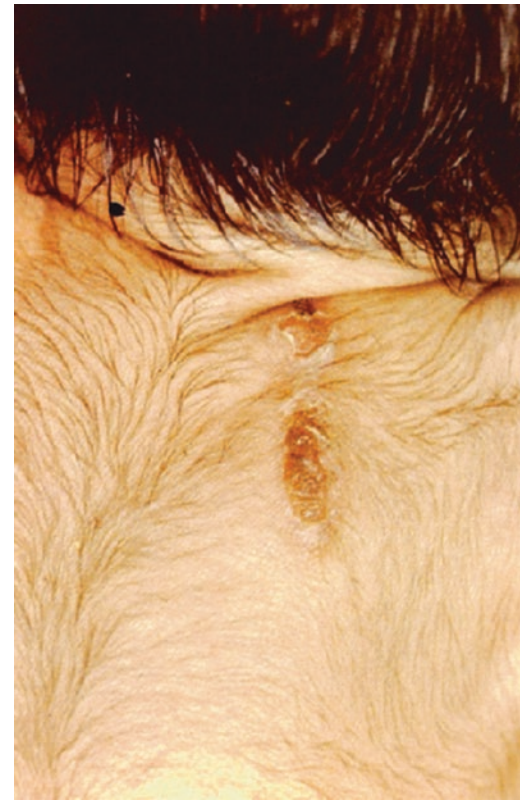

Fig. 2.20 Infant with fat necrosis

At this time you suspect:

1. Congenital melanocytic nevi
2. Superficial streptococcal infection
3. Fat necrosis
4. Café au lait spots

This infant has subcutaneous fat necrosis (SFN) which is a benign condition occurring in the neonatal period, characterized by inflammation and necrosis of subcutaneous fat tissue and typically presenting with subcutaneous purple-bluish hard nodules. Nodules may evolve with subcutaneous calcifications. SFN usually occurs in full-term infants over the trunk, arms, buttocks, thighs, and cheeks as firm, mobile, erythematous nodules and plaques following tissue trauma. The lesions are frequently tender with a taut and shiny skin.

Answer: 3

At this time, you:

1. Discharge the patient with follow-up within 24 h
2. Hold discharge and order selective blood tests

Full sepsis workup is not indicated in absence of risk factors for sepsis. At this time, holding the discharge would be most appropriate pending blood test result.

Answer: 2

How would you manage this case?

1. Draw serum calcium levels.
2. Skin biopsy of the lesion.
3. Start antibiotics pending blood test results.
4. You reassure the parents that it is just a bruise.

SFN is not just a bruise and these babies need blood test and close follow-up. In some infants, liquefied fat may present as fluctuant bullae or extensive calcification within the lesions. SFN may be complicated by hypercalcemia in some infants [17]. Uncomplicated SFN does not require treatment, but occurrence of hypercalcemia does require treatment. All neonates with subcutaneous fat necrosis should have their serum calcium level monitored regularly during the episode and during follow-up. Hypercalcemia usually develops when the subcutaneous fat necrosis begins to resolve, but the onset of hypercalcemia can be delayed several months after the development of skin manifestations which emphasizes the importance of prolonged follow-up.

Babies with hypercalcemia may present with irritability, constipation, poor weight gain, calcification of the kidneys, and, very rarely, heart rhythm disturbance, so treatment of SFN focuses on management of hypercalcemia.

Hypercalcemia may be treated by increased fluid intake, low-calcium milk feeds, furosemide, corticosteroids, and bisphosphonates [18].

In most cases, SFN is a self-limited process; the skin nodules and plaques spontaneously regress within weeks to a few months without cutaneous sequelae although cutaneous scarring and atrophy may occur at sites of involuted lesion. Serum calcium levels were done prior to discharge on this infant.

Answer: 1

How would you manage the bruising on the feet of this baby?

1. Observation only.
2. Do a CBC.
3. Do a coagulation panel.
4. Do bilirubin levels.

Bruising and petechiae may be noted on the presenting parts of the newborn. In this case there is bruising of the feet due to breech presentation. Bruising of the scrotum in frank breech presentation warrants testicular evaluation; similarly bruised eyelids warrant a full eye evaluation. Bruising is usually self-limiting and resolves spontaneously within 1 week, but significant bruising can be a major risk factor for severe hyperbilirubinemia. Observation with reevaluation within 2–3 days of hospital discharge to assess the infant for progressive jaundice is most appropriate in this case.

Answer: 1

Clinical Pearls

1. All newborns born after abnormal fetal presentations require a thorough pediatric examination.
2. Cesarean section does not eliminate the possibility of birth trauma, especially when prior attempts have been made at delivery with vacuum extraction or forceps.
3. The extracranial injuries of caput succedaneum and cephalohematoma usually

resolve spontaneously without any intervention.
4. Subgaleal hemorrhage can result in massive blood loss and, if not detected and managed appropriately, may lead to shock and death.
5. Clinical signs of an extremity fracture include crepitus, pain, swelling, and decreased limb movement.
6. Most clavicular and skull fractures resolve spontaneously and can be managed conservatively with observation alone.
7. CT of the head is indicated for a depressed skull fracture.
8. Upper arm palsy (Erb-Duchenne) caused by damage to the fifth and sixth cervical nerve roots is the most common peripheral nerve injury.
9. It is important to distinguish traumatic facial nerve palsy from congenital hypoplasia of the depressor anguli oris muscle.

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