Labial Adhesions

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Commentary

The Importance of Amy Lynn

Recently I became a grandfather for the first time, certainly not an unusual event. However, the gestation and birth of this little girl made me more keenly aware of my responsibilities and role as a physician and specifically as a pediatrician. Preoccupation with the practice of medicine and life in general may have clouded the memory of my own children as infants.

In clinical residency, we often would distance ourselves emotionally from seriously ill patients by various defense mechanisms; it was difficult to experience children dying and their parents’ grief. Too often, we would use terms or expressions that would be abhorrent to the lay public.

As a general pediatrician, I always have felt myself to be caring and conscientious. However, it is easy to become too involved with blood gas values, cultures, and the other scientific aspects of medicine. Additionally, in this time of high insurance rates and astronomical office expenses, it is easy to become more of a businessman and less of a physician. There are payrolls to meet and rents to pay. However, our business is dealing with children, not selling computers or used cars. We cannot forget the importance of the infant, the love and concern of the family, and the fact that most families have financial and emotional problems too.

There is danger of compromised judgment and decisions if we become too involved emotionally in the care of our patients. However, my new experience has made me more empathetic with the parents and family and more cognizant of the love involved. I can better understand their anxieties. I hope that it will make me a better pediatrician.

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Editorial Board

Abstract

Labial Adhesions


Labial adhesions are a common gynecologic finding among infants and prepubertal children. They typically are found in girls between the ages of 4 months and 6 years and are thought to be secondary to local inflammation and resulting agglutination.

Labial adhesions have a characteristic appearance and, therefore, are not difficult to diagnose. With complete adherence, the vulva appears flat with a central line of fusion. The urethra is not visualized, and one might see urine dribbling at the anterior end, just posterior to the clitoris. With partial adhesion, this central line of fusion would be visualized at the posterior fourchette, extending to varying lengths anteriorly. The genitalia otherwise appear normal.

Development of the adhesions typically depends on the presence of inflammation (i.e., vulvovaginitis) in a low-estrogen environment. In such a low-estrogen setting, injured or inflamed epithelium is more likely to agglutinate. Hence, labial adhesions rarely are seen in newborns (who were exposed to high levels of maternal estrogen) or in postpubertal-premenopausal women.

Nonspecific vulvovaginitis is the most common precipitant to labial adhesions. Typically, this nonspecific vulvovaginitis is asymptomatic, and its presence may not be detected on physical examination at the time of labial adhesion diagnosis. Poor perineal hygiene is the most common cause of this inflammatory process. Specific etiologic organisms are less common. These include Enterobius vermicularis (pinworm), in which one may elicit a history of nocturnal pruritus; candidiasis, in which there often is a history of recent antibiotic use or diabetes mellitus; and specific infectious organisms. The most common bacterial cause of nongonorrheal vulvovaginitis in prepubertal females is group A streptococci, which may be associated with a recent pharyngitis or a skin infection. Other causes of infectious vulvovaginitis are less likely to present with labial adhesions because they are less likely to be asymptomatic or they are less common among this age group.

These include Neisseria gonorrhoeae, Trichomonas sp, Chlamydia sp., Gardnerella vaginalis, Shigella sp (associated with a gastroenteritis), and Hemophilus vaginalis.

While the labial adhesion itself must be addressed, unless the local inflammation that precipitated the adhesion is treated or prevented, labial adhesions are likely to recur. If there is evidence of a vulvovaginitis, a detailed history for concurrent infections, recent antibiotic use, and if indicated, a history of sexual abuse must be obtained. In addition, appropriate cultures should be performed. Positive cultures should be treated as indicated; if organisms associated with sexually transmitted diseases are identified, sexual abuse must be ruled out.

If the adhesion is complete or urinary flow is interrupted, a sterile urine culture should be obtained; if it
is positive, it should be treated appropriately.

Good perineal hygiene is essential. Caretakers must be instructed in thorough wiping after each bowel movement and always wiping anteriorly to posteriorly. If there is any evidence of inflammation on physical examination, two 20 min sitz baths per day, using tap water or Burow solution, should be prescribed. Diapers should be changed regularly and shortly after each bowel movement. Underwear should be made of cotton, loose fitting, and changed daily or earlier if soiled.

The management of labial adhesions themselves has changed considerably over the past 20 years. Before the 1970s, surgery was recommended for all patients who had labial adhesions. Today, treatment is the topical application of a 1% conjugated estrogen cream applied over the length of the adhesion at bedtime for 3 weeks. The cream is best applied with a cotton applicator that is gently but firmly pressed down the length of the adhesion. This method has an 80% to 90% cure rate, but may result in mild vulvar pigmentation, vulvar erythema, and occasionally, mild breast enlargement or tenderness (almost all of which is reversible). After resolution of the adhesion, a bland ointment such as petroleum jelly should be applied nightly for 1 to 2 months.

Of note, spontaneous resolution of asymptomatic labial adhesions without surgical or medical therapy has been reported, with a 50% resolution within 6 months and a 100% resolution within 18 months. In addition, there were no recurrences.

Therefore, if a patient is asymptomatic and her parents can be reassured that there is nothing to be alarmed about, no specific treatment other than good perineal hygiene is indicated. If a parent remains duly concerned, the patient should be treated with estrogen cream.

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Congenital Nasolacrimal Duct Obstruction


Abnormalities of the lacrimal system in infants and children generally are due to congenital obstruction of the nasolacrimal duct. Other rare causes, such as impatent or absent puncta, fistula of the lacrimal sac, or mucocele of the lacrimal sac, do exist.

The lacrimal system is comprised of the lacrimal gland and ducts where tears are produced and is located along the superotemporal aspect of the orbit. The tears flow over the surface of the eye, are caught by the lid margins, and then enter the upper or lower punctum near the medial canthus. Each punctum drains to the lacrimal sac, which is just inferior to the medial palpebral ligament, normally empty, and cannot be felt. The sac narrows to the nasolacrimal canal, which passes through the maxilla into the nose. Most congenital obstructions of this system occur at the point where the duct exits into the nose. If pressure over the lacrimal sac does not cause reflux of mucoid material into the eye, perhaps the obstruction is due to one of the more uncommon causes listed previously and should be referred to the ophthalmologist for further evaluation.

Approximately 6% of newborns present with congenital obstruction. The lacrimal drainage system is formed from a solid cord of ectoderm that atrophies at the center to form a patent duct at about 3 months' gestation. Obstruction may result from remaining membrane that failed to atrophy or from adhesions to the nasal mucosa. Occasionally this membrane can be felt to pop when the duct is being probed.

Typical signs on presentation can occur anywhere from days to weeks after birth. One may see a large tear meniscus on the lower lid, overflowing of tears onto the cheek without stimulus, or accumulation of dried mucoid material in the medial canthus. More serious signs consist of reflux of mucopurulent contents of the lacrimal sac or conjunctival injection and erythema of the skin near the puncta and lid margins. An infant may have all of these signs over a period of days to weeks, and they can be aggravated by an upper respiratory infection.

A blocked nasolacrimal duct essentially is a cul-de-sac and, therefore, prone to recurrent infections. The lacrimal sac becomes irritated, leading to constant reflux of mucus into the eye, which usually is not uncomfortable for the infant but bothers the parents. Infection can become severe, however, producing swelling and obstruction of the canaliculus, and possibly, cellulitis of the adjacent skin on the face.

Most obstructions clear either spontaneously or from conservative management consisting of warm soaks, antibiotics, and Crigler massage. The parents are instructed to apply pressure with the tip of the finger over the lacrimal sac and then stroke downward toward the duct to increase hydrostatic pressure and possibly rupture the membrane that occludes the duct. The parents should follow this regimen two to six times a day. In addition, some ophthalmologists recommend that an antibiotic ointment such as erythromycin be applied once a day to prevent infection and more often if the eye discharge is mucopurulent. The ointment may be applied directly to the eyeball because the tears will distribute it in the lacrimal system.

More than 90% of patients' obstructions will either clear spontaneously or resolve with conservative management by the age of 9 to 13 months. If these measures fail, ophthalmologists will probe the duct. Some children require more than one probing for the obstruction to be relieved. More complicated procedures are performed if these probings prove unsuccessful, such as placement of a silicone tube into the duct or a dacryocystorhinostomy.

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Myocarditis


Myocarditis is an inflammation of the myocardium and generally is grouped as one of the cardiomyopathies. It is not unusual for the terms cardiomyopathy and myocarditis to be used interchangeably, but the term myocarditis should be reserved for the inflammatory disorders.

As in other lesions of an infectious nature, myocarditis frequently begins with a typical viral prodrome: low-grade fever, a history of an upper respiratory tract infection, or gastrointestinal symptoms. Abdominal discomfort is not an infrequent presenting complaint. Myocarditis may be part of a generalized inflammatory process such as acute rheumatic fever, rheumatoid arthritis, and other autoimmune disorders. It also may be secondary to the toxic effects of certain pharmacologic agents (eg, sulfa drugs). Cardiovascular symptoms depend entirely on the severity of the disorder. In mild forms, no symptoms other than mild tachypnea or abdominal pain may be present; the diagnosis is made only when an electrocardiogram, chest radiograph, or echocardiogram is obtained. There may be no signs of cardiac decompensation. Many forms of idiopathic dilated cardiomyopathy may have had their onset with a subclinical form of viral myocarditis years earlier. Cardiovascular signs and symptoms, when present, are those of congestive heart failure—mainly, tachypnea, tachycardia, possibly rales, and hepatomegaly. The liver may be tender. The heart sounds may appear muffled and a gallop rhythm heard. If there is significant cardiovascular compromise with decreased myocardial contractility, signs of low cardiac output may be manifest: cold sweats, weak and thready pulses, and marked hypotension.

A chest radiograph usually shows cardiomegaly with increased pulmonary vascular markings. The electrocardiogram shows a sinus tachycardia with generalized low voltage in all leads. ST-T wave abnormalities, particularly in leads 1, AVL, and the left-sided chest leads (V5 and V6), may be present. These ST-T wave abnormalities may take many forms—flattening, depression and inversion, or even ST-T wave elevation. Rarely, there also may be conduction disturbances (diphtheritic myocarditis, in particular, is associated with varying degrees of heart block). Ventricular or atrial ectopy also may occur. These arrhythmias and conduction disturbances, however, are relatively infrequent, even in severe cases.

An echocardiogram will differentiate myocarditis from structural abnormalities in patients who are presenting with cardiovascular collapse and in whom heart sounds may be too muffled to demonstrate the expected murmurs. It also will rule out a large pericardial effusion, which can manifest a similar clinical presentation. Typically, the shortening fraction and/or ejection fraction on echocardiogram will be reduced and left ventricular (LV) dilatation will be evident. LV wall stress, another parameter of contractility, also will be affected.

Endomyocardial biopsy recently has been used to confirm the diagnosis and to assist in determining the etiology of myocarditis. The biopsy is carried out during right ventricular cardiac catheterization and consists of a biopsy of the right ventricle only. Four or five specimens are obtained. For the specimen to be clearly diagnostic of active myocarditis, significant myocyte degeneration or necrosis with associated inflammation (usually lymphocytic) at multiple sites should exist. Absence of demonstration of activity on biopsy, however, does not exclude an ongoing myocarditis. Rarely are cultures of myocardium positive for either bacterial or viral agents.

When myocarditis is suspected, standard blood cultures should be obtained, as well as viral cultures from blood, the upper respiratory tract, and stool. Viral titers should be evaluated in both acute and convalescent serum. Generally a fourfold rise in viral titers is considered significant, though not necessarily diagnostic. The white blood cell count may be elevated as well as the erythrocyte sedimentation rate. Cardiac enzymes may be abnormal, as may liver enzymes due to chronic passive congestion.

The etiology of myocarditis is variable. Any infectious agent can be responsible for active myocarditis; most commonly, however, it is viral. Although many viruses have been implicated, the enteroviruses, particularly Coxsackie, are the most frequent offenders. Others include echoviruses and adenovirus. In the newborn, herpes simplex should be considered. Human immunodeficiency virus (HIV) also has been implicated in cardiomyopathies, but rarely does this infection present as an acute myocarditis.

Management generally is supportive, and treatment for congestive heart failure or low cardiac output is instituted only when signs and symptoms are present.

Digitalis should be used judiciously, as the inflamed myocardium has been found to be especially sensitive to this agent. In those who have low cardiac output, intravenous pressors such as dobutamine and dopamine may be required. Arrhythmias are treated only if significant, but rarely present a problem except in the most severe cases.

When the diagnosis of acute myocarditis is confirmed by biopsy, the question of immunosuppressive therapy is raised. Suppressive therapy in acute myocarditis remains controversial. Prednisone and azathioprine or cyclosporine have been employed successfully. It is well known, however, that supportive therapy alone can result in spontaneous improvement. Specific antiviral therapy has not been helpful. Recent reports have shown a major
improvement in myocardial function in a group of patients treated with IV gamma globulin at a dose of 2 g/kg, given in conjunction with conventional supportive anti congestive measures.

The outcome in newborns who have fulminant myocarditis is guarded. Older patients have a much better prognosis, with up to 80% showing full clinical recovery. Others may develop chronic cardiomyopathy subsequently.

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Comment: Although mentioned in the body of the abstract, HIV cardiomyopathy is increasingly a consideration in a child who presents with congestive failure outside of the age group in which congenital heart disease should be considered. Though rarely presenting as acute myocarditis, the cardiomyopathy of HIV disease is debilitating and often has many of the same presenting signs and symptoms as acute myocarditis. With the increasing prevalence of this particular virus-related cardiomyopathy, consideration of it in children other than infants must continually be kept in mind in populations at higher risk.

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Abstracts Editor

ABSTRACT

Foreign Body Aspiration

Laryngotracheal Foreign Bodies in Children. Esclamado RM, Richardson MA. AJDC. 1987;141:259–262

Asphyxiation from foreign body aspiration is a common cause of accidental death among children younger than 5 years. Despite efforts by the Consumer Product Safety Commission to restrict the production of small items in toys marketed for young children, there still is a significant aspiration risk from exposure to a sibling’s toy, small food (especially peanuts), and other small objects.

In most cases, it is evident that the child has aspirated a foreign body because either the event was witnessed or the child began choking. In a study of 20 cases of laryngotracheal foreign bodies, Esclamado et al found that 90% of children had a history of choking. The most common presenting symptoms were stridor, wheezing, sternal retractions, and cough. Similarly, in a study of 83 children who had tracheobronchial foreign bodies, Puhakka et al found that most common presenting symptoms were cough, stridor, and dyspnea. These symptoms also may be seen in other common illnesses that cause upper airway obstruction, such as asthma, croup, and epiglottitis. Infectious etiologies, however, usually are associated with fever, drooling, or hoarseness, and children who have asthma usually have a history of wheezing and respond to bronchodilator therapy. Still, establishing the correct diagnosis can be difficult because there often is no clear history of foreign body aspiration. Puhakka notes that in several cases the parents believed that the foreign body had been expelled spontaneously by coughing and that after an asymptomatic period of several weeks, the wheezing related to the aspirated foreign body might be mistaken for an asthmatic episode.

Most aspirated foreign bodies are radiolucent and, thus, are not clearly evident on a radiograph. One must look for associated pulmonary changes caused by the foreign body. The most common findings on a chest radiograph are obstructive emphysema, atelectasis, or pneumonia. Expiratory radiographs may aid in the diagnosis of obstructive emphysema by showing a persistently hyperinflated lung segment. However, if the foreign body is in the trachea, there usually is no evidence of obstruction, and the chest radiograph will be normal. In this case, a posteroanterior and lateral neck study may show subglottic density or swelling.

Aspirated foreign bodies should be removed as soon as the diagnosis is suspected or confirmed. Depending on the location of the foreign body, removal is by either direct laryngoscopy or bronchoscopy by an experienced physician. Most complications (other than complete airway obstruction) are the result of delayed diagnosis, the foreign body itself, or attempts at removal. In children who have bronchial foreign bodies, pneumonia is the most common complication. In the study by Esclamado, 45% of children who had laryngotracheal foreign bodies had complications; 67% occurred in children whose diagnosis was delayed. The complications in that group included subglottic edema, respiratory failure, and pneumonia. The American Heart Association recently revised the guidelines for pediatric basic life support in children who have foreign body aspiration. If the child can cough, breathe, or speak, the foreign body may be dislodged spontaneously. However, if a foreign body aspiration is witnessed or strongly suspected and the child is unconscious, is not breathing, has increased respiratory difficulty with stridor, or has an ineffective cough, one should attempt to clear the airway. For a child, use a series of Heimlich subdiaphragmatic abdominal thrusts in an effort to
create an artificial cough, which forces air, and it is hoped, the foreign body out of the airway. In infants, a combination of five backblows followed by five chest thrusts is recommended as opposed to the Heimlich maneuver, which may cause a fatal liver laceration in this age group. “Blind” finger sweeps should not be performed at any age because the foreign body possibly could be pushed back into the airway, causing further obstruction. In all cases, the emergency medical system should be activated as soon as possible.

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ABSTRACT

Common Red Cell Transfusion Reactions


Clinical Significance of White Cell Antibodies in Febrile Nonhemolytic Transfusion Reactions. Brubaker DB. Transfusion. 1990;30:733–737


Common reactions to the transfusion of red blood cells in children usually are not serious. While fewer than 4% of blood transfusions are associated with reactions, more than 40% of these reactions are febrile without hemolysis, almost 60% are urticarial, and fewer than 1% are hemolytic. Febrile reactions rarely occur after a single transfusion.

Febrile transfusion reactions may be the result of interleukin-6 and tumor necrosis factor alpha, which accumulate in the platelets, leukocytes, and plasma of units of blood after 3 to 7 days of storage, according to Muylle. These substances are endogenous pyrogens. Febrile reactions can be prevented by predischarging the patient with acetaminophen.

Brubaker’s studies showed that 70% of 24 patients who had febrile nonhemolytic transfusion reactions had white blood cell antibodies involved. Thus, many febrile reactions cannot be prevented without the use of a leukodepletion filter (Pall Corporation, Glenn Cove, NY).

Fever, chills, and sweating are treated with acetaminophen and steroids. If severe, a narcotic such as meperidine is added to therapy.

Urticarial transfusion reactions may be seen after a single transfusion. Such reactions may occur as the result of allergy to antigens carried in the donor plasma. Localized urticaria can be treated by interrupting the transfusion and administering an antihistamine. Premedication with an antihistamine often prevents these reactions. Repeated urticarial reactions mandate the use of washed or frozen/washed red blood cells as well as premedication with an antihistamine. If, despite these measures, reactions continue to occur, corticosteroid administration should be added to the pretransfusion regimen.

Hemolytic transfusion reactions are the result of incompatibility between the donor and recipient blood; an antigen reaction results in complement activation. Most often, ABO blood group incompatibility is the cause, but other incompatibilities may exist. Pinkerton notes that careful crossmatching of donor and recipient blood by using the direct and indirect antiglobulin test (Coombs tests) prevents most hemolytic reactions. When blood is mislabeled in the blood bank, or when a patient receives blood designated for another recipient, these serious reactions can occur.

Patients experiencing a hemolytic reaction often relate a sensation of impending doom. These reactions are attended by fever, chills, and pain in the abdomen and lower back. Low blood pressure and tachycardia may progress to kidney failure and shock.

Once the transfusion is interrupted, blood is drawn and tested with the Coombs reagent. A filled capillary tube is spun; pink to red plasma represents hemoglobinemia. Anemia and hemoglobinuria are noted, and spherocytes can be seen on the peripheral blood smear. Disseminated intravascular coagulation often is observed.

Steroids, a saline bolus for hypotension, mannitol to maintain renal cortical blood flow, and pressor drugs to maintain blood circulation are all mainstays of therapy. Despite these measures, the mortality of those who have hemolytic transfusion reactions is high and bears a direct relationship to the volume of
incompatible blood transfused.

Ness evaluated the significance of delayed hemolytic transfusion reactions. He found they occurred in only 0.12% of 854 transfused patients tested. These reactions tend to occur 3 to 10 days following a blood transfusion and are associated with many of these symptoms of acute hemolytic reactions. Minor blood group antigen incompatibilities are documented in almost all cases. Although symptoms usually are milder, severe anemia may develop. When anemia and hyperbilirubinemia develop in a multiply transfused patient 3 to 10 days following a transfusion, a positive Coombs test will document the nature of the anemia. The development of a minor blood group antibody (most often an anti-E and/or an anti-Jka antibody) in the patient is identified. Recognition of such an antibody is important for the future accurate crossmatching of blood for this patient.

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