Exercises may include higher resistance with fewer repetitions. Formal physical therapy may be indicated for severe injuries. Other treatment modalities include whirlpool therapy, ultrasound, or soft tissue massage. Although rarely indicated, surgery may be indicated for patients with severe sprains or elite athletes who are likely to have repeated joint stresses.

Patients can return to play once full, painless range of motion and full strength are obtained and the athlete is able to perform sport-specific tasks. Athletes who return to play with persistent pain and swelling are at risk for reinjury. Athletes playing high-risk sports will benefit from bracing or taping to prevent recurrent injury and improve proprioception.

Although the prognosis for sprains generally is very good with adequate treatment and rehabilitation, complications can include stiffness from prolonged immobilization, recurrent instability, or osteochondral defects. Persistent pain for more than 6 to 8 weeks after an injury may indicate a need for further imaging to rule out a fracture.

Indications for referral to an orthopedist include fracture, dislocation, subluxation, syndesmosis injury (high-ankle sprain), tendon rupture, intra-articular wound, hand injuries, and neurovascular compromise. Clinicians should remind patients to wear appropriate helmets, protective padding, wrist guards, and high-top shoes or lace-up braces to prevent injuries.

Comments: When last we published an In Brief on sprains in 2008, the Ottawa Ankle Rules, which provide indications for radiography in patients with trauma to the ankle, had been well validated only for adults. No surprise that studies involving children were late to come, but, at last, patients no longer have to be old enough to vote for the Rules to apply. As Drs Canares and Lockhart report, the Rules have been validated for children older than 6 years, covering the great majority of pediatric patients with ankle injuries and allowing us to safely reduce their exposure to radiation.

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Editor, In Brief

Perinatal Varicella

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Varicella-zoster virus (VZV) is the human herpesvirus that causes primary varicella infection, known as chickenpox. Latent VZV infection can reactivate as herpes zoster, also called shingles. Before the availability of a highly effective vaccine, VZV was responsible for nearly universal infection in childhood, with very few adults remaining susceptible to primary disease.

The clinical manifestations of chickenpox include a widespread, intensely pruritic vesicular rash. Affected individuals typically have between 250 and 500 lesions in varying stages of evolution, often associated with fever or other systemic symptoms. Infection in immunocompetent hosts generally is self-limited, but may vary greatly in severity. Adolescents, adults, and people who are immunocompromised are at particularly high risk for significant complications from this disease.

Although the most common complication of chickenpox is bacterial superinfection of skin lesions, varicella pneumonia is the most common cause of mortality, and pregnant women appear to be at particularly high risk of mortality from this complication. Other complications of chickenpox infection include postinfectious acute cerebellar ataxia, encephalitis, thrombocytopenia, and Reye Syndrome.

Chickenpox is contracted through contact with respiratory droplets or fluid from the skin lesions of infected persons and also may be transmitted transplacentally. Viral replication occurs initially in the respiratory tract, followed by invasion of local lymph nodes, viremia, and widespread dissemination. The virus then continues to replicate, causing a more significant viremia that results in the eruption of the characteristic skin lesions. New cutaneous lesions emerge with subsequent periods of viremia. The incubation period for varicella typically is between 10 and 21 days, although the administration of varicella-zoster immune globulin (VZIG) or intravenous immune globulin (IVIG) can extend the incubation period up to 28 days, and compromised immunity can decrease the incubation time. Patients with varicella are considered contagious from 1 to 2 days preceding the development of the rash until all lesions have crusted over.

The preferred method for the diagnosis of VZV is polymerase chain reaction or direct fluorescent antibody testing of vesicular swabs or scrapings. Other methods are not as rapid or reliable: viral culture may take up to 1 week to yield results, and Tzanck smear is not specific for VZV. Serial serological testing for immune globulin G (IgG) antibodies may be useful in immunocompetent hosts, but commercially available IgM testing is not reliable.

After primary varicella infection, the virus becomes latent in the dorsal root ganglia and may reactivate later as herpes zoster or shingles. Symptoms of shingles include a vesicular rash in a dermatomal distribution that may be painful and can be followed by postherpetic neuralgia, especially in adults. Shingles may become disseminated in immunocompromised hosts and may be contagious via direct contact with vesicles.

VZV infection during pregnancy is uncommon because of widespread immunity in women of childbearing age. However, two distinct entities may cause significant complications in neonates whose mothers are infected with varicella during pregnancy. The terminology used to describe these disorders can be a source of confusion if it is not used precisely.

Congenital varicella syndrome (CVS) results from the exposure of a fetus to VZV early in pregnancy. This syndrome also has been referred to as fetal varicella syndrome, varicella embryopathy, or varicella fetopathy. The highest risk (2%) occurs when mothers are infected during weeks 13 to 20 of gestation, but CVS may occur with earlier infection or as late as 28 weeks of gestation. The typical features of CVS include a characteristic scarring skin lesion known as a cicatrix that occurs often in a dermatomal distribution. In addition, limb abnormalities such as hypoplasia and atrophy, eye abnormalities such as chorioretinitis, and central nervous system abnormalities such as microcephaly may be seen. Cognitive impairment, seizures, and growth deficiency may be present, and early death is common.

It is important to note that there is a wide range of disease severity, with some children affected severely, whereas others have few symptoms and normal development. Approximately 25% to 36% of women infected during pregnancy will transmit VZV to their fetuses, but fewer than 2% of maternal infections result in CVS. Sampling of fetal blood or placental tissue does not reliably identify cases of CVS as opposed to simple fetal infection. Ultrasonography may be helpful if limb abnormalities or other anomalies can be visualized.

When pregnant women develop varicella infection around the time of delivery, neonatal varicella may occur. Some sources refer to this condition as perinatal varicella or congenital varicella; but neonatal varicella should not be confused with CVS, which occurs with infection early in gestation. When varicella infection occurs just before delivery, infants may be exposed to the virus as it crosses the placenta, but the infant may not remain in utero long enough to receive protective maternal antibody. As a result, these infants may become severely or even fatally ill in the postnatal period. When maternal infection develops from 5 days before to 2 days after delivery, infants are at particularly high risk for severe varicella infection, having an untreated mortality rate as high as 31%. Death typically is from varicella pneumonia.

Infants who are exposed to VZV in utero at more than 28 weeks' gestation, and more than 5 days before delivery, generally are protected from severe infection by the transfer of maternal IgG antibodies across the placenta. However, some of these infants will develop asymptomatic varicella infection in utero and may have positive serology or develop herpes zoster infection in infancy or early childhood, without having had chickenpox infection postnatally.

Prevention of VZV infection through vaccination is key to the prevention of neonatal complications. A highly effective, live-attenuated virus vaccine is available widely in the United States, but vaccination is contraindicated in pregnant women. Because of their increased risk for severe complications from varicella infection, pregnant women who are exposed to VZV should have their susceptibility to the virus determined as soon as possible, generally by serological testing, and postexposure prophylaxis with VZIG should be strongly considered for those who are susceptible.

VZIG should be given as soon as possible, ideally within 72 to 96 hours of exposure, although administration may be useful up to 10 days postexposure. IVIG may also be considered if VZIG is unavailable. VZIG has not been proven to prevent CVS or neonatal varicella

infection, but it is given to decrease the risk of severe maternal complications of infection. Antiviral therapy with acyclovir also may be indicated, especially if severe disease or varicella pneumonia develops.

If signs of maternal chickenpox infection develop from 5 days before through 2 days after delivery, infants should be given VZIG immediately after birth (or IVIG if VZIG is unavailable), even if maternal VZIG was administered. These infants should be observed closely, because many will still develop chickenpox infection, although their risk of severe infection will be decreased. Antiviral therapy with acyclovir may be indicated.

Healthy, term infants who are exposed to VZV postnatally typically are protected by maternal IgG antibody, and postexposure prophylaxis with VZIG generally is not indicated, although some would give VZIG to babies in the first 2 weeks after birth whose mothers are not immune. For preterm infants exposed to chickenpox postnatally, VZIG is indicated for postexposure prophylaxis in those born at less than 28 weeks' gestation, or who have a birthweight 1,000 g or less, and is indicated also for preterm infants born at more than 28 weeks' gestation to susceptible mothers, who will not have passed protective antibody to their infants. Intravenous acyclovir may be useful for the treatment of severe infection or potentially severe infection in any infant with chickenpox. Strict isolation of exposed or infected patients is of critical importance, especially on labor and delivery units, and in NICUs or hospital nurseries.

Although the long-term effects of the administration of VZIG and acyclovir in the perinatal period have not been evaluated well, it is likely that the benefits of mitigating severe, potentially life-threatening VZV infection outweigh the theoretical risk.

Comment: Varicella vaccine, because it contains live virus, is contraindicated during pregnancy, and any young woman of childbearing age who needs vaccination should be warned not to become pregnant for at least a month. What to do when the unfortunate happens?

Of the more than 600 women entered in the VARIVAX Pregnancy Registry* having been vaccinated against varicella shortly before or during pregnancy, none bore a child with features of the congenital varicella syndrome nor was there evidence of congenital varicella in any cases of spontaneous or elective abortion. These reassuring data are not an excuse to be careless, but they do provide a helpful foundation for the difficult decision facing a pregnant woman who was inadvertently vaccinated.

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Answer Key for December 2012 Issue:
Hypertension: 1. A; 2. D; 3. D; 4. D; 5. D
Developmental Dysplasia of the Hip: 1. D; 2. C; 3. E; 4. D; 5. A
Pediatric Headache: 1. C; 2. E; 3. A; 4. C; 5. C

Answer Key for January 2013 Issue:
Menstrual Disorders: 1. C; 2. D; 3. B; 4. B; 5. B
Childhood Obsessive-Compulsive Disorder: 1. E; 2. D; 3. C; 4. D; 5. C
Adolescent Sexuality: 1. E; 2. D; 3. C; 4. A; 5. B

*VARIVAX© is a varicella vaccine manufactured by Merck & Company, Inc, Whitehouse Station, NJ.

Approval by the Food and Drug Administration may not apply to all uses of acyclovir, VZIG, and IVIG discussed in this article.

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