Infective Endocarditis in a Collegiate Wrestler

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ABSTRACT: A 21-year-old collegiate wrestler was admitted to the hospital suffering from acute left lower quadrant abdominal pain. Blood cultures taken at the time of admission showed *Staphylococcus aureus.* The results of a computed tomography scan and a two-dimensional echocardiogram were consistent with a diagnosis of infective endocarditis. Therapy consisted of a 14-day hospitalization, a 28-day course of parenteral antibiotics, and subsequent follow-up visits. He returned to full participation in wrestling after 15 weeks.

The term bacterial infective endocarditis denotes infection of the endocardial surface of the heart due to the presence of a bacterial organism. It is classically associated with infection caused by *Staphylococcus aureus* and other staphylococcal and streptococcal species.[4,10] Infective endocarditis is uncommon in healthy patients with normal heart valve structure and function.[10] A review of the literature yields no reports of infective endocarditis specifically linked to athletic participation.

Considered universally fatal only 45 years ago, this disease still carries great health risks to the infected athlete. Therefore, it is important for the athletic trainer and physician to be aware of the potential of this life-threatening disease and to be ready to initiate appropriate diagnostic and treatment procedures.

CASE REPORT

A 21-year-old Division I collegiate wrestler presented with intermittent fevers and sudden onset of acute left lower quadrant pain that radiated to the groin. The athlete had gone to bed asymptomatic, but awakened several times throughout the night as pain became more frequent and severe. By morning, he was unable to rise from bed unassisted.

Upon admission to the hospital, he exhibited an elevated white blood cell count of $2.19 \times 10^7\ L^{-1}$ and a temperature of 40°C (104°F). Blood cultures drawn at the time of admission subsequently returned positive for *S aureus.* He was observed in the hospital overnight, throughout which time his pain became more intense. He exhibited distinct peritoneal signs. Although localization of the pain was not consistent with appendicitis, this diagnosis could not be ruled out with certainty. Meckel's diverticulitis was also considered.

A diagnostic laparoscopy was performed to determine the source of his abdominal discomfort. The only abnormal finding was the presence of a slight fluid accumulation in the pelvic cavity. Culture of this fluid subsequently proved negative. An abdominal and pelvic computed tomography scan showed low density splenic lesions consistent with multifocal abscesses. An echocardiogram was obtained to rule out infective endocarditis, and an internal medicine consultation was obtained.

The two-dimensional echocardiogram showed an irregular, vegetative lesion on the aortic valve, mild tricuspid insufficiency, and trivial mitral valve insufficiency. A repeat echocardiogram obtained 10 days after the first revealed nodular thickening of the noncoronary cusp of the aortic valve and of the anterior loop of the mitral valve. These findings were also interpreted as consistent with vegetative lesions.

Findings obtained from the initial echocardiogram, coupled with the *S aureus* bacteremia, led to the diagnosis of infective endocarditis. The patient was given intravenous antibiotics (2 g cefazolin sodium IVq8h (Kefzol; Lilly, Indianapolis, IN) and 200 mg gentamicin sulfate IVq8h (Garamycin; Schering, Kenilworth, NJ). Repeat blood cultures were obtained daily for the next 7 days.

The patient's fever subsided gradually over the initial week of hospitalization. After 7 days, his temperature returned to 37°C (98.6°F) and remained there for the rest of his hospital course. His left lower quadrant and left inguinal pain improved daily and he was given physical therapy for assistance with ambulation. He was treated with etodolac (Lodine 300 mg; Wyeth-Ayerst, Philadelphia, PA) and hydrocodone bitartate/acetaminophen (Lortab 2.5 mg/500 mg; Witby, Richmond, VA) as needed for control of pain.

At the time of discharge, his white blood cell count was $9.7 \times 10^6\ L^{-1}$. He was mildly anemic, which is characteristic of chronic disease with hemoglobin of 11.7 g/dL. All other hematologic indices were within normal limits. Additional blood cultures obtained for follow-up showed no growth of *S aureus.* At the end of 14 days of hospitalization, we decided that he could continue the remainder of the 28-day course of antibiotics as an outpatient.

Even before the completion of the antibiotic treatment, the patient was allowed to begin light exercise and physical therapy under the direction of the team physicians and the athletic training staff at the university. He resumed limited activity with the wrestling team 8 weeks following the onset of symptoms and gradually returned to full participation after 15 weeks without complications.

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Upon diagnosis, the athlete was asked about previous history of skin trauma, abuse of street drugs or steroids, or a history of sharing needles. He vehemently denied drug abuse, but indicated a history of wrestling with uncovered wounds because dressings would not stay in place.

A bacterial culture of the wrestling surface revealed *S aureus* in all of the samples tested. Testing to determine the strain of the microorganism was initiated, but samples of the patient's blood were discarded before evaluation. Unfortunately, the haste with which the patient's blood samples were discarded limited the ability to make the definitive link between the disease, the microorganism, and the wrestling surface.

DISCUSSION

Once the incident was reported, definitive diagnosis was critical to the favorable outcome of this athlete's condition. However, simple preventive measures could have prevented this incident from occurring.

Clinical Manifestations

The amount of time that passes between an event likely to cause bacteremia and the onset of infective endocarditis can be as short as 2 weeks.[8] Fever rarely spikes above 39.8°C (103°F)[11] and routinely lasts for no longer than 9 days. Patients may also complain of abdominal pain secondary to emboli to the gastrointestinal tract, liver, spleen, and kidneys.[5]

The patient's history will often reveal fatigue, weakness, nausea and vomiting, headache, and severe muscle pain.[10] He or she may have a history of an abnormal heart condition that results in turbulent blood flow across the heart's valvular surface. Rheumatic heart disease, congenital heart disease (especially patent ductus arteriosus, ventricular septal defect, and bicuspid aortic valve), Marfan's syndrome, and mitral valve prolapse may all cause the turbulence that allows bacteria to attach and proliferate,[4,6,9] although nearly one third of the cases attributed to *S aureus* occur in individuals with no clinically detectable cardiac disease.[1,10] In acute infective endocarditis, the most common infective agent is the *S aureus* bacterium, an agent causing death in nearly 40% of all reported cases.[6]

Pathologic Changes

Bacterial infection of the heart may lead to destruction of the underlying valve, perforation of the valve leaflet, and rupture of the chordae tendinae, intraventricular septum, or papillary muscle. The associated bacteremia may cause pathologic changes in other organs. Kidney abscesses, infarction, and glomerulonephritis have all been reported.[4] Splenic abscess formation, although uncommon, has been reported,[5,10] as have splenic enlargement and infarction.[11]

Treatment

The blood culture is the single most important laboratory test performed to diagnose infective endocarditis.[5] A two-dimensional echocardiogram may be performed to evaluate the heart and associated valve structures. The use of echocardiography in the diagnosis of infective endocarditis was first reported in 1973, and has accurately identified lesions on all valves.[10] A positive echocardiogram should serve as adjunctive evidence to clinical manifestation in order to warrant aggressive treatment with antibiotics and surgical intervention.[3,7]

Although the infective organism may be extremely sensitive to the antibiotics used, complete eradication takes several weeks.[6] Relapse is not uncommon. Parenteral antibiotics are recommended over oral drugs because of the importance of sustained antibacterial activity.[6] To fully eradicate the etiologic agent and reduce the risk of relapse, extended drug administration for 4 to 6 weeks is necessary.

The choice of antibiotic depends on the etiologic agent and should produce a rapid bactericidal effect. In infective endocarditis where *S aureus* is the etiologic agent, synergistic combinations of antibiotics such as a penicillin (nafcillin, methicillin, or oxacillin, 1.5 to 2 g IVq4h) or a cephalosporin (cefazolin, 2 g IVq8h, or cephalothin, 2 g IVq4h) and gentamicin (1.0 to 1.7 mg/kg IVq8h), are often used.

RECOMMENDATIONS

A review of the sports literature on illness in athletics yields no information to suggest that athletes participating in wrestling are at greater risk for infective endocarditis than are other healthy athletes. However, because wrestlers tend to participate on porous mats in humid rooms with inadequate ventilation, bacterial colonization is facilitated. Any microorganism easily transferred via body fluids or direct body contact can infect multiple team members in the course of a single practice session and can cause significant health complications. For example, the transmission of herpes simplex virus type 1, herpes gladiatorum, has been a recognized health risk for wrestlers since the mid-1960s.[2]

In this case report, the athlete had a history of open skin wounds which he managed poorly during practice sessions. Although cause and effect cannot be demonstrated with certainty in this case, the findings strongly suggest a causal relationship between the wrestling activity, the wrestling surface, and the unusual occurrence of infective endocarditis in an otherwise healthy 21-year-old athlete.

This case demonstrates the need for proper hygiene and thorough daily scrubbing of practice surfaces to prevent bacterial colonization, as well as the necessity of proper care of skin wounds. It also stresses the need for immediate, accurate diagnosis, care, and treatment of acute diseases in which etiology is difficult to determine.

To reduce the risk of bacterial infection, the athletic trainer and team physician must work with the coach and the team to inhibit the colonization and spread of bacteria through the practice room. Such an effort requires implementation of daily mat cleaning with a bactericidal agent and installation of an adequate ventilation system in the practice area. Athletes should be encouraged to shower with an antibacterial soap before and after practice, and all members of the team harboring bacterial or viral infections should be removed from

practice until they are deemed noncontagious. Furthermore, athletes with skin abrasions, lacerations, or eczema should be taught proper wound cleansing techniques, be required to practice with the wounds covered with a sterile dressing, and be withheld from participation if the integrity of the skin is seriously compromised.

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