Complete Heart Block in a Child With Varicella

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A case of varicella myocarditis in a previously healthy 6-year-old child was reviewed. The patient presented with third-degree heart block and shock as the sole manifestation of her cardiac involvement. Bradyarrythmias required temporary transvenous pacing. Intravenous acyclovir was used. The patient recovered without permanent sequelae. The natural history, clinical presentation, and treatment of varicella myocarditis are reviewed. (Am J Emerg Med 1993; 11:602-605. Copyright © 1993 by W.B. Saunders Company)

Varicella is a common childhood illness from which most patients recover uneventfully. Serious complications are rare but may be life-threatening, including Reye's syndrome, encephalitis, and pneumonia.[1-6] Myocarditis is an unusual complication that may be lethal due to the sudden development of arrythmias or pump failure. We present the case of a child with varicella who presented with complete heart block to illustrate this rare, serious, but treatable complication of this usually benign illness.

CASE REPORT

A previously healthy 6-year-old female developed typical varicella skin lesions 4 days before admission. Two days before admission the patient developed vomiting and fever. The morning of admission she complained of generalized weakness, sleepiness, and blurred vision. She was unable to get out of bed or walk. Her father found her skin to be cool and could not palpate a pulse. She was brought to the hospital by private car.

In the emergency department (ED), the child was awake but ill-appearing. Her skin was cool and clammy. The temperature was 98.1°F, and her respiratory rate was 30 breaths/min. Her initial pulse was 40 beats/min but rapidly decreased to 20 beats/min. No blood pressure could be auscultated. Femoral pulses were palpable but distal pulses were not, and capillary refill was poor.

Several papular, vesicular, and crusted skin lesions consistent with acute varicella were noted over the patient's trunk and face. Except for bradycardia, the cardiac examination was normal. The lungs were clear, and there was no hepatosplenomegaly. Neurologically, the child was initially alert and appropriately responsive, but as her heart rate decreased, she became increasingly lethargic.

An arterial blood gas was drawn on 100% oxygen by facemask demonstrated a ph of 7.19, a Pco_2 of 20 mm Hg, and a Po_2 of 360 mm Hg. Blood urea nitrogen was 37 mg/dL; creatinine was 2.2 mg/dL; and serum HCO_3 was reported as <10 mEq/L, with otherwise normal electrolytes and glucose. A complete hemogram was normal. The initial creatine phosphokinase (CPK) was 1,726 IU/L with an MB fraction of 14%. The total lactic dehydrogenase (LDH) was 903 IU/L with 49% LDH-1.

Chest x-ray showed a normal cardiac silhouette and clear lung fields. The cardiac monitor initially showed a second-degree heart block with a variable (6:1 or 7:1) conduction (Figure 1). It spontaneously degenerated to a compete heart block with a wide ventricular escape (Figure 2). The electrocardiogram confirmed third-degree heart block but was otherwise normal.

In the ED the child was treated twice with atropine (.02 mg/kg) with transient increases in heart rate and corresponding peripheral pulses after each dose. Once again, the patient become profoundly bradycardic, lethargic, and lost peripheral pulses Epinephrine (.01 mg/kg) administration resulted in an accelerated junctional rhythm associated with strong peripheral pulses and improved mentation. The patient then vomited and developed ventricular tachycardia (Figure 3). This reverted spontaneously to a junctional rhythm in 60 seconds. The child remained awake without change in perfusion during this episode. After resumption of third-degree block, an isoproterenol infusion was initiated (0.1 μg/kg/min). The patient's pulse increased to 120 beats/min with a blood pressure of 105/85 mm Hg and an improvement in peripheral perfusion. A transcutaneous pacemaker was placed but did not capture. The patient was hemodynamically stable and was transported to the pediatric intensive care unit.

The isoproterenol infusion was maintained, and a functioning external pacer was kept on standby. The patient was begun on intravenous acyclovir (1,500 mg/m²/d).

An echocardiogram demonstrated normal left ventricular function. No congenital defects were noted. The patients's hospital course was complicated by two episodes of asystole responsive to

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Manuscript received December 16, 1992; revision accepted April 3, 1993.

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Key Words: Heart block, myocarditis, varicella.

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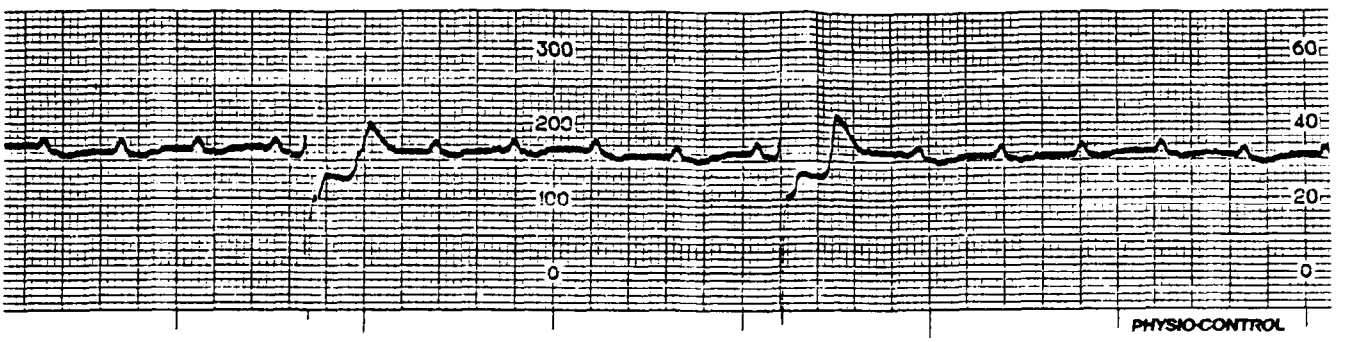

FIGURE 1. Second degree heart block with variable conduction.

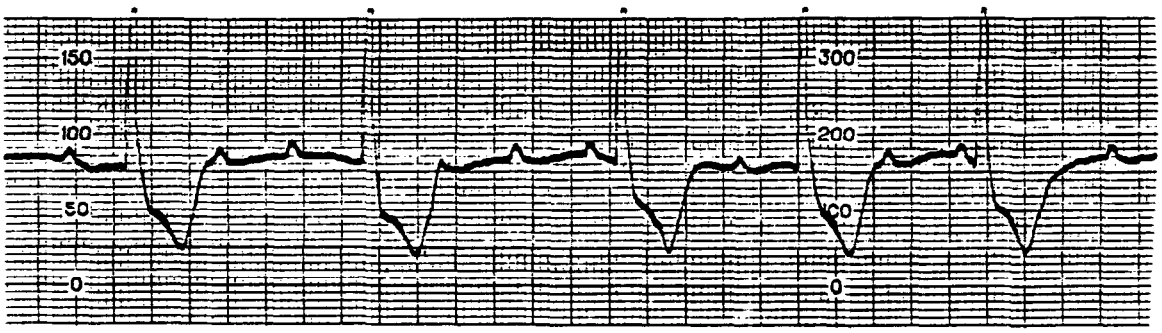

FIGURE 2. Third-degree heart block with ventricular escape rhythm.

standard advanced cardiac life support measures. A transvenous pacemaker was placed, but there was no further episodes of bradycardia or heart block requiring pacing.

Subsequent laboratory studies showed resolution of azotemia and metabolic acidosis and confirmed the presence of CPK/MB and LDH-1 isoenzymes. Follow-up electrocardiogram demonstrated new lateral T-wave abnormalities (Figure 4). A repeat echocardiogram now showed increased ventricular wall thickness suggestive of myocardial edema consistent with myocarditis.

No new varicella skin lesions were noted after hospitalization. The pacemaker and intravenous acyclovir were continued for 6 days. The CPK and LDH normalized by the fifth hospital day. Telemetry demonstrated sinus rhythm without further heart block or ectopy. The child was discharged on no medications on the 10th hospital day. To date, she remains asymptomatic.

DISCUSSION

This case illustrates a rare but recognized complication of varicella—myocarditis-causing conduction and rhythm disturbances. Our patient's course was severe enough to result in shock and ultimately asystole.

Varicella myocarditis was initially described in 1953.[7] The histopathological findings are similar to other viral myocarditides, showing interstitial edema and myofibril degeneration and necrosis. Focal collections of inflammatory cells are also observed. It is unclear whether infection of the myocardium or immune factors are more important in causing myocardial inflammation.[8,9]

The true incidence of varicella myocarditis is not known. Combined retrospective studies in more than 2,500 children hospitalized with varicella have described no patients with symptomatic myocarditis.[2-5] However, autopsies have demonstrated myocardial inflammation in patients dying of varicella, regardless of whether myocarditis had been suspected clinically.[10-13] Furthermore, a prospective study of 312 children admitted with varicella showed that 5.8% had electrocardiographic changes consistent with myocarditis.[14] None of these children were symptomatic, and all electrocardiographic changes resolved over time. No study has prospectively addressed the role of serial enzyme determination or echocardiography in the detection of myocarditis in varicella patients. It is probable that more cases of myocarditis would be identified with more aggressive investigation.

There is little literature describing the natural history and presentation of varicella myocarditis. Waagner and Murphy reviewed 18 cases collected during a 33-year period.[15] The mean age was 6 years. Seventy percent of the patients presented 3 to 5 days after onset of rash, although some presented as late as 2 weeks after skin eruption. Our patient was typical both in age and in the timing of onset of myocarditis.

Less than half of the patients of Waagner and Murphy presented with syncope, dysrhythmia, or congestive heart failure. The remainder were found to have myocarditis during evaluation of abnormal mentation associated with varicella eruption. Myocarditis can mimic a primary neurological complication, on the basis of brain hypoperfusion caused by arrythmias or poor ventricular function. Although neurological sequelae of varicella are more common, abnormal mentation in the setting of varicella should prompt consideration of myocarditis as well.

There is limited mortality information available for varicella myocarditis. In a review of seven infants with acute viral myocarditis, two had died.[16] Of the 16 patients in the review by Waagner and Murphy for whom follow-up was reported, only 9 survived. Two survivors required a permanent pacemaker, and one was left with persistent heart failure.[15,17] Thus morbidity and mortality is significant in symptomatic patients.

Treatment of acute varicella myocarditis is largely supportive. Infectious myocarditis is a well-recognized cause of sudden death in young patients.[18-20] Although data regarding varicella in particular is sparse, recurrent and persistent conduction defects are well-described complications of viral myocarditis in general,[15,21,22] mandating monitoring if the diagnosis is suspected. Tachyarrythmias and conduction defects may occur without warning[23] and have been initial manifestations of myocarditis, unassociated with left ventricular dysfunction.[24] Therefore, temporary transvenous pacing may be the safer choice in a patient who has already demonstrated heart block than reliance on pharmacological agents or external pacing alone.

Acyclovir, an inhibitor of herpesvirus DNA replication, was used in this child's treatment. Because of the rarity of

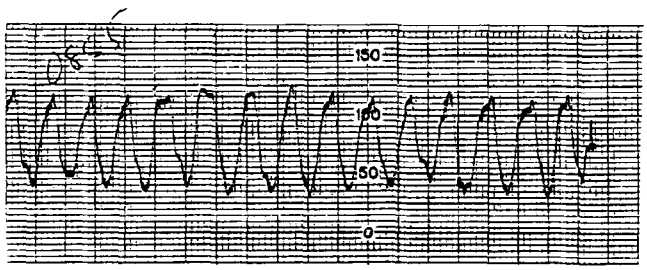

FIGURE 3. Ventricular tachycardia.

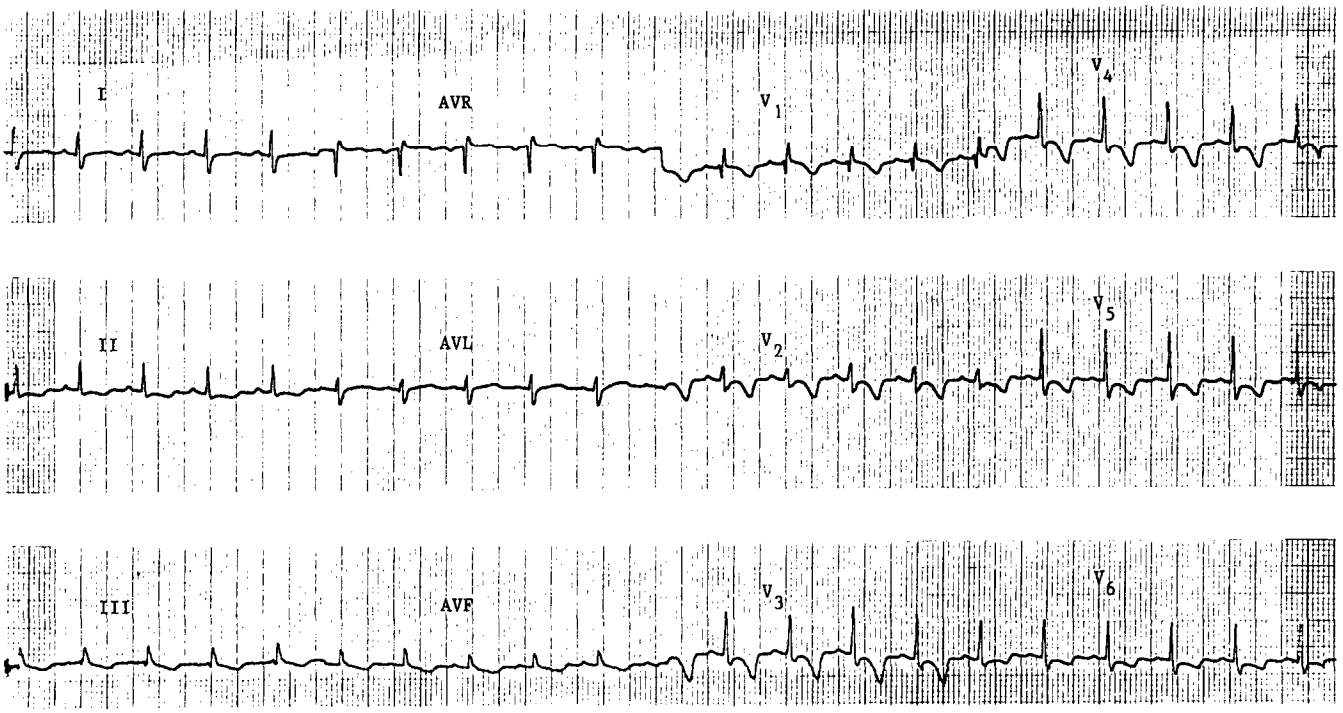

FIGURE 4. Electrocardiogram on discharge.

varicella myocarditis, there is no specific data to support its effectiveness for this particular indication. However, acyclovir is of clear benefit in the treatment of immunocompromised patients with acute varicella.[25,26]

In addition, several recent well-designed studies of immunocompetent children showed that oral acyclovir was effective in reducing the severity of constitutional symptoms, as well as shortening the duration of skin eruptions when used early in acute varicella.[27,28] There was no statistical difference in complication between the acyclovir and placebo groups, probably because of the infrequency of sequelae in this usually benign illness.

In the treatment of immunocompetent adults with varicella, acyclovir is more widely used. However, conclusive data on its efficacy are lacking. One small prospective and several retrospective reports suggest clinical benefit,[29,30] and most experts recommend its use in adults with varicella complications.[25,31] Given its minimal toxicity, acyclovir is probably warranted in the setting of acute symptomatic varicella myocarditis.

Steroids have been used experimentally in patients with myocarditis.[32] However, there have been no controlled studies supporting their effectiveness in infectious myocarditis.[8] Similarly, immunosuppression has not been shown to prevent long-term cardiac morbidity. The use of immunosuppressive drugs for myocarditis remains controversial.

SUMMARY

This case demonstrates the rare but potentially lethal complication of myocarditis in a previously healthy child with varicella. Although varicella myocarditis is likely to be asymptomatic, it may also present with congestive heart failure, tachyarrythmias, or as in this case, acute conduction defects requiring emergent intervention. Treatment is largely supportive. Acyclovir, if used, should be initiated early. Transvenous pacing should be considered in any patient with a high degree atrioventricular block. Because of the potential for serious immediate and long-term morbidity and death, this rare complication should be considered when evaluating patients with acute varicella.

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