



Arkansas Department of Health

4815 West Markham Street • Little Rock, Arkansas 72205-3867 • Telephone (501) 661-2000

Governor Mike Beebe

Paul K. Halverson, DrPH, FACHE, Director and State Health Officer

February 18, 2010

Abbreviated Pandemic Influenza Plan Template for Primary Care Provider Offices:

http://www.cdc.gov/h1n1flu/guidance/pdf/abb_pandemic_influenza_plan.pdf

Fulminant Myocarditis Associated With Pandemic H1N1 Influenza A Virus in Children,

András Bratincsák, Howaida G. El-Said, John S. Bradley, Katayoon Shayan, Paul D., Grossfeld, and Christopher R. Cannavino, *J. Am. Coll. Cardiol.* published online Feb 10, 2010;

<http://content.onlinejacc.org/cgi/content/full/j.jacc.2010.01.004v1>

Acute myocarditis is a well-recognized, albeit rare, manifestation of numerous viral infections with a broad spectrum of symptoms and clinical features. Fulminant myocarditis may present with fatal arrhythmias, atrioventricular block, and/or varying degrees of cardiogenic shock. The prevalence of myocardial involvement in influenza infection ranges from 0 to 11 percent depending on the diagnostic criteria used to define myocarditis.

Fulminant myocarditis is an uncommon complication, typically diagnosed in association with circulatory collapse or at autopsy in patients with influenza-associated fatal outcomes. A few case reports and series represent the incidental diagnoses of influenza-associated acute fulminant myocarditis, but the true prevalence remains unknown. Here is presented the first known report of acute myocarditis in a pediatric population associated with the present pandemic H1N1 influenza A virus infection. Four cases occurred within a 30-day period, and 3 of them were diagnosed as fulminant myocarditis with fatal or near-fatal outcomes.

A retrospective chart review was conducted on all patients admitted to Rady Children's Hospital-San Diego with the diagnosis of H1N1 influenza A infection during October 2009. Criteria for fulminant myocarditis included echocardiographic and clinical evidence of severely decreased left ventricular systolic function and/or lymphocytic infiltration of the myocardium documented at autopsy.

Within a 30-day period, 80 children were admitted with H1N1 influenza A infection to Rady Children's Hospital San Diego. Serum troponin I and creatine phosphokinase myocardial band levels were obtained in 11 children, and echocardiography was performed in 8 children. Included were 4 H1N1 influenza associated myocarditis cases based on elevated cardiac enzymes ($n = 2$), significant acute decrease in left ventricular systolic function demonstrated by the echocardiogram ($n = 3$), or histologic evidence of severe myocarditis. Three children presented with fulminant myocarditis, 1 with a fatal outcome and 2 requiring extracorporeal membrane oxygenation support. None of the children with fulminant myocarditis had evidence of sepsis or

bacterial infection (negative blood, urine, and tracheal aspirate cultures). Two of the 3 children with decreased systolic function experienced recovery in 5 to 7 days. All 4 children had a positive rapid influenza enzyme immunoassay test result from a nasopharyngeal swab sample that was subsequently confirmed as H1N1 by reverse-transcriptase polymerase chain reaction performed at the San Diego County Department of Health.

Fulminant myocarditis due to viral infection is an uncommon form of acute myocarditis. Influenza A virus associated fulminant myocarditis is exceedingly rare, with only a few cases reported in the literature. We report the 1st 4 cases of acute myocarditis in children associated with the pandemic H1N1 influenza A virus, all occurring within a 30-day period. Our tertiary care hospital serves a geographic region that includes approximately 800 000 children. During the past 3 years, there was an annual average of 2 cases of acute myocarditis due to suspected viral etiology, none of which had evidence of influenza infection. Within a 30-day period in October 2009, there were 3 cases of acute fulminant myocarditis and 1 case of acute perimyocarditis at Rady Children's Hospital San Diego, all associated with confirmed H1N1 influenza A infection. There was serologic, echocardiographic, and/or histologic evidence of myocardial involvement in all cases. Three children had echocardiographic evidence of an acutely decreased myocardial function. One child died likely due to acute atrioventricular block, as suggested by severe lymphocytic infiltration of the conduction system. Two children required extracorporeal membrane oxygenation support with gradual improvement of the ventricular systolic function over a 1-week period, which is typically observed in patients with fulminant myocarditis.

The prevalence of influenza-associated fulminant myocarditis is not known because of the lack of comprehensive screening, with only a handful of clinical cases and autopsy findings reported in the literature. Our documented 4 cases within a 30-day period, compared with our previous experience, raise the possibility that the novel [pandemic] H1N1 influenza A virus is more commonly associated with a severe form of myocarditis than previously encountered influenza strains.

Our observations warrant a high index of suspicion for myocarditis in children with H1N1 influenza A infection. Early detection and aggressive management are paramount. Timely intervention with circulatory support may decrease morbidity and mortality, with the potential for a favorable cardiac prognosis.

If you have any questions please feel free to contact Dr. Sandy Snow at 501-661-2169 or fax to 501-661-2300 or e-mail to Sandra.snow@arkansas.gov