our data clearly show that patients with CLD undergoing major hepatic resection fall under a high-risk designation. With the decision to use prophylactic methods against VTE frequently predicated on an assessment of individual patient risk, this information may prove valuable when used in conjunction with the American College of Chest Physicians’ Evidence-Based Clinical Practice Guidelines.4

References

2009 Influenza A(H1N1) Infection and Associated Myocardial Dysfunction

To the Editor:
The authors described the characteristics of patients with A(H1N1) and cardiac dysfunction. However, if we look carefully at the table, the following points merit attention. All the patients had some underlying comorbidity that directly or indirectly affects the cardiac function. The baseline ejection fraction of case 4 was 40%, which might decrease not only because of A(H1N1) infection per se but also because of aseptic bacterial pneumo-monia/sepsis (which was not clearly described in the article). Cases 5 and 6 were pregnant patients in their third trimester. There is a possibility that these two cases may represent peripartum cardiomyopathy (criteria for diagnosis: cardiac failure within last months of pregnancy or within 5 months postpartum, no determinable cause for failure, no previous heart diseases, left ventricular dysfunction with ejection fraction <45%) that improved with treatment. More importantly, the PaO2/Fio2 fraction in all except case 5 (PaO2/Fio2 >300) fulfilled the criteria for ARDS (PaO2/Fio2 ≤200). ARDS is a reasonably well-characterized cause of acute cor pulmonale. Whether A(H1N1) virus induces disproportionate pulmonary vascular disease is not known, although preliminary autopsy results may be compatible with this finding. The thin-walled right side of the heart is particularly susceptible to ischemia and failure in the face of acute increases in afterload. Right-sided heart dysfunction has direct effects on left ventricular diastolic and systolic function. In cases 3 and 5, the APACHE (Acute Physiology And Chronic Health Evaluation) II score in the other four cases varied from 20 to 28, suggesting that these patients were severely ill. In severely ill patients, multiple factors contribute to myocardial dysfunction, including sepsis, pneumonia, ARDS, and associated other organ dysfunction (as described in case 1, the dose of oseltamivir was reduced because of associated severe renal impairment). The rapid downhill course of case 1 (being intubated within 24 h of hospitalization) and partial response to diuretics and inotropes suggests the above possibilities rather than reversible cardiac dysfunction alone.

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Response

To the Editor:

We thank Dr Das for his interest in our case series published in CHEST (May 2010).1 In his letter, Dr Das emphasizes some of the key clinical data presented in Table 1 of our report on six patients with both pandemic 2009 influenza A(H1N1) (A[H1N1]) and cardiac dysfunction. We agree that other conditions, including but not limited to sepsis, multiple organ dysfunction syndrome, and peripartum cardiomyopathy, may have contributed to the cardiac dysfunction observed in our case series. Limited by the nature of a small case series, we were careful not to assign a causal relationship to the observed association between A(H1N1) and cardiac dysfunction. Regarding Dr Das’s proposal of right-sided heart dysfunction contributing to left-sided heart dysfunction, transthoracic echocardiographic assessment of right-sided heart function was normal in all of our cases except the patient in case 6, who had moderate right ventricular dysfunction. We agree that in hospitalized patients it is difficult to distinguish whether cardiac dysfunction is attributable to direct virally mediated myocarditis, systemic inflammatory response, or other comorbidities. However, we believe it prudent for clinicians to be aware of a high prevalence of cardiomyopathy associated with A(H1N1) in hospitalized patients.

Subsequent to submission of our case series, several reports have emerged suggesting a direct effect of A(H1N1) on the myocardium. For example, Puzelli et al2 described an 11-year-old child with A(H1N1) in Italy who died of myopericarditis and had A(H1N1) isolated from both myocardial tissue and pericardial fluid, providing direct evidence for A(H1N1) effects on myocardial cells. Histologic confirmation of myocardial infiltration with A(H1N1) was obtained in a 5-year-old girl whose death was documented in a series of four cases from a single hospital in California over just a 30-day time period.3 Haessler et al4 reported a case of a previously healthy 24-year-old woman with A(H1N1) and left ventricular dysfunction that improved on serial echocardiograms, consistent with our observations. Beyond traditional echocardiographic assessment, tissue Doppler measurements enabled detection of subclinical cardiac dysfunction in patients from Turkey hospitalized with A(H1N1).5 Furthermore, a report from Japan identified that myocarditis associated with A(H1N1) seemed more common than observed with prior seasonal influenza outbreaks.6

We agree that it is difficult to causally relate cardiac dysfunction to a specific etiology in critically ill patients within a small case series. However, it is our belief that physicians managing patients with A(H1N1) should be aware that transient cardiac dysfunction can occur and may be related to viral myocarditis.

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References


Femoral Venous Oxygen Saturation May Still Be a Valuable Tool

To the Editor:

We read with great interest the study reported by Davison et al1 in a recent issue of CHEST (July 2010). We completely agree with their hypothesis and part of their conclusion: Femoral venous oxygen saturation (Svo2) cannot always be used as a substitute for central mixed Sv2 in patients who need critical care. However, we think that femoral Svo2 can be used to guide resuscitation. It has been established that regional differences in oxygen consumption account for the regional differences in Svo2; hence, Svo2 measurements in the superior vena cava (SVC), inferior vena cava (IVC), and pulmonary artery (PA) can diverge significantly.12 Cerebral and cardiac consumption account largely for the Svo2 in the SVC and PA, whereas IVC Svo2 reflects mainly liver, kidney, gut, and skin consumption.3 Thus, the oxygen content in the IVC reflects only a part of the total oxygenation of a patient who is critically ill, but the clinical difference with the Svo2 in the SVC is lessened if the trend in femoral Svo2 is used, rather than the absolute value. Trends can be extremely helpful.

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