Acute Shock Dengue Myocarditis

To the Editor,

The recent report on acute shock dengue myocarditis is of great interest. Guadalajara-Boo et al. report this finding in a case of dengue. In fact, fatal dengue myocarditis due to a dengue virus-induced cardiac lesion was reported a few years ago. An interesting question concerns the increased severity of the disease. According to the previous report from a highly endemic area, Thailand, dengue myocarditis is rare but is not fatal. This observation has many possible explanations. Basic fluid management is the key therapeutic strategy for any dengue case and proper management avoids severe complications. However, because cardiac complications of dengue seem to be a rare presentation, diagnosis can be delayed, resulting in high fatality. This problem can be seen in nonendemic areas with a new emerging dengue problem where physicians lack experience in the management of dengue. In addition, a remaining question is whether there are any genetic mutations in the dengue virus that may have increased its cardiac pathogenicity. Further research on this topic is required.

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Acute Shock Dengue Myocarditis. Response

To the Editor,

I appreciate Viroj Wiwanitkit comments on our recently published paper: “Histologic and Angiographic Imaging of Acute Shock Dengue Myocarditis”. I would like to emphasize a couple of points:

• In this case, dengue shock was treated with intravenous fluids and norepinephrine to maintain tissue perfusion, which could not have been achieved with intravenous fluids alone.
• The acute myocarditis appeared on the eighth day of admission and was treated in phase 1 of myocarditis (viral replication) with antiviral therapy (etiological treatment) and methylprednisolone, avoiding autoimmune inflammatory myocardial damage (phase 2). This management prevented permanent myocardial damage (phase 3) and eventual death. Spontaneous remission has previously been reported, and therefore we cannot be sure whether this would have occurred in our patient or whether our treatment really changed natural course of disease.

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REFERENCES


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