Reply to Thomas et al

To the Editor—We agree with Thomas et al that classifying patients with dengue can be difficult [1]. However, dengue shock syndrome (DSS) is a unique clinical entity that follows a very typical course; after 4–5 days of general systemic symptoms, previously healthy children and young adults present around the time of defervescence with substantial plasma leakage and hypovolemic shock, sometimes accompanied by bleeding. Urgent intervention is crucial to prevent progression to profound or refractory shock, which is often complicated by severe hemorrhage, organ failure, and death [2]. We use the standard definition for DSS that is familiar to most physicians working in endemic areas—a history consistent with dengue, with hemodynamic compromise defined as either narrowing of the pulse pressure or hypotension for age together with evidence of impaired perfusion—with the caveat that the definition refers to hypovolemia due to vascular leakage rather than due to blood loss [3]. Although gastrointestinal losses may result in some degree of dehydration during the initial febrile phase, it is highly unlikely that “straightforward dehydration” could explain these clinical signs. First, patients presenting with DSS usually look surprisingly well. They do not exhibit any of the conventional signs of dehydration, such as dry lips, sunken eyes, or reduced skin turgor, and yet in our series the median percentage hemoconcentration at presentation was 35% (interquartile range, 27%–45%). This level of dehydration should be immediately obvious to the treating physician, and indeed it is partly because most patients look tired but otherwise unremarkable that close attention to pulse pressure and general cardiovascular status are so strongly encouraged, to allow prompt identification during the early phase of compensated shock. Second, although pleural and peritoneal effusions are rarely present at onset of DSS, within 12–24 hours of commencing fluid resuscitation, approximately 30% of our subjects developed clinically detectable effusions [3], and >95% of those assessed radiologically had detectable effusions (unpublished data); this is not consistent with hypovolemia due to dehydration alone.

Thomas et al also suggest addition of complex criteria to the diagnosis of DSS, including a number of laboratory investigations, use of intensive care unit severity scoring systems, and presence of “indisputable diagnostic features of plasma leakage.” However, the vast majority of DSS cases are managed in healthcare facilities without immediate access to blood gas analysis, coagulation screening, or urgent radiological investigations, and none of the scoring systems mentioned are relevant to children (the primary DSS population) or have been evaluated in dengue-endemic areas to assess their utility. Finally, “indisputable evidence of leakage” is rarely apparent until after resuscitation has commenced. We feel strongly that the diagnosis of DSS should remain practical and simple [2], designed to be immediately within the grasp of everyday clinicians working in environments where they are faced with identifying and treating hundreds of such cases annually. In our case series of direct admissions, patient outcomes were excellent, largely due to prompt diagnosis and skilled management by experienced personnel. Undoubtedly, mortality is higher in referred cases or those diagnosed late in the illness evolution [4–6].

Note

Potential conflicts of interest. Both authors: No reported conflicts.
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References


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