

To: Fulminant myocarditis associated with the H1N1 influenza virus: case report and literature review

Para: Miocardite fulminante associada ao vírus influenza H1N1: relato de caso e revisão de literatura

To the Editor,

We would like to discuss the recent publication titled “Fulminant myocarditis associated with the H1N1 influenza virus: case report and literature review.”⁽¹⁾ In this report, Lobo et al. noted that “the H1N1 influenza virus should be considered an etiologic agent of myocarditis”⁽¹⁾ and concluded that “the use of extracorporeal membrane oxygenation therapy appears promising but has not yet been routinely implemented in underdeveloped countries.”⁽¹⁾ Indeed, myocarditis is sporadically reported in the course of H1N1 influenza infection. In our experience, the use of extracorporeal membrane oxygenation therapy is effective.⁽²⁾ Nevertheless, complications can also occur after using extracorporeal membrane oxygenation therapy. Oda et al. reported spinal infarction as an important complication.⁽³⁾ Focusing on other alternative treatments, Busani et al. recently reported the effectiveness of levosimendan.⁽⁴⁾ The efficacy of this new alternative treatment should be further assessed. Finally, cardiac pathology due to H1N1 influenza infection can be reversible.⁽⁵⁾ Hence, aggressive management and supportive care is required. The case reported by Lobo et al.⁽¹⁾ was diagnosed with H1N1 influenza virus infection based on a positive PCR test of nasopharyngeal secretions swab. However, this case of fulminant myocarditis could have resulted from either a direct clinical association with H1N1 influenza or a coincidental concomitant illness. To determine whether the H1N1 influenza virus induced myocarditis, an RT-PCR test should be performed to confirm the presence of the virus in the tissue specimen.⁽⁶⁾ In fact, the existence of myocarditis in cases with H1N1 influenza might or might not relate to the clinical presentation of H1N1.⁽⁷⁾ Thus, in the present case, the possibility of pre-existing silent myocarditis due to other causes cannot be ruled out. The histopathological finding of “lymphocyte infiltration with degeneration of some myocytes” in the present case report is also discordant with a previous report that the hallmark histopathological finding is “lymphocyte and macrophage infiltration with surrounding cardiomyocyte necrosis”.⁽⁶⁾

Conflicts of interest: None.

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AUTHORS' RESPONSE

We appreciate the points that you raise, and we understand that your discussion is very relevant to improving the care of patients with fulminant myocarditis.

While studies suggest that extracorporeal membrane oxygenation (ECMO) is an effective therapy for fulminant myocarditis,⁽¹⁾ ECMO is not routinely available in our unit. In fact, ECMO was first implemented in our ward after the reported case, and since then, we are advancing our ability to implement this form of care.

In regard to levosimendan, despite some promising results, there are currently no official indications for its use in patients under 18 years of age. It is also an expensive drug, and it is not widely available in developing countries. Although positive pediatric experiences with this drug have been reported,^(2,3) these clinical perceptions remain to be demonstrated in randomized controlled studies.⁽⁴⁾

We also agree that cases of fulminant myocarditis must be aggressively managed and that supportive care should be provided.

We recognize that it is not known whether the H1N1 influenza virus was the direct etiological agent of fulminant myocarditis in our patient, because a tissue RT-PCR test was not available in our unit. On the other hand, the etiology of fulminant myocarditis was likely to be influenza H1N1 in our patient because of the patient's

clinical history, previous good health, and initial clinical presentation with fever, cough and rhinorrhea, followed by acute signs and symptoms of heart failure, together with a positive nasopharyngeal secretion test. Furthermore, no other microbiological agent was identified.

Regarding the histopathological findings in this patient, Bratincsák et al.⁽⁵⁾ defined lymphocytic infiltration of the myocardium at autopsy as one of the criteria for fulminant myocarditis. Cabral et al.⁽⁶⁾ described the case of a 10-year-old boy with fulminant myocarditis associated with influenza A virus infection; the histopathological findings at autopsy were multifocal infiltrates comprising mostly lymphocytes. You suggest that the degeneration of some myocytes described in our report differs from the cardiomyocyte necrosis that has been observed previously; however, we believe that our findings are similar to previous findings and that a translation error may have been to blame for any perceived difference.

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