

Glucocorticoid and immunoglobulin to treat viral fulminant myocarditis

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This Commentary refers to: 'Coronavirus fulminant myocarditis saved with glucocorticoid and human immunoglobulin', by H. Hu et al., doi:10.1093/eurheartj/ehaa190.

There is little experience in the treatment of coronavirus fulminant myocarditis.¹ Glucocorticoid treatment of viral myocarditis is controversial. Because of a lack of high-quality clinical research evidence, the timing, dosage, and duration of glucocorticoid usage are still uncertain. Glucocorticoids have the function of suppressing the immune response, acting as anti-inflammatories, against shock, eliminating an allergic reaction, inhibiting inflammatory oedema, and reducing adverse effects of toxins and inflammatory factors on the myocardium. In 2013, a meta-analysis showed that the left ventricular function improved more obviously in viral myocarditis cases treated with glucocorticoid. Replication of the virus did not increase, which means that glucocorticoid is relatively safe.²

Intravenous immunoglobulin (IVIG) has dual functions: antiviral and anti-inflammatory. It can to a certain extent inhibit virus replication. Some studies indicate that IVIG has a certain value in treating fulminant myocarditis;³ but, most importantly, it should be used at a sufficient dosage at an early stage!

The position paper from the ESC Working Group on the aetiology, diagnosis, management, and treatment of myocarditis recommends that glucocorticoid and IVIG can be considered in selected patients.⁴ We observed that early use of glucocorticoids and IVIG in some patients can shorten the course of fulminant myocarditis and reduce the incidence of multi-organ failure. We are carrying out studies on this.

Viral myocarditis should be treated with antiviral therapy. However, few human studies have been reported.⁵ Meanwhile, viral invasion, replication, and direct damage to the heart often occur at an early stage of the disease. The patient in this case had already been sick for several days before going to see a doctor and had no symptoms such as viraemia and viral pneumonia. Therefore, we did not give specific antiviral drugs in this case. In view of the clinical evidence of bacterial infection in this case, we gave him antibacterial treatment.

We believe that the application of a glucocorticoid and IVIG in the treatment of fulminant myocarditis is reasonable and worthy of further exploration. As the mechanism of coronavirus myocardial damage is still not yet confirmed and may be different from this case, we should be cautious in the use of a glucocorticoid and IVIG for COVID-19 patients with myocardial damage.

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