Editorial

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Beyond neurology: unravelling Nipah virus's cardiovascular conundrum—an editorial

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Dear Editor,

Nipah virus (NiV) is a zoonotic pathogen belonging to the genus *Henipavirus* within the family Paramyxoviridae. First identified in Malaysia in 1998 during an outbreak of severe encephalitis in humans and respiratory illness in pigs, NiV has since emerged periodically in outbreaks primarily in South and Southeast Asia[1]. While NiV is primarily recognized for its neurological manifestations, recent studies have highlighted its potential cardiovascular implications (Fig. 1) shedding light on a broader spectrum of clinical presentations and complications associated with this deadly pathogen[2,3].

Cardiovascular manifestations of NiV infection can be profound and diverse. Although neurological symptoms dominate the clinical picture, involvement of the cardiovascular system can significantly impact disease severity and prognosis. One of the key cardiovascular manifestations observed in NiV infection is myocarditis, characterized by myocardial inflammation[4,5]. Myocarditis can lead to myocardial dysfunction, arrhythmias, and even heart failure[5–7]. Histopathological studies have revealed lymphocytic infiltrates in the myocardium of NiV-infected individuals, indicating an inflammatory response directly affecting the heart[4,8].

Furthermore, NiV infection is associated with vascular compromise, including vasculitis and endothelial dysfunction[9]. The virus can directly infect endothelial cells, leading to endotheliitis and the disruption of vascular integrity[10]. This endothelial damage may contribute to the development of disseminated intravascular coagulation (DIC), a serious complication characterized by widespread activation of coagulation factors, leading to thrombotic occlusion of blood vessels and subsequent organ dysfunction[11,12]. DIC can further exacerbate cardiovascular compromise, leading to multi-organ failure and mortality[13].

Additionally, autopsies of NiV-infected individuals have revealed microvascular thrombosis in various organs, including the heart[14]. These thrombotic events can impair coronary blood flow, leading to myocardial ischemia and infarction. The combination of myocardial inflammation, endothelial dysfunction, and microvascular thrombosis underscores the complex interplay between the NiV and cardiovascular system.

Electrocardiographic abnormalities such as sinus tachycardia are commonly observed in NiV-infected patients and may reflect underlying myocardial involvement[15,16]. These electrocardiographic changes may serve as valuable markers of cardiovascular complications and can aid in the risk stratification and management of patients with NiV infection.

Moreover, the systemic inflammatory response triggered by the Nipah virus infection can contribute to endothelial activation and dysfunction, predisposing individuals to thrombotic events and atherosclerosis[17]. Chronic inflammation and endothelial dysfunction may persist even after the resolution of acute viral infection as seen in hamster models, increasing the risk of long-term cardiovascular complications such as myocardial infarction and stroke[18].

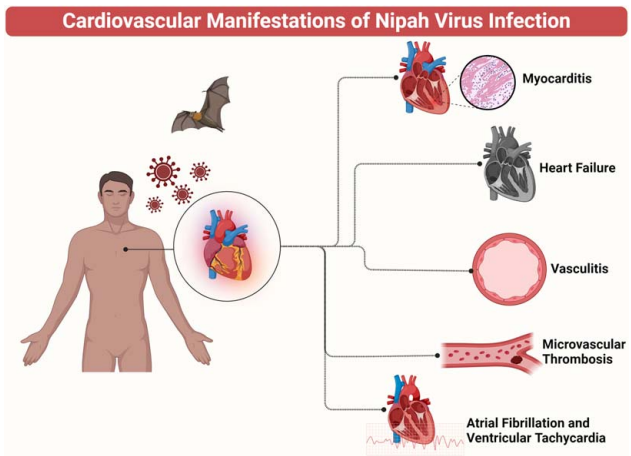

Figure 1. Clinical manifestations of the Nipah virus (NiV) infection. [Created in Biorender.com].

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In conclusion, NiV infection is associated with significant cardiovascular manifestations including myocarditis, endothelial dysfunction, vascular compromise, and thrombotic events. These cardiovascular complications can contribute to morbidity and mortality associated with NiV infection and underscore the importance of comprehensive monitoring and management of cardiovascular health in individuals affected by this deadly pathogen. Further research is needed to elucidate the mechanisms underlying NiV-induced cardiovascular injury and to develop targeted therapeutic strategies to mitigate its impact on patient outcomes.

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