Introduction
Non-polio enteroviruses (EVs) are a frequently encountered group of RNA viruses from the family Picornaviridae. In neonates and young infants, the majority of documented EV-associated tachyarrhythmias are ventricular in origin (Freund et al., 2010; Hawkes & Vaudry, 2005). Among the few supraventricular tachycardias related to EV myocarditis, most cases have been reported to be resistant to antiarrhythmic agents with subsequent ventricular or atrial dilation and impaired cardiac function (Petroni et al., 2012; Simpson et al., 2009). In this context, isolated elevation of transaminases may be falsely attributed to cardiac failure instead of viral hepatic toxicity. Mild courses of EV infections are likely to elude diagnosis.

Case report
Prior to routine vaccination, a 3-month-old female infant presented with heart rates of 280–300 beats min\(^{-1}\) (b.p.m.). A 1-day-history of mild irritability and feeding difficulties preceded the diagnosis of tachycardia. Infant and family members had not experienced fever or other symptoms of infection. Upon admission to hospital, the patient’s electrocardiogram (ECG) revealed a narrow-QRS-complex tachycardia with no visible p-waves and a maximal heart rate of 300 b.p.m. After diagnosis of supraventricular re-entry tachycardia, administration of adenosine successfully re-established sinus rhythm with no signs of pre-excitation. Oral propranolol medication was started and gradually increased to 3 mg kg\(^{-1}\) day\(^{-1}\). Two self-limiting episodes of recurrent tachycardia (heart rate 280 b.p.m.) were noted before the final dose of propranolol was achieved. Echocardiography was unremarkable with normal cardiac structure and contractility (shortening fraction 35%, ejection fraction 72%).

Laboratory investigations yielded elevated liver enzymes [aspartate aminotransferase (AST) 155 U l\(^{-1}\), reference value < 35 U l\(^{-1}\); alanine aminotransferase (ALT) 144 U l\(^{-1}\), reference value < 65 U l\(^{-1}\)], elevated alkaline phosphatase (435 U l\(^{-1}\), reference value 124–341 U l\(^{-1}\)), and a normal total bilirubin level of 0.98 mg dl\(^{-1}\) (reference value < 1.2 mg dl\(^{-1}\)). Markers of infection and cardiac enzymes were within normal ranges. Further diagnostic work-up disclosed the presence of EV/rhinovirus RNA in both stool samples and nasopharyngeal swab aspirate using a RespiFinder SMART 22 Assay (Pathfinder). Results were negative for cytomegalovirus, toxoplasmosis, hepatitis virus A, B and C, herpes simplex viruses 1 and 2, parvovirus, rubella virus and Epstein–Barr virus.

Keywords: concurrent hepatic inflammation; oral propranolol medication; recurrent supraventricular re-entry tachycardia; systemic enterovirus infection.

Received 25 March 2015
Accepted 3 April 2015
Propranolol medication was continued and the infant’s general condition remained stable. Peak values of transaminases were measured on day 7 (AST 174 U l\(^{-1}\); ALT 160 U l\(^{-1}\)), followed by a significant decrease (AST 123 U l\(^{-1}\); ALT 125 U l\(^{-1}\)) on dismissal 10 days after admission. Follow-up Holter ECGs displayed no relapse of rhythm disorders within a period of 4 weeks.

**Discussion**

Cardiovascular manifestations and hepatitis carry the highest rates of mortality and morbidity among organ-specific complications of EV infections (Hawkes & Vaudry, 2005). Neonates and young infants in particular are at risk of severe damage, with serious chronic sequelae (Freund et al., 2010). Clinical manifestations of EV myocarditis include tachyarrhythmia and progressive heart failure (Lin et al., 2003; Petroni et al., 2012). However, the patient we present in this report displayed a relatively benign course of the disease. Supraventricular re-entry tachycardia proved sensitive to adenosine. Under continuous propranolol medication, only two short, self-limiting relapses occurred. In previous reports, the mode of supraventricular tachycardia was diversified as ectopic atrial flutter, multifocal atrial tachycardia or junctional rhythm (Banjac et al., 2014; Petroni et al., 2012; Simpson et al., 2009). Several authors have emphasized difficulties in medical treatment of EV-associated dysrhythmias (Barton et al., 2015; Brunetti & DeSantis, 2008; Petroni et al., 2012; Simpson et al., 2009). Combinations of two or more antiarrhythmic agents were required to prevent progression of congestive heart failure. The mechanisms of arrhythmia in EV myocarditis remain unclear. Both direct viral-mediated cytotoxicity and indirect injury via the immune response have been suggested to be causative (Brunetti & DeSantis, 2008). Despite a lack of elevated cardiac enzymes and inflammatory parameters in our patient, the presence of focal atrial myocarditis was highly probable. Limited areas of damage may account for ventricular structural integrity. In the context of EV infection, hepatic necrosis and coagulopathy have been associated with a high rate of mortality (24%; Lin et al., 2003). Fortunately hepatic involvement in our patient was restricted to an elevation of serum transaminase levels greater than three times the normal level, with a maximum level 7 days after cessation of the supraventricular re-entry tachycardia. No evidence of extensive hepatic necrosis or haemorrhage emerged. Treatment options include supportive therapy and intravenous immunoglobulin in immunocompromised patients; no specific antiviral agent is available.

The concurrence of supraventricular re-entry tachycardia and elevated liver enzymes may point to an underlying EV infection in an otherwise oligosymptomatic infant. Identification of the pathogen will expedite efficient clinical management of the highly variable and potentially life-threatening cardiac and hepatic manifestations.

**Acknowledgements**

The publication of this case report is in accordance with a vote from the local ethical committee of the University of Witten/Herdecke. None of the authors reports any conflict of interest relevant to this case report.

**References**


