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CASE REPORT

Sustained polymorphic ventricular tachycardia in a calf

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Öz

Naseri A, Ider M, Ok M. Bir buzağıda sürekli polimorfik ventriküler taşikardi.

Sürekli polimorfik ventriküler taşikardi, ventrikülerin birden fazla odağından kaynaklanan bir ventriküler aritmidir ve ventriküler fibrilasyon gibi ölümcül aritmilere ve ani ölüme yol açabilmektedir. Yedi günlük Holştayn dişi bir buzağı Selçuk üniversitesi, Veteriner Fakültesi, büyük hayvan hastanesine ani kollaps şikayeti ile getirildi. Elektrokardiyografi bulguları sürekli polimorfik ventriküler taşikardi'yi gösterdi. Kardiyak hasar şüphesi kardiyak troponin I (cTnI), kreatin kinaz- MB (CK-MB) ve kreatin fosfokinaz (CPK) düzeylerindeki artışla doğrulandı.

Anahtar kelimeler: Buzağı, ventriküler taşikardi, polimorfik, kardiyak biyobelirteç.

Abstract

Naseri A, Ider M, Ok M. Sustained polymorphic ventricular tachycardia in a calf.

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Sustained polymorphic ventricular tachycardia is a type of ventricular dysrhythmias which originated from more than one focus in ventricle and may lead to fatal arrhythmias like as ventricular fibrillation and sudden death. A seven days old female Holstein calve was referred to the Large Animals Hospital of the Faculty of Veterinary Medicine of Selcuk University with a history of sudden collapse. Electrocardiography examination showed sustained polymorphic ventricular tachycardia. Cardiac damage suspicion confirmed by elevation in serum cardiac troponin I (cTnI), creatine kinase-MB (CK-MB) and creatine phosphokinase (CPK) concentrations as cardiac damage enzymes.

Keywords: Calf, ventricular tachycardia, polymorphic, cardiac biomarkers.

Cardiac arrhythmias are abnormalities in the heart rate, rhythm, or conduction pattern (Smith 2009). From a clinical perspective, it is useful to categorized cardiac dysrhythmias by their site of origin (supraventricular or ventricular) and by their rate (bradydysrhythmias or tachydysrhythmias) (Bonagura and Miller 1985, Bonagura and Miller 1986). Most abnormal arrhythmias of horses and cattle are tachyarrhythmias (Smith 2009). Ventricular arrhythmias commonly occur in the severe cardiac disease and final stages of heart failure (Radostits et al 2007). Principal concern of ventricular arrhythmias is hemodynamic consequences (Reed et al 2004) and usually if uncorrected may lead to ventricular fibrillation and death (Radostits et al 2007).

A seven days old female Holstein calve was referred to the Large Animals Hospital of the Faculty of Veterinary Medicine of Selcuk University with a history of sudden collapse. In the primary examination, calf was comatos and in lateral recumbency. After admission to the intensive care unit, clinical, laboratory and electrocardiographic examinations were performed. In the first evolution, the cardiac rhythm was irregular and the heart rate interestingly was 2 fold (HR: 220/min) upper the maximum limits of normal values. In the ECG study, base apex lead system was performed for explore the genus of arrhythmia.

Recorded ECG traces showed sustained polymorphic ventricular tachycardia which characterized by rapid heartbeats accompanied by continuously bizarre QRS complexes and abnormal T waves (Figure 1, 2). Before preparing any antiarrhythmic treatment, ventricular tachycardia degenerated to ventricular fibrillation (Figure 3) and calf died. Laboratory findings showed significant increase in serum concentrations of cTnI, CK-MB and CPK (Table 1).

Ventricular tachycardia (VT) is a cardiac arrhythmia characterized by a rapid rhythm originating in the ventricle (Smith 2009). A series of four or more premature ventricular contractions is diagnostic of ventricular tachycardia (McGuirk and Muir 1985). Polymorphic ventricular tachycardia occurs when the ventricular premature complexes (VPCs) originate from more than one focus in the ventricle, creating abnormal QRS and T complexes of different orientations (Marr and Bowen 2010). Ventricular re-entry is an important mechanism for the development of sustained VT (Smith 2009).

Ventricular tachycardia can occur when there is myocarditis, myocardial necrosis, bacterial endocarditis, autonomic nervous system imbalance, hypoxia, ischemia, electrolyte or metabolic disturbances, sepsis or unknown causes (Smith 2009).

Cardiac injury in calves has been identified by increased cTnI in endotoxemia (Peek et al 2008) and myocarditis (Tunca et al 2008, Karapinar et al 2010). Additionally, elevation of CK-MB as cardiac damage biomarker in critically ill calves (Aydogdu et al 2016) and less specific cardiac damage isoenzyme, CPK, may support the prospective injury of cardiac tissue (Kemp et al 2004, Peek et al 2008, Smith 2009). In the present case, association of sustained polymorphic ventricu-

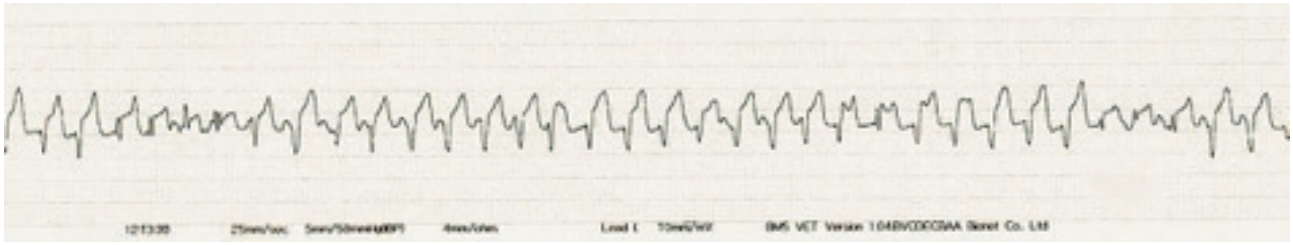

Figure 1. ECG tracing shows abnormal QRS complexes and T waves (Lead I, 25 mm/sec and 10 mm/mV).

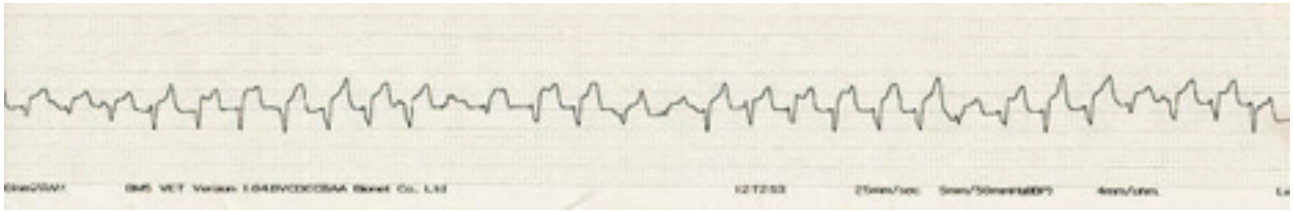

Figure 2. ECG tracing shows continuously polymorphic complexes at 200 beat/min heart rate (Lead II, 25 mm/sec and 10 mm/mV).

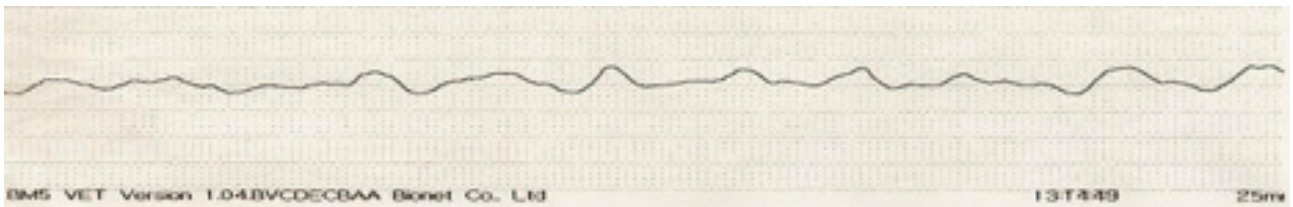

Figure 3. ECG showing ventricular fibrillation. There is no any coordinated contraction (Lead II, 25 mm/sec and 10 mm/mV)

Table 1. The Levels of cardiac biomarker of calf

Parameters	Value	Reference ranges	
cTnI	40.1 pg/mL	0-26	pg/ml
CK-MB	808 U/L	17-23	U/L
CPK	1777 U/L	66-120	U/L

lar tachycardia and elevation of cTnI, CK-MB and CPK enzymes levels may be confirmed development cardiac damage and poor prognosis.

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