Cardiopathological Observation on a Case of Persistent Ventricular Tachycardia in a Pony Mare

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ABSTRACT. A 20-year-old pony mare with persistent ventricular tachycardia (VT) was examined cardiopathologically. At necropsy, the heart was enlarged and rounded with both ventricular dilatation. In a longitudinal section of the ventricular septum, a large grayish white patchy lesion (5 x 25 mm) was detected in the relatively higher portion. Microscopically, the lesion was extensive myocardial fibrosis located in the vicinity of the proximal part of the left bundle branch. Partially the fibrotic lesion was in contact with the branch. Such a lesion might play an important role in creating a suitable background for the development of VT via automaticity or reentry mechanism.—Key words: cardiopathology, horse, ventricular tachycardia.


Ventricular tachycardia (VT) is an arrhythmia due to a number of ventricular ectopic beats occurring in succession. The suggested mechanisms of VT include enhanced automaticity at a focal ectopic site and reentry within the ventricular specialized conduction system [13]. Paroxysmal or persistent VT, although uncommon in horses, is a dangerous arrhythmia because it may progress to fatal ventricular fibrillation [7].

Cardiopathological changes, such as myocardial fibrosis, myocardial infarction, and myocarditis, have been demonstrated in some horses with this type of cardiac arrhythmia [5, 6, 12]. In general, however, equine VT seems to have been associated with gastrointestinal diseases, septicemia, toxemia or electrolyte imbalances [2, 3, 11, 15, 18, 19]. VT has also been reported in apparently healthy horses [6]. Thus, there may not always be clear evidence of a causal relationship between VT and primary cardiac disease.

This paper describes electrocardiographical and cardiopathological findings in an aged pony mare with persistent VT.

A 20-year-old crossbred pony mare was examined with a complaint of anorexia, depression, and distension of the abdomen (day 1). There was marked edema in the neck, axilla, sternum, and lower extremities. The abdominal girth was increased, and a fluid wave was demonstrable. Auscultation revealed variation in intensity of the first heart sound in the presence of highly regular tachycardia at the rate of approximately 160 beats per min.

An electrocardiogram demonstrated an atrioventricular dissociation due to VT with a slower atrial rhythm at a rate of 53 per min independent of a markedly faster ventricular rate (160 per min) (Fig. 1). P waves could be detected in or between the QRS-T complexes, demonstrating a lack of relationship between the P waves and the QRS complexes. The R-R intervals were regular. The QRS conformation was rS type in a bipolar chest lead (A-B lead) and not markedly different from the horse's normal QRS complexes recorded in this lead. The QRS duration of such beats was within, or slightly above, the normal range. Such findings on the configuration and duration of the ventricular beats appeared to suggest an ectopic pacemaker or a reentry pathway located in or near the proximal part of the specialized conduction system of the ventricle. No ventricular captures or fusion beats occurred during more than 30 min electrocardiographic examination.

Hematological findings were all within normal range. Biochemical abnormalities detected were elevated serum GOT, γ-GTP, and LDH activities. Serum electrolyte values were as follows: sodium 139 mEq/l, potassium 4.9 mEq/l, chloride 100 mEq/l, and they were within normal range.

A clinical diagnosis of congestive heart failure, probably due to persistent VT, was made on the basis of these findings. The mare was given a two-week course of 200 mg of oral ubidecarenone therapy, which showed no clinical improvement.

She was given intravenous propranolol hydrochloride therapy (0.02 mg/kg) on day 14 and quinidine sulfate therapy (two doses of 6 g each) on day 40 (Fig. 2), but none of them led to conversion of VT to sinus rhythm. The mare was followed up periodically by clinical and electrocardiographic examinations for about 5 months thereafter. Although sustained VT was always recorded with no conversion to sinus rhythm, she was asymptomatic over the period without any further therapy.

On the morning of day 201, the mare was noticed by the owner to be slightly dyspneic and lethargic. Ventricular premature beats, which were of a type different from the beats constituting the VT, were seen in the electrocardiographic recordings at this time (Fig. 3). Runs of VT were produced by the same type of ectopic beats as previously recorded. The rates of the P waves were almost equal to those of the QRS complexes (125 per min) but there was no correlation between them. Ventricular captures could be occasionally found (Fig. 3). The captures were slightly narrower than the QRS complexes during tachycardia. The mare's condition worsened, and she collapsed and died in the evening.

At necropsy, there was special emphasis on systemic circulatory disturbances, cardiac lesions, and pulmonary edema. Extensive subcutaneous edema was present. The abdominal and thoracic cavities contained a considerable amount of straw-coloured fluid with a few gelatinous fibrin clots.
The pericardial sac contained about 2 litres of yellowish serous fluid. The heart was grossly enlarged and rounded (Fig. 4). Both ventricular cavities were noticeably dilated. When the ventricular septum was cut parallel to the posterior border of the heart and tissue slices of about 5 mm thick were examined, a large grayish white patchy lesion (5×25 mm) lay high in the septal wall. Microscopically, the patchy lesion was extensive myocardial fibrosis located in the vicinity of the proximal part of the left bundle branch (Fig. 5). Partially the fibrotic lesion was in contact with the branch. A small number of degenerative and necrotic muscle fibers and scattered mononuclear cells were seen within and near the fibrotic lesion. However, Purkinje fibers of the left bundle branch were intact. Other minute fibrotic lesions (less than 2×2 mm) were found in the other parts of both the ventricular septum and the left ventricle with similar histological features. Vascular changes were observed throughout the heart.

Intramural coronary arteries and arterioles revealed intimal thickening and narrowing of the vascular lumen due to edematous swelling and/or proliferation of collagenous fiber (Fig. 6). Qualitatively and quantitatively the vascular lesions were marked in the ventricular septum, especially within and around the fibrotic lesion in the vicinity of the left bundle branch.

The lungs, which were edematous and congested, were heavy and brownish to grayish, and when cut the surface exuded a large amount of frothy fluid. Histological findings showed extensive congestion of pulmonary vessels and severe pulmonary edema with serous effusion in the bronchioles, alveolar ducts, and alveoli. Numerous heart failure cells were found in the alveolar spaces. The liver was slightly enlarged and very firm, and showed a nutmeg appearance on the cut surface. Evidence of chronic passive congestion was found on histological examination.
In the present case, VT lasted for more than 6 months and treatment with ubidecarenone, propranolol, or quinidine failed to effect a cure. Although one of the causes of ventricular arrhythmia is electrolyte imbalance [7], the VT in this case was not associated with electrolyte imbalances. Furthermore, such underlying conditions as gastrointestinal diseases [2, 11, 15, 17], systemic or regional infectious disease [2, 5, 6, 10] which may be involved in the genesis of VT in horses were not observed. The VT in the present case may have been associated with a primary heart lesion.

Electrocardiographically, the QRS duration in the present case was within or slightly above normal, indicating almost normal conduction time through the ventricle. The shapes of the QRS complexes in this case were not significantly different from those observed in horses with normal sinus rhythm, indicating that the ectopic pacemaker or reentry pathway of the VT may be located in the relatively higher portion of the ventricular septum. The extensive fibrotic lesion, which was regarded as an ischemic change due to arteriosclerotic narrowing of the vascular lumen of intramural coronary arteries, was located in the vicinity of the proximal part of the left bundle branch.

According to Bigger et al. [1], one of the general factors contributing to ventricular arrhythmias seen in myocardial ischemia is the geometry of the musculature involved. The important geometric factors include the size of the ischemic area and the interconnection of cell types in and around the ischemic zone. It has been suggested that following acute myocardial infarction surviving Purkinje cells in or near the infarcted area play an important role in the genesis of ventricular arrhythmias [8, 9, 14]. Thus, ventricular arrhythmias seem to result from electrical stimulation by the injury currents originating from specialized myocardial fibers in or near the area of the myocardial lesion.

Cohen et al. [4] reported VT arising from an ectopic pacemaker in the left bundle branch. This produced waves of depolarization that also arrived later in the right bundle branch and right ventricle than normal His conduction, but only a slight widening of the QRS complex was
produced because the impulse traveled mainly through the specialized conduction system. Furthermore, VT with QRS complexes having a duration and contour similar to the present case were described in a 3-year-old colt [16] in which a focal fibrosis in the upper portion of the ventricular septum was considered to play an important role as an ectopic focus [12].

Wellens and Lie [20] have demonstrated, using programmed electrical stimulation of the heart, that a reentry mechanism was the most likely basis for VT. They suggested that the site of reentry could be in the main bundle branches, Purkinje fibers with or without adjacent myocardium, infarcted or fibrotic tissue, or combinations of these.

The electrocardiological and cardiopathological findings with the fibrotic lesion in the vicinity of the proximal part of the left bundle branch strongly support the possibility that the ectopic pacemaker or reentry pathway of the VT was located in the left bundle branch in this case.

REFERENCES