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CASE REPORT

Cardiac trauma and third degree AV block in a dog following a road accident

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ABSTRACT

A nine and a half year old dog was referred to the Department of Clinical Veterinary Medicine, University of Cambridge for investigation of bradycardia after being hit by a car. Electrocardiography revealed a third degree atrioventricular (AV) block. The dog died during a syncopal attack. Post-mortem examination revealed an atrial septal tear with haemorrhage and myocardial degeneration affecting the AV bundle.

INTRODUCTION

Although various cardiac injuries and electrocardiographic abnormalities secondary to trauma have been described in dogs (Liedtke and others 1974, Harpster and others 1974, Alexander and others 1975, Madewell and others 1977, Macintire and Snider 1984) there are no reports in the literature of third degree heart block secondary to cardiac trauma in which histological examinations of the heart were performed. This report describes a case of third degree atrioventricular (AV) block and the subsequent post-mortem findings in a dog struck by a car.

CASE HISTORY

A nine and a half year old neutered male labrador-cross was referred to the Department of Clinical Veterinary Medicine, University of Cambridge (DCVMUC) for investigation of marked bradycardia. The dog had been well and active until three days previously, when it had been struck heavily on the flank by a car. The dog vomited one hour later and again the following night, producing food with some streaks of fresh blood. During the day, the animal was tachypnoeic and dyspnoeic with breathing which was described by the owner as "crackly".

The same evening the dog suffered two episodes of syncope with panting and apparent loss of consciousness lasting two minutes.

On hospitalisation with the referring veterinary surgeon the animal was noted to be very weak with a pulse rate of 30 to 40 beats per minute. No further episodes of syncope were observed. Antibiotic and steroid treatment was given and a lateral chest radiograph taken. This showed a mild cranial pneumomediastinum outlining the brachiocephalic trunk, with a generalised increase in interstitial lung pattern most obvious in the caudodorsal lung fields. A radio-opaque line was visible 1 cm ventral to the spine extending from the level of the seventh to the tenth thoracic vertebrae, outlining a region of increased radiodensity within which was a gas-filled, circular structure of 2 cm diameter.

On arrival at DCVMUC the dog collapsed and lost consciousness. Panting, limb spasms and urination were observed and a pulse rate of 32 per minute with good volume was maintained throughout the attack. Mucous membranes were pale and cyanotic with a capillary refill time of four seconds. The rectal temperature was 37.1ºC. Clinical examination

was otherwise unremarkable. Oxygen was administered and the dog regained consciousness.

An electrocardiogram (ECG) performed immediately showed third degree AV block with an atrial rate of.140 beats per minute and a regular idioventricular rhythm of 30 beats per minute (fig 1). The predominant rescuing pacemaker was considered to be ventricular as most QRS complexes were wide and bizarre, although occasional narrow complexes suggested an additional junctional pacemaker. Intravenous administration of 1 ml atropine sulphate (600µg/ml, C-Vet Ltd) produced no change in the ECG.

Jugular blood samples were taken and a normal saline intravenous drip set up. The samples revealed normal blood urea, creatinine, glucose, sodium, potassium and plasma protein levels. Alkaline phosphatase was increased to 854 iu/l (normal range, 3 to142 iu/l), alanine aminotransferase was 288 iu/l (normal, 21 to 59 iu/l) and aspartate aminotransferase was 247 iu/l (normal, 20 to 32 iu/l). Red blood cell parameters were within normal limits and there was a stress leukogram.

The dog was taken to the radiography department but suffered another syncopal attack before any radiographs had been taken. Oxygen was administered but the dog suffered respiratory and cardiac arrest. Cardiopulmonary resuscitation was not attempted.

POST-MORTEM EXAMINATION.

Externally there were no grossly visible signs of trauma. There was haemorrhage into the subcutaneous tissues over the area of the right thoracic wall. The thorax showed some retropleural haemorrhage on the right side, corresponding with the area of subcutaneous haemorrhage. There was haemorrhage into the cranial and caudal mediastinum although the thoracic cavity itself was free of blood. The abdomen contained approximately one litre of fibrinous fluid. Retroperitoneal haemorrhage was present in the mesenteric root and at the caudal end of the mesoduodenum. Both lungs showed multiple blood-filled bullae ranging from 5 mm to 60 mm in length. Some areas of lung showed haemorrhage which did not form bullae. The epicardium had areas of haemorrhage up to 10 mm in diameter in the interventricular region near the atrioventricular junction. The right atrium had mottled areas of haemorrhage on the epicardial surface. A tear approximately 6-8 mm in length, with attached fibrin, was present in the interatrial septum dorsocaudally to the right atrioventricular septal cusp, adjacent to an area of haemorrhage up to 10 mm diameter. The tear allowed a small degree of communication between left and right atria. There was a partial rupture, 2-3 cm in length, in the greater curvature of the gastric wall, with the mucosal surface intact but the serosal surface split and haemorrhagic. Serosal haemorrhage and tearing were present at the attachment of mesoduodenum to the caudal duodenum. The liver appeared congested and had a coating of fibrin over its capsule. Apart from some adjacent retroperitoneal haemorrhage the kidneys appeared normal. The brain appeared grossly normal.

HISTOLOGICAL EXAMINATIONS

The rupture of the serosal surface of the duodenum extended through the outer muscular tunic and into part of the inner muscular tunic. A section of affected stomach showed rupture of the wall from serosa through the muscular tunics to the level of the submucosa. Some localised submucosal haemorrhage was present in the region of the tear. Sections of the haemorrhagic lung bullae revealed a central blood-filled cavity surrounded by a pseudocapsule of compressed lung tissue. Blood was present in surrounding alveoli and the bronchioles contained some blood and proteinaceous fluid. Some alveolar walls had ruptured, with formation of small emphysematous foci.

Sections of heart taken from the interventricular septum and atrial septum confirmed the presence of marked haemorrhage, inflammation and degeneration of atrial myocardium dorsal to the AV bundle. Fibroblast proliferation and myocyte atrophy were present and the inflammatory infiltrate was mixed, with neutrophils as the predominant component. Fibrin deposition and oedema were prominent throughout the area of haemorrhage. There was haemorrhage into epicardial fat over the interventricular septum on the left side. The right atrium had multifocal areas of haemorrhage within the myocardium, with acute inflammation. There was an area of inflammation and haemorrhage just dorsal to the AV bundle (Fig. 2) associated with the atrial tear which, near the proximal end at the AV node, extended into the conduction tissue itself. At this point the conduction fibres were surrounded by a fibroblastic reaction with small amounts of haemorrhage and a mixed inflammatory cell infiltrate (Fig. 3). No lesions were found in the brain.

DISCUSSION

Motor vehicle accidents are a common cause of traumatic injury to dogs, responsible for 53 per cent of trauma accessions in one survey (Kolata and others 1974). Mortality from motor vehicle accidents was estimated at 12 per cent. Injuries typically involved multiple regions of the body with the thorax and abdomen being involved in 94 per cent of cases (Kolata 1980).

In humans, the incidence of traumatic myocarditis after blunt chest injury is between 10 per cent and 75 per cent (Alexander and others,1975). Ventricular premature contractions (VPCs) and ST segment abnormalities are commonly observed in these circumstances, although ventricular tachycardia and fibrillation, cardiac standstill, second & third degree AV block, AV dissociation, atrial tachycardia, atrial flutter and fibrillation, and right and left bundle branch blocks have all been reported (Leidtke and DeMuth.1973). Only one other clinical case of third degree AV block after blunt trauma has been reported in a dog (Abbott and King 1993) and in that animal a post-mortem was not carried out. Other post traumatic ECG abnormalities which have been more frequently described in dogs are ST depression or elevation, ventricular tachycardia, VPCs, sinus tachycardia and atrial fibrillation (Alexander

and others 1975, Madewell and others 1977, Macintire and Snider 1984, Murtaugh and Ross 1988).

In people, post-traumatic AV-block is often self-resolving (Brennan and others 1979) unless there is accompanying cardiac perforation (Paulin and Rubin 1956). Transient, sometimes lethal, immediate third degree AV block in a canine experimental model of trauma was described by Leidtke and others (1974). It has been suggested that complete AV block may be common after blunt thoracic trauma but is often not recognised since it is usually early and transient (Brennan and others1979). However, the case described by Abbott and King (1993) remained in third degree AV block five days after the incident. In the case reported here, the arrhythmia appeared to persist for three days before the dog died. Other causes of complete AV block are described by Tilley (1992). Severe hyperkalaemia due to muscle crush injury was considered as a possible cause in this case but was ruled out on finding normal blood electrolyte levels. Histological examinations of the dog's heart revealed acute inflammation within the conduction tissue and very close to the AV bundle, indicating that the trauma was the cause of this animal's third degree block.

Atrial septal tears following cardiac trauma have been reported in man but not in dogs (Cohn and Braunwald 1984). In the canine experimental trauma model (Liedtke and others 1974), none of the dogs suffered septal or free wall ruptures although many had myocardial contusions. However, these animals were struck on the sternum and not on the flank. Other types of myocardial rupture have been described in dogs. Cautley and Pasfield (1936) described a dog injured in a car accident which suffered haemothorax and transverse rupture of the heart. Harpster and others (1974) described rupture of the left ventricular anterior papillary muscle, with associated myocardial necrosis and multiple endocardial tears of the left atrium. Leidtke and DeMuth (1973) described 353 cases of myocardial rupture in man secondary to non-penetrating trauma. These were discovered post-mortem and a cardiac lesion was suspected clinically in only one case. Post traumatic cardiac injury may arise from compression of the heart between the ribs, and the sudden acceleration of the chest may slap the heart against the chest wall or sternum (Alexander and others1975). Ischaemic myocardial damage may also arise after a traumatic incident as a result of shock or neurogenic effects (Macintire and Snider 1984). This case suffered no broken ribs and several authors have noted that cardiac injury is often greater in the abscence of rib fractures (Abbott and King 1993, Liedke and DeMuth 1973). They suggest these fractures help to dissipate the energy of impact.

In contrast to a previous report (Abbott and King, 1993) this dog appeared to suffer "effort syncope" (Beckett and others 1978). There are two probable reasons for the severity of clinical signs and rapid death in this case. Firstly, the idioventricular pacemaker was predominantly ventricular in origin and was therefore slower and inherently more unstable than the predominantly junctional pacemaker described by Abbott and King (1993).

Secondly, there was a small atrial tear which could have allowed some left to right shunting of blood and a further decrease in cardiac output. During the syncopal episode observed, the dog retained a regular pulse showing no evidence of asystole or ventricular arrhythmias.

Pneumomediastinum was noted on the referring veterinary surgeon's radiograph. This is often seen in thoracic trauma in dogs. The generalised increase in interstitial lung pattern was typical of pulmonary contusion and this was probably the cause of the reported "crackly" respiration. The blood-filled bullae found at post-mortem may have been haematomas originally or may have started as air-filled pneumatocoeles (traumatic cysts) . These are often associated with severe blunt chest trauma (Suter and Lord 1984). The fact that at least some of these bullae represented a transformation from pulmonary pneumatocoeles is suggested by the gas filled structure ventral to the spine seen on the radiograph.

This case shows that severe internal injuries may be sustained in a car accident with little external evidence of trauma. The development of cardiac arrhythmias and myocardial damage are potentially fatal sequelae of such incidents, and the onset of arrhythmias may be delayed 1 to 48 hours after the incident (Macintire and Snider 1984, Murtaugh and Ross 1988). To the authors' knowledge, this is the first report of post-traumatic third degree AV block in the dog with associated post-mortem and histological findings.

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Figure legends

Fig 1.   Electrocardiogram performed immediately after collapse. Lead II (continuous trip); 25 mm/sec, 1 cm/mV showing third degree atrioventricular block with ventricular rescuing pacemaker

Fig. 2.   Atrial myocardium dorsal to the atrioventricular bundle.   Prominent acute inflammation and haemorrhage can be seen. Masson's trichrome, x 49.

Fig. 3.   There is inflammation affecting the AV bundle (centre). Masson's trichrome, x 49.