Short Communication

Management of Gastric Dilatation/Volvulus and its Cardiac Complication in a Saint Bernard

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Abstract

A five-year-old Saint Bernard was presented at the Small Animal Hospital, Chulalongkorn University with clinical signs of acute abdominal distention and severe pain. The gastric dilatation and volvulus (GDV) was diagnosed and was confirmed at surgery. The volvulus appeared in both stomach and spleen in which both organs were partially damaged. The dog developed cardiac arrhythmia the next day after surgery starting with accelerated idioventricular rhythm. Multiple types of arrhythmias were seen including ventricular tachycardia with R on T phenomenon and paroxysmal supraventricular tachycardia. The antiarrhythmic drugs, amiodarone and carvedilol were given to convert to normal sinus rhythm and drugs were withdrawn a week later without recurrent arrhythmia. The dog was recovered completely without any medication after 3 weeks of hospitalization.

Keywords : Cardiac arrhythmias, gastric dilatation volvulus, amiodarone, carvedilol

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บทคัดย่อ

ภาวะแทรกซ้อนหัวใจเต้นไม่เป็นจังหวะในสุนัขพันธุ์ Saint Bernard ที่มีกระเพาะขยายใหญ่และบิด

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สุนัขพันธุ์เซนต์เบอร์นาร์ด อายุ 5 ปี เข้ามารักษาในโรงพยาบาลสัตว์เล็ก คณะสัตวแพทยศาสตร์ จุฬาลงกรณ์มหาวิทยาลัย ด้วยอาการท้องขยายใหญ่ และเจ็บปวด ได้ทำการวินิจฉัยว่าสุนัขมีอาการกระเพาะขยายใหญ่และบิด จากการผ่าตัดพบกระเพาะอาหารและม้ามบิดและมีการคั่งเลือดของอวัยวะทั้งสอง สุนัขเริ่มมีอาการหัวใจเต้นไม่เป็นจังหวะ (arrhythmia) ในวันต่อมาโดยเริ่มเกิด accerelated idioventricular rhythm พบการเกิดการเต้นไม่เป็นจังหวะหลายรูปแบบรวมทั้งการเกิด ventricular tachycardia มี R ทับอยู่บน T wave และพบ paroxysmal supraventricular tachycardia สุนัขได้รับยา amiodarone และ carvedilol เพื่อให้การเต้นของหัวใจกลับมาเป็น normal sinus rhythm ได้หยุดยาอีก 1 สัปดาห์ถัดมาโดยไม่พบการเต้นไม่เป็นจังหวะกลับมาอีก สุนัขหายเป็นปกติใน 3 สัปดาห์ภายหลังเข้ารับการรักษาในโรงพยาบาล

คำสำคัญ : หัวใจเต้นไม่เป็นจังหวะ, กระเพาะขยายใหญ่และบิด, amiodarone, carvedilol

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Introduction

Gastric dilatation volvulus (GDV) is a condition of multiple severe life-threatening disorders that occurred mostly in large and giant breed dogs (Glickman et al., 2000[a]). The previous study showed 43% of mortality rate (Muir, 1982[a]). However, later reports were between 15%-24.3% (Brockman et al., 1995; Brourman et al., 1996; Glickman et al., 1998). The mortality rate is high in dogs that lack prompt surgical correction and intensive critical care treatment. The guidelines for GDV syndrome were established in many hospitals in order to reduce mortality rate. A previous report showed that 40% of GDV dogs developed cardiac arrhythmias and may not related to the outcome (Brockman et al., 1995). The arrhythmia occurred most frequently between 12 and 36 hours after the onset of GDV and was usually ventricular in origin. However, malignant ventricular arrhythmia may lead to high risk of fatality and sudden death after surgical correction. The purposes of this report were to demonstrate the management of GDV and show multiple types of arrhythmias in a dog that developed after a surgical correction of GDV. The effectiveness of antiarrhythmic drugs was also discussed.

History and Physical Examination

A five-years-old, female Saint Bernard dog weighing 49 kilogram was presented at the Small Animal Hospital, Faculty of Veterinary Science, Chulalongkorn University at 22:00 h with a history of acute abdominal pain for approximately one and a half hour. The last meal was fed to the dog at 18:00 h and a light exercise was proceeded at 20:00 h On physical examination, it was revealed that the stomach was markedly distended with gas. An intravenous catheterization (16 gauge needle) was performed on a cephalic vein. Blood samples were collected for complete blood count, blood chemistries and blood gas analysis. Electrocardiographic measurement (ECG) was also carried out. Fluid therapy using a balanced electrolyte solution was infused at the rate of 3.0 ml/kg/min. Dexamethazone was administered at the dose of 1 mg/kg intravenously. A gastric decompression was achieved by a combination of orogastric intubation and needle gastrocentesis. An orogastric tube, 1 cm in diameter, was inserted into the stomach with some

difficulties and 4 liters of gastric content and gas were drained.

Surgical Correction

Abdominal radiographs were performed after gastric decompression and revealed gastric and splenic displacement. An additional catheterization through the dorsal pedal artery was accomplished for direct blood pressure measurement. The endotracheal intubation was made following standard general anesthesia using a combination of 2 μg/kg of fentanyl citrate (Fentanyl®, Janssen-Cilag, Belgium), 0.2 mg/kg of diazepam (Diapine®, Atlantic Lab. Co.Ltd., Thailand) and 4 mg/kg of propofol (Lipuro®, B-Braun, Germany), intravenously as an induction and maintained with isoflurane (Minrad Inc, USA) in 100 % oxygen. Acetate Ringer was maintained at a rate of 20 ml/kg/hr. The packed cell volume (PCV), total solid (TS), mucus membrane, capillary refilling time (CRT), heart rate, blood pressure and pulse quality were monitored during surgery. The synthetic colloid, Hetastarch (Haemaccel®, Aventis, limited, Germany) was administered to maintain plasma volume and oncotic pressure. The systolic blood pressure was maintained above 100 mmHg.

After aseptic preparation, a cranioventral abdominal midline incision was performed. The orogastric tube was intubated for further gastric decompression. The stomach and spleen were rotated back to their normal positions. Gastropexy was performed by suturing to fix pylorus with right peritoneum. A stomach was lavaged with warm water via an orogastric tube. After repositioning the spleen and stomach, the greater curvature of the stomach was still congested. The active bleeding vessels at the caudal pole of the stomach next to the spleen were carefully ligated. Approximately 1,500 ml of blood was removed from the abdominal cavity during surgery. The gastric and splenic blood flow were evaluated by colorization and none of the tissue was excised. The abdomen was lavaged with warm 0.9% NaCl and closed with a routine manner. Bandage was placed to protect the incision site. After surgery, 0.5 mg/kg of morphine sulfate (FDA, Thailand) and 1 μg/kg of fentanyl citrate were administered to relief pain.

Post-operative management

The dog was admitted at the intensive care unit after surgery and was monitored for continuous ECG, blood gas, blood pressure and urine output. The CBC, blood chemistries and blood gas were checked intermittently. A combination of 1 μg/kg of fentanyl citrate and 0.5 mg/kg of morphine sulfate were given intramuscularly during the first 3 days. The synthetic colloids and balanced electrolyte solutions, potassium chloride, glucose, and amino acid were given in the first week. Oxygen was supplemented periodically. The antibacterial agents, imipenam (Tienam®, Merck, USA) and 10 mg/kg metronidazone (B-Braun, Germany), were given intravenously on the first week. Other supportive medications were provided as needed. Small amount of food and drinking water were initially given on day 3 after surgery. The mental status was subjectively good. The results of complete blood count and plasma chemistry profiles were shown in Table 1 and 2. The dog was anemic. Therefore, a blood transfusion was carried out on day 3. Although the reticulocyte count seemed to be increased (6.3%), the PCV was still low. Erythropoietin (Epokine®, CJ corp., Korea) was injected at a dose of 100 units/kg on day 10 and 12 and the PCV was raised up to 40%.

Renal function was evaluated on the first day which showed mild azotemia with creatinine of 2.5 mg%. Urinalysis performed the next day after surgery showed the red blood cell in the urine along with clump of white blood cells. The specific gravity was 1.014 with the urine pH of 7. The urinalysis analyzed on day 5 and 6, the pH was 6. The urine started to be clear and fewer cells appeared in the urine. The glomerular filtration rate measured by endogenous creatinine clearance on day 7 after surgery was 141 litres/day (1.90 ml/kg/min). The plasma creatinine became normal on day 2. Urine culture performed on day 13 showed no bacterial growth.

Table 1 The complete blood count in GDV dog

	Day												Ref.
Complete blood count	**0**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**18**	**23**	**range**
RBC (x 10^6 / ul)	6.3	3.6	2.9	4	3.5	3.8	3.3	4.13	3.6	4.1	4.95	5.6	**4.9-8.2**
Hb (g/dl)	16.8	9.5	7	10	9	10	9	8.9	10	10	12.3	14	**10-20.6**
PCV (%)	49	28	23	30	27	30	27	29	31	34	40	40	**35-58**
MCV (fl)	75	75	77	75	76	74	75	72	78	75	75	72.3	**64-76**
MCH (pg)	26	26	26	25	26	26	26	21	26	26	25	25.5	**21-26**
MCHC (g/dl)	34	34	33	34	36	34	35	29	33	34	34	35.2	**31-36**
PLT (x10^3/ul)	216	52	31	45	54	154	190	145	136	159	261	332	**>200**
WBC (/ul)	10200	9800	15900	17600	18000	17600	12000	24300	19200	13700	11200	7800	**4500-17000**
Neutrophil (%)	80	84	84	83		84	68	87	86	85	72	70	
Band Cell (%)	5	6	6	5		2	6	1	2	1	4	0	
Eosinophil (%)	1	1	0	0		4	1	1	3	4	3	9	
Basophil (%)	0	0	0	0		0	0	0	0	0	0	0	
Lymphocyte (%)	6	5	5	6		6	20	8	6	6	16	19	
Monocyte (%)	8	4	5	6		4	5	3	3	4	5	2	

Table 2 Blood chemistries in GDV dog

Blood	Day												Ref.
Chemistries	**1**	**2**	**3**	**4**	**5**	**7**	**9**	**10**	**11**	**14**	**18**	**23**	**range**
Na^+ (mEq/L)	147	156	158	166	158	146	143	153	142	144	145	146	**138-153**
K^+ (mEq/L)	4.8	4.3	4	2.7	3	3.2	3.1	4.2	3.7	4.4	4.1	4.7	**3.9-5.7**
Ca^{2+} (mEq/L)	9	9.1	9.4		8.7	8.4	8	6.4		9.6	9.8	10.1	**7.6-11.6**
Pi (mg/dl)	6.7	3.4	3.6	3.3	5.2	2.8	3.2	4.5		7.6	3.8	4.9	**2.8-6.2**
AST (unit)							65	65			28	29	**15-80**
ALT (unit)	33	1200	1165	1003	666	342	194	173	153	101	67	46	**15-90**
Alk Phos (unit)	101	293	470	453	888	758	509	519	480	467	300	218	**0-140**
Choles (mg/dl)			146				195	182			241	243	
TG (mg/dl)							74	97			71	105	
Total bil (mg/dl)							0.4	0.3					
glucose (mg/dl)		99				108	89	81				89	**59-121**
BUN (mg/dl)	29	36	33	32	31	18	11	17	15	20	23	20	**7.0-26.6**
Cr (mg/dl)	2.4	1.9	1.4	1	0.8	1.1	1	0.8	0.9	1.1	0.9	1.1	**0.68-2.04**
TP (g/dl)	6.9	2.7	3.4	5.3	4.6	4.3	4.1	4.5	4.2	5.2	6.2	6.6	**5.5-8.0**
Alb (g/dl)	3.8	2	2	2.8	2.5	2.3	2	2.7	2.2	2.6	2.7	2.9	**2.3-4.1**

The acid base status was evaluated intermittently. Metabolic alkalosis was seen only on day 1 (pH = 7.473) with a HCO_3^- of 21.3 mmol/L. Blood pH was recorded continually which had the values of 7.382, 7.479, 7.393, 7.404 and 7.426 on day 2, 3, 4, 6 and 9, respectively.

Cardiac complications

When admission, the ECG was evaluated and showed sinus tachycardia. Sinus tachycardia still persisted until 12:00 h of day 2 after surgery. (Fig 1). However, after 14:00 h, the dog developed the ventricular arrhythmia known as accelerated idioventricular rhythm (Fig 2). The ectopic ventricular complexes were emerged with the rate closely couple the sinus rate. No medication was given at that time. At night, the ventricular tachycardia developed and persisted until next morning (Fig 3, 4 and 5). On day 4 after surgery at 15:45 h, the ventricular excitation was prominent and the superimposition of the QRS wave on preceding T wave of ECG (R on T phenomenon) was detected (Fig 6). During tachycardia, the animal showed signs of weakness and exercise intolerance. The bolus of 50 mg of lidocaine hydrochloride (Xylocaine®, Astra Zeneca, Sweden) was injected intravenously at a dose of 1 mg/kg twice and maintained at a rate of 40 μg/kg/min to convert the ECG to sinus complexes. The ventricular ectopic rhythm still persisted periodically and amiodarone (Cordarone®, Sanofi, France) was given both intravenously at a dose of 1 mg/kg and maintained orally (4 mg/kg twice daily) in order to replace lidocaine hydrochloride. The drug successfully converted the ECG to normal sinus rhythm (Fig 7 and 8). The heart rate was controlled but premature atrial complexes were detected periodically from day 3 to day 11. The paroxysmal supraventricular tachycardia was detected starting from day 12 (Fig 9 and 10). Carvedilol (Caraten®, Berlin Pharmaceutical Industry Co. Ltd., Bangkok, Thailand), a beta adrenergic blocking agent, was added orally at a dosage of 12.5 mg once daily starting from day 12 while amiodarone was tapered off. After day 13, heart rate decreased (Fig 11) and became normal sinus respiratory pattern with a rate between 65-90 beats per minute (bpm) afterward. Carvedilol was also tapered off starting from day 16. Normal respiratory sinus arrhythmia was maintained until the last day of recording (day 20) (Fig 12). The dog was recovered completely and was discharged from the hospital at day 22.

The GDV dogs showed a higher incidence in male more than female but it was not significant (58% vs. 42%) (Glickman et al; 2000[a]). The mean age was 7.5 years. The risk of GDV was associated with increasing age (Glickman et al., 2000[a]; 2000[b]). The giant breed dog was predisposed for GDV including Great Dane, Weimaraner, Saint Bernard, Golden Setter, Irish Setter, German Shepherd and standard poodle. The disease appears to have a familial predisposition. Thoracic-depth/width ratio appears to predispose dogs to GDV (Broome and Walsh, 2003). Other predisposing factors are dietary factors include particle size, frequency of feeding, speed of eating, aerophagia and an elevated feed bowl. However, in giant breed dogs, the risk of GDV was the highest for dogs fed a large volume of food once daily (Raghavan et al., 2004). In this case, the dog was fed once daily with a high quantity of food and elevated feed bowl. Rather than eating behavior, GDV is possibly caused by temperament and stressful events (Glickman et al., 1997). However, no significance was found between occurrence of GDV and atmospheric temperature and humidity (Dennler et al., 2005). The treatment of GDV requires electrolyte and vascular stabilization, therapy of shock, careful anesthetic monitoring, gastric decompression, surgical correction and postoperative care.

The short-term mortality rate was between 15-24 % (Broome and Walsh, 2003, Beck et al., 2006). The risk factors which were associated with death prior to suture removal were clinical signs shown longer than 6 hours after the onset, combined splenectomy and partial gastrectomy, hypotension, peritonitis, sepsis and disseminated intravascular coagulation (DIC). Moreover, age, gastrectomy and DIC were risk factors of hypotension (Beck et al., 2006). In this dog, both spleen

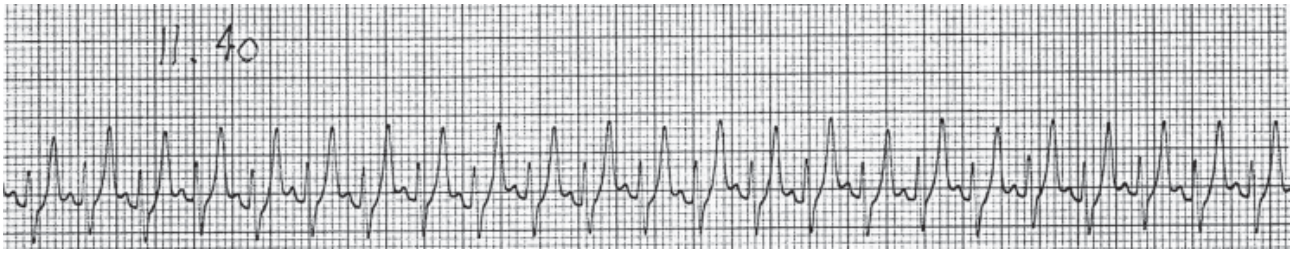

Figure 1 ECG recorded on day 2 after surgical correction at 11:40 h showed sinus tachycardia with HR of 200 bpm

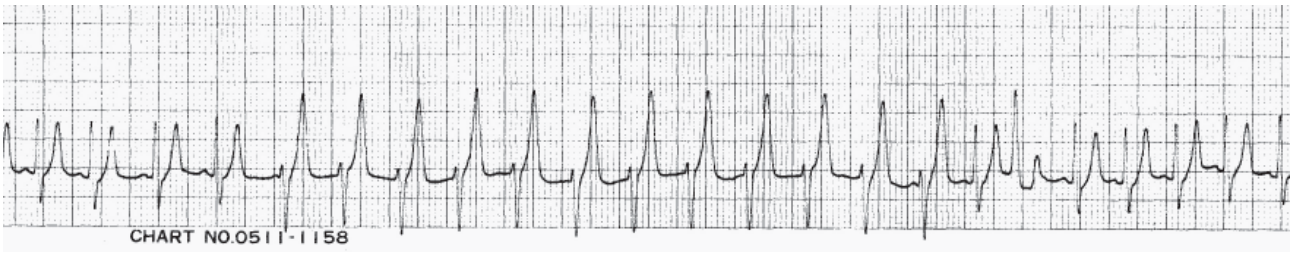

Figure 2 ECG recorded on day 2 after surgical correction at 14:00 h showed accelerated idioventricular rhythm (AVR) with HR of 150 bpm

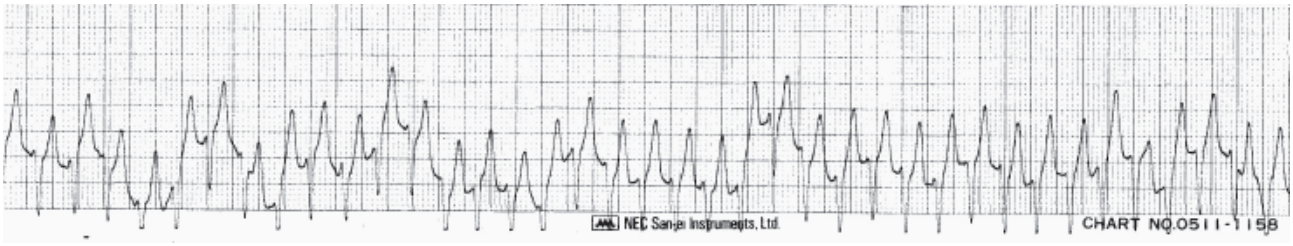

Figure 3 ECG recorded on day 2 after surgical correction at 23:00 h showed ventricular tachycardia with HR of 250 bpm

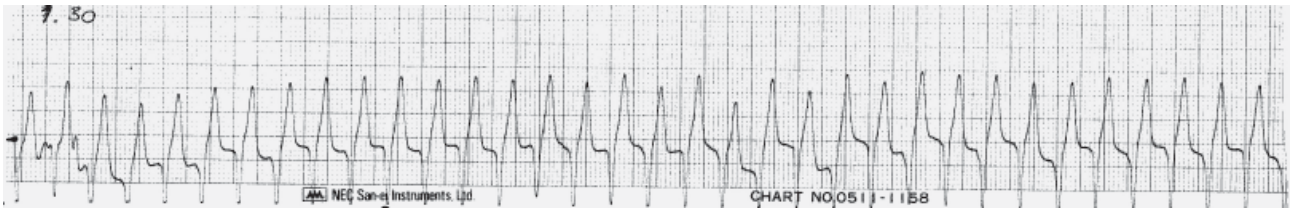

Figure 4 ECG recorded on day 3 after surgical correction at 1:30 h showed ventricular tachycardia with HR of 190 bpm

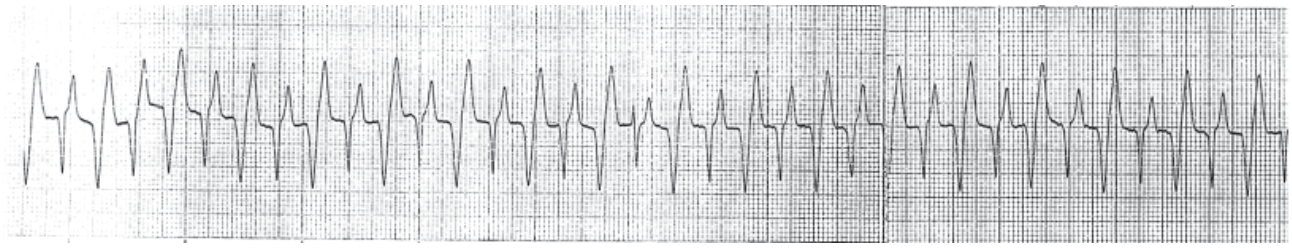

Figure 5 ECG recorded on day 3 after surgical correction at 10:00 h showed ventricular tachycardia with HR of 190 bpm and persisted throughout the day.

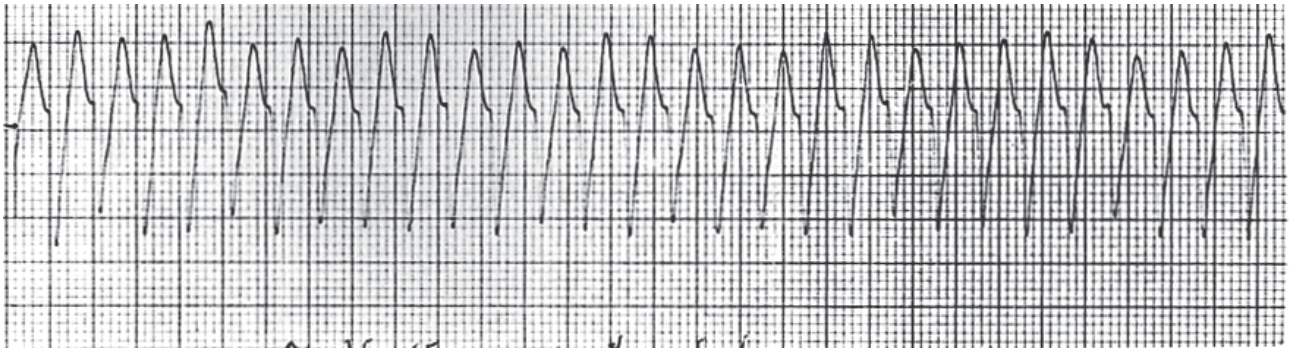

Figure 6 ECG recorded on day 4 after surgical correction at 15:45 h showed ventricular tachycardia with R on T phenomenon. Heart rate was 350 bpm. Lidocaine was injected to control the arrhythmia. Amiodarone was chosen to maintain thereafter.

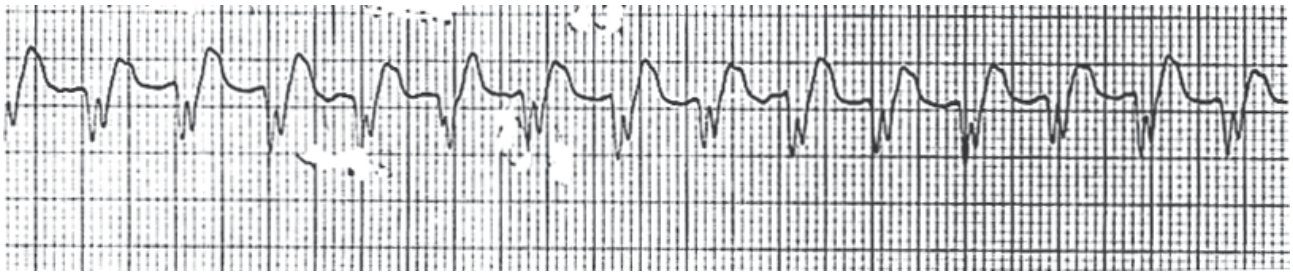

Figure 7 ECG recorded on day 4 right after anti-arrhythmic drug was introduced. Normal sinus rhythm was restored with heart rate of 150 bpm

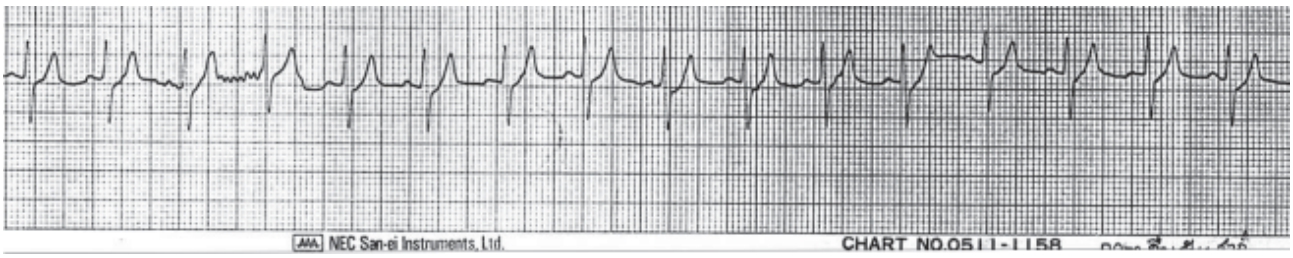

Figure 8 ECG recorded 6 hour after anti-arrhythmic drug was introduced. Normal sinus rhythm was sustained with heart rate varied between 110 and 150 bpm.

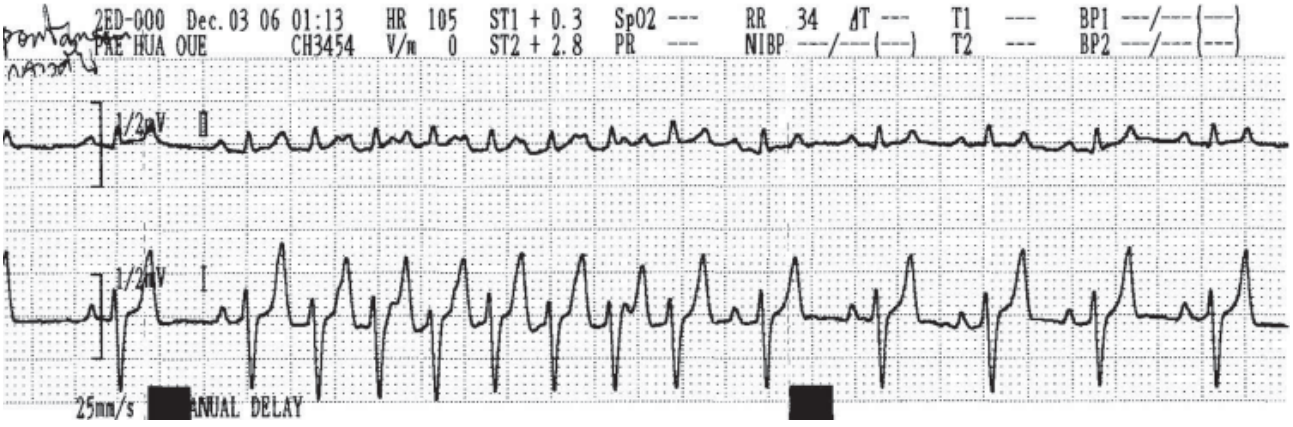

Figure 9 ECG recorded on day 12 after surgical correction at 1:13 h showed paroxysmal supraventricular tachycardia. Amiodarone was still given orally starting from day 4.

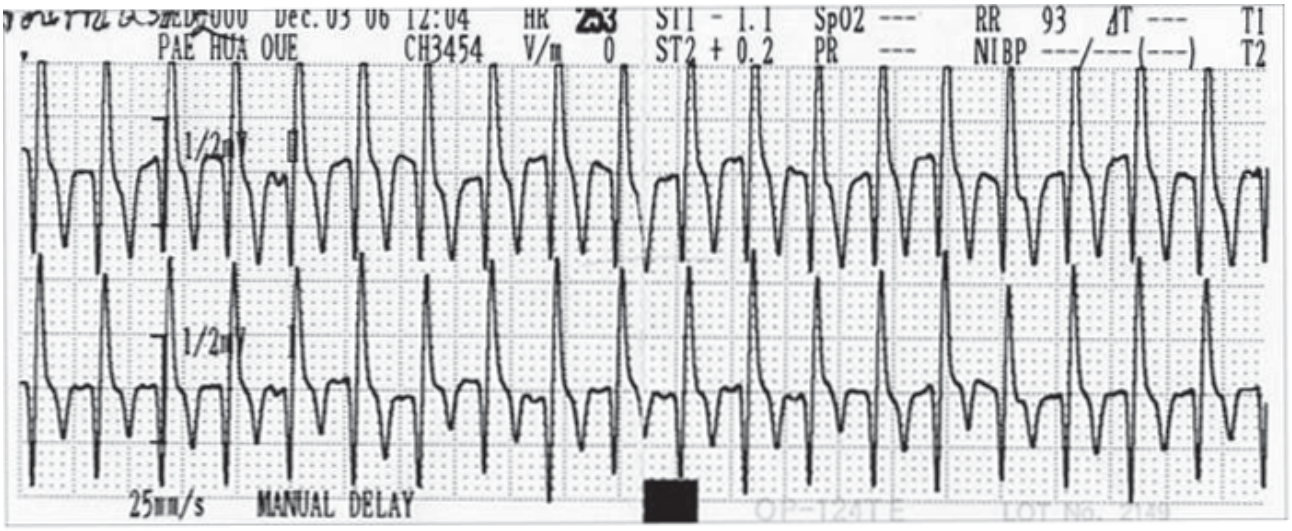

Figure 10 ECG recorded on day 12 after surgical correction at 12:04 h showed train of supraventricular tachycardia persisted from 30 minutes to 3 hours. Heart rate was approximately 250 bpm. Carvedilol was added on day 13 while amiodarone was tapered off to control arrhythmia.

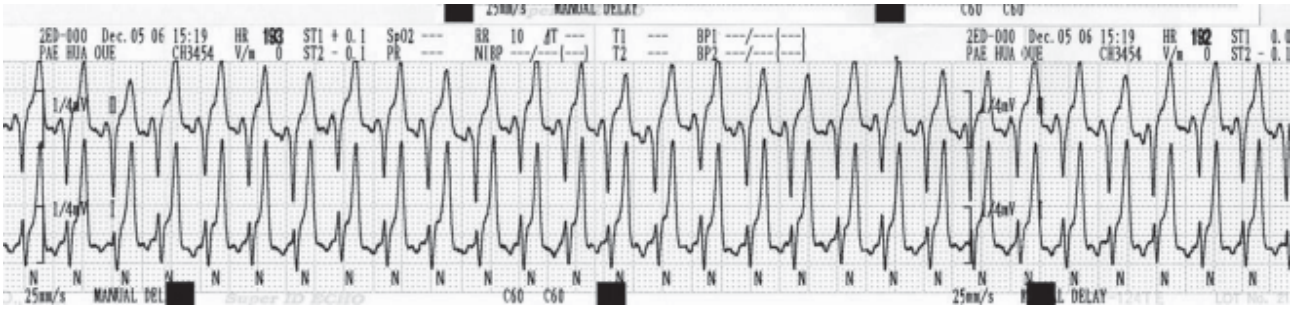

Figure 11 ECG recorded on day 14 after surgical correction at 15:19 h showed persistent supraventricular tachycardia still occurred but the rate was reduced to 190 bpm. On day 16, heart rate declined without paroxysmal arrhythmia

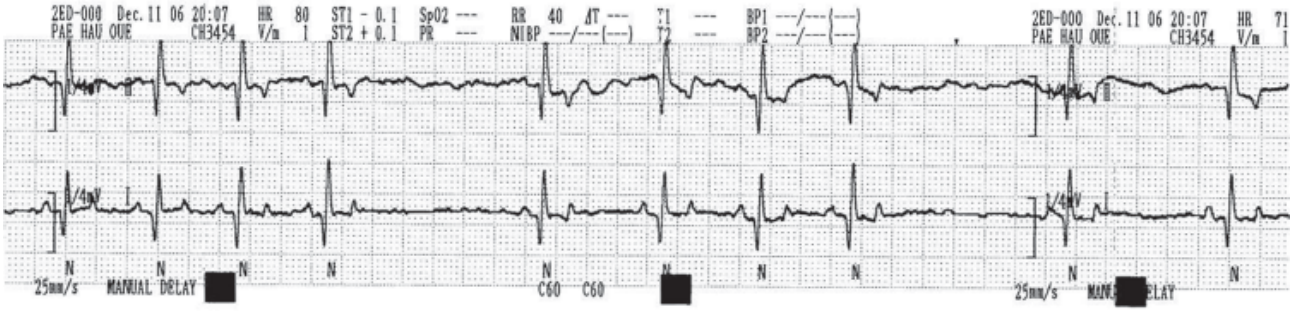

Figure 12 ECG recorded on day 20 after surgical correction at 20:07 h showed respiratory sinus arrhythmia with heart rate of 70 bpm.

and stomach were still intact. Moreover, the dog was presented to the hospital about 4 hours after the onset of clinical signs. Peritonitis and sepsis were not present in this dog. Although the blood pressure was within a normal range, the hypotension may be encountered during some types of cardiac arrhythmia. A study in 295 cases of GDV showed that tube gastropexy, a simple clinical guideline procedure for GDV, was performed on 86.5% of these cases. Among GDV dogs that survived, the mean time between the onset of clinical signs and admission was 5.5 ± 9.6 hr (Brockman et al., 1995). The mean time between admission to the ER room and induction of anesthesia was 2.7 ± 3.2 hr while mean anesthetic time was 2.3 ± 0.7 hr which was similar to those of this dog.

The goal for GDV management is the immediate correction for adequate tissue perfusion. The significant reduction in capillary blood flow in the stomach wall measured by laser Doppler flowmetry was found (Monnet et al., 2006). There was a report of the greater blood flow to gastric mucosa and submucosa compared to the muscularis and serosa (Delaney and Grim, 1964). Some vascular, splenic and hepatic abnormalities were found. No evidence of delayed gastric emptying was found in GDV dogs (van Sluijs and van den Brom, 1989).

The large volume of lactated Ringer's solution (LRS) was administered before surgery to prevent shock in this case. Hypertonic saline - dextran (HSD) solution was reported to be used as effective as LRS (Schertel et al., 1997). The cardiopulmonary function was rapidly improved in HSD group. Previous report also showed the beneficial effect of 7% NaCl in 6% dextran (Allen et al., 1991). After laparotomy a large amount of bloody fluid by approximately 1,500 ml was lost and HSD solution was added to the infusate. Blood pressure was maintained stable throughout the operation period.

Surgical correction was successfully performed in this dog. The spleen was enlarged with no evidence of necrotic tissue and need not to be resected. Gastrectomy was not required in this case due to improved color of the stomach wall. The gastropexy was performed in order to prevent a recurrence of GDV. The rate of recurrence was 10.6% in 85 cases that were followed for up to three years (Glickman et al., 1998).

Many non-surviving dogs had massive gastric necrosis and other complications such as gastric perforation. Many measurements were developed to predict the gastric necrosis such as an increase in plasma lactate concentration (de Papp et al., 1999) or abnormal fibrin degradation product concentration, activated partial thromboplastin time and antithrombin III activity (Millis et al., 1993) since gastric necrosis was associated with significant higher mortality. However, by performing partial gastrectomy or splenectomy, mortality rate was significantly increased and even more increased with combined procedures (Brourman et al., 1996). The post-operative complications were found such as aspiration pneumonia, DIC, septic peritonitis, circulatory collapse and cardiopulmonary arrest. Peritonitis, DIC, sepsis and arrhythmia rather than partial gastrectomy were risk factors for death (Beck et al., 2006). However, evidence of bacterial translocation from the stomach could not be demonstrated in GDV dogs (Winkler et al., 2003). Thrombocytopenia was found in many dogs which may be related to a high tendency of hemorrhage thereafter. It may be an indicator of DIC but it cannot be used to predict gastric necrosis or mortality rate (Millis et al., 1993).

Although metabolic acidosis and hypokalemia were commonly found in GDV dogs (Muir, 1982[b]), they did not occur in this case. Previous study showed the controversial results of normal values of pH (Wingfield et al., 1982) and alkalosis (Muir et al., 1982[b]; Kagan, 1983). Alkalosis may probably be a result of excessive loss of gastric hydrochloric acid due to repeated vomiting and volume depletion. The hypokalemia in this dog may be a result of vomiting, decreased food intake and also the mechanism of compensatory metabolic alkalosis. Alkalosis may enhance H^+ conservation of the kidney in

exchange of K^+ excretion. Also intracellular extrusion of H^+ to buffer alkalemia occurs in exhange with K^+ uptake intracellularly.

The preoperative arrhythmias were found in some cases and were associated with higher mortality (Brourman et al., 1996). In this case, the arrhythmia was first examined 2 days after operation. A study in 295 GDV dogs demonstrated that cardiac arrhythmias appeared in 40% of surviving dogs compared with 38% of dogs that did not survive from GDV (Brockman et al., 1995). Cardiac arrhythmias were diagnosed at a similar rate of 42% among GDV dogs (Muir, 1982[a]). The arrhythmias include intermittent premature ventricular conduction, sustained ventricular tachycardia, paroxysmal ventricular tachycardia, ventricular tachycardia, second degree AV block and atrial fibrillation (Muir and Lipowitz, 1978). Thus, cardiac arrhythmia may not be a cause of death. The ventricular arrhythmia was not related to type of fluid therapy since they could be detected in dogs receiving either LRS or HSD (Schertel et al., 1997).

A previous study showed no differences of time lapse between onset of signs and admission, time between admission and induction of anesthesia, duration of anesthesia. Moreover, no clear relationship was found between development of cardiac arrhythmias and increase risk of sudden cardiac death in GDV dogs (Brockman et al., 1995). The precise mechanisms for initiation and maintenance of arrhythmia remained unidentified. At least 3 factors may be involved. Firstly, the decreased cardiac output and coronary blood flow may lead to ischemia. In experimental induced GDV dogs, decrease in cardiac output (64%), mean aortic pressure (48%), left ventricular end diastolic pressure (LVEDP) (68%) and ventricular dp/dtmax during 180 minute of gastric dilatation volvulus were demonstrated (Orton and Muir, 1983[a]). The decreased LVEDP implied the decrease in ventricular preload consistent with a sudden reduction in venous return. The LVEDP increased suddenly after gastric decompression supporting the obstruction of the great vein. Moreover, increased heart rate and decreased coronary blood flow during diastole caused further cardiogenic shock.

Secondly, the low molecular weight proteins from hypoxic organs may be released and caused a reduction of myocardial performances. Pancreatic ischemia results in the formation of myocardial depression factor which has been postulated to contribute to gastric dilatation volvulus syndrome (Wingfield et al., 1976). However, the isovolumetric indices and humeral cardioactive substance bioassay could not be detected in experimental induced GDV dogs (Orton and Muir, 1983[b]). Myocardial ischemia and hypoxia are probably causes of cardiac arrhythmias in this case.

Thirdly, changes in autonomic neural activity can cause arrhythmia in GDV dogs. Gastric dilatation and inflammation are associated with both sympathetic and parasympathetic overactivity (Bukley et al., 1969; Bowen and Dinesh, 1977). Sympathetic stimulation augments the development of ventricular tachycardia by increasing excitability, vulnerability and altering refractoriness of myocardial tissues.

Although K^+ and pH were slightly altered in this case, they may not be significant factor for arrhythmia and can be corrected within a few days. The serious arrhythmia can cause cardiac damage leading to death. The cardiac troponins (cTnI and cTnT) increased within 48 hours and was presented in 93% of dogs with GDV (Burgener et al., 2006). A study of 85 dogs with GDV showed a different concentration of troponin among groups based on the severity of ECG abnormalities (Schober et al., 2002). The peak serum concentration of cTnI was observed between 48 and 72 hours after surgery for a correction of GDV and may be a result of arrhythmia. The GDV dogs that died had significantly higher serum cTnI and cTnT concentrations than dogs that survived (Schober et al., 2002). Increase in cardiac troponin may be an indicator of acute myocardial damage in dogs with GDV since myocardial cell injury was confirmed at necropsy in 4 dogs with high serum cTn concentrations (Schober et al., 2002).

The ventricular arrhythmia known as accelerated idioventricular rhythm first seen on the next day after surgical correction was considered benign. However, ventricular tachycardia developing on day 3 showed an appearance of R on T phenomenon which is predisposed to ventricular fibrillation causing hemodynamic instability (Buranakarl et al., 2001). The dog was immediately injected with bolus infusion of lidocaine hydrochloride which can initially be given at 4 mg/kg. It can be maintained with constant infusion at a rate of 25-50 μg/kg/min. The ventricular arrhythmia was abolished for a short term duration (30 minutes). Although the 90% successful treatment with lidocaine hydrochloride or procainamide hydrochloride was reported earlier (Muir and Lipowitz, 1978), ventricular arrhythmia recurred after an initiation of lidocaine infusion. Since lidocaine can cause dysrhythmia and toxicity of grand mal seizure (Pfeifer et al., 1976), another antiarrhythmic drug, amiodarone, was selected to control dysrhythmia.

Amiodarone, a class III antiarrhythymic drug, was selected to control dysrhythmia in this case. Although it is classified in class III, it also possesses the class I, II and IV activities (Awaji et al., 1995). Recently, amiodarone was also proved to have direct scavenging activity against free radicals (Ide et al., 1999). The drug is lipophilic with primary clearance by liver. An intravenous injection form may be considered in an emergency situation. The dose should be titrated starting from 1 mg/kg up to 5 mg/kg of body weight, then followed by oral route at the dose of 5-10 mg/kg daily to maintain plasma concentration. The intravenous dose of 6.67 mg/kg still did not prolong QT interval, but decreased the mean arterial blood pressure (Awaji et al., 1995) and depressed cardiac contractility (Landymore et al., 1984). However, at the dose higher than 25 mg/kg, amiodarone may produce significant side effects (Bicer et al., 2002).

On the third day after onset of amiodarone treatment, carvedilol was added to prevent long-term side effects of amiodarone. Carvedilol is a nonspecific beta blocker which possesses an alpha-1 adrenergic blocking activity and free radical scavenging activity. It was demonstrated that the lipid peroxidation associated with reperfusion injury is important in the pathogenesis and high mortality of GDV. Tissue malondialdehyde concentration was significantly greater in duodenum, jejunum, colon, liver and pancreas (Badylak et al., 1990). Moreover, this novel class of beta blockers also has antiarrhythmic and anti-proliferative activities. The half life of this drug is about 54 minutes with a large volume of distribution due to lipid solubility (Sawangkoon et al., 2000). The therapeutic dose range in dogs is still not established but at the dose between 0.1 to 0.5 mg/kg of body weight have beta blocking activity without any side effects on hemodynamics (Abbott et al., 2005). The recommended dose in dogs with mitral regurgitation is between 0.2-0.4 mg/kg (Uechi et al., 2002) and the duration of beta blocking action is between 12-24 hours at a high dose (Gordon et al., 2006).

In conclusion, cardiac arrhythmia may occur in most of the case suffering from GDV. A careful monitoring at the time after surgical correction is crucial. Aggressive antiarrhythmic therapy will decrease the fatality rate. Amiodarone and carvedilol were selected to control arrhythmia in this case.

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