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Severe Intestinal Distension in a Dog with Primary Hypoparathyroidism

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Abstract : An 1-year-old, female, mixed-breed dog weighing 17 kg was referred for abrupt collapse. She had remarkable hypocalcemia and hyperphosphatemia, and survey radiographs revealed a severe gas-filled intestine. Treatment with serial injections of calcium gluconate was initiated promptly and most of the gastrointestinal distension disappeared after 4 h. However, the clinical signs were not resolved completely. The serum intact parathyroid hormone concentration was not elevated in the context of hypocalcemia, which suggested primary hypoparathyroidism. The clinical signs and laboratory abnormalities in the patient were resolved completely 3 days after administration of calcium gluconate and calcitriol. This case describes the unique presentation of severe gastrointestinal distension in a dog diagnosed with primary hypoparathyroidism.

Key words **:** canine, hypocalcemia, ileus, intestinal obstruction, primary hypoparathyroidism.

Introduction

Among the intestinal motility disorders of small animals, intestinal pseudo-obstruction is an uncommon clinical syndrome, which is characterized by a clinical presentation that resembles bowel obstruction, i.e., failure of intestinal motility associated with pain and intestinal distention, but the absence of mechanical obstruction (9). In humans, intestinal pseudo-obstruction is divided into two unrelated forms, namely, acute and chronic (6,7). However, intestinal pseudo-obstruction in small animals has only been described as the chronic, and there is still a lack of information regarding the clear pathophysiology of intestinal pseudo-obstruction in dogs compared with humans.

Primary hypoparathyroidism is an uncommon endocrine disease, which occurs as a consequence of an absolute or relative deficiency of parathyroid hormone (PTH). PTH deficiency causes hypocalcemia, which produces neuromuscular signs (4). The definitive test for primary hypoparathyroidism is an evaluation of the serum PTH concentration and the concurrent ionized calcium concentration (4). To the best of our knowledge, however, there is a lack of information available on severe intestinal distension in dogs with hypoparathyroidism. Herein, we describe the development and management of severe intestinal distension mimicking intestinal pseudo-obstruction caused by hypocalcemia in a dog with hypoparathyroidism.

Case

An 1-year-old, intact female mixed-breed dog weighing 17 kg was referred for abrupt collapse and ataxia, which had developed suddenly in the morning. There was no previous history of parturition and the owner reported that the dog was depressed and anorectic for at least 3 days before presentation. At presentation, the dog was already unconscious and recumbent with generalized tetanus. The rectal temperature was 40°C, the heart rate was 220/min, the indirect systolic blood pressure, determined using the Doppler method, was 115 mmHg, and the dog was panting. An electrocardiogram identified mild Q-T interval prolongation.

Blood analyses and survey radiography were performed. The electrolytes and biochemistry tests revealed severe hypocalcemia and hyperphosphatemia (Table 1), whereas the complete blood counts detected no remarkable findings. The concentrations of serum intact parathyroid hormone (iPTH) and magnesium were measured to differentiate the diagnosis of the cause of hypocalcemia. The serum magnesium level was slightly higher than the normal range (0.63 mmol/L [1.52 mg/dL], reference interval [RI] 0.28-0.49 mmol/L [0.69-1.18 mg/dL]), but the serum iPTH concentration was lower than the RI (1.33 pmol/L [12.5 pg/mL], RI: 2.01-13.01 pmol/L [18.9-122.6 pg/mL]). On the basis of these findings, the dog was diagnosed to primary hypoparathyrodism, while other possible causes of hypocalcemia and hyperphosphatemia were ruled out based on the history and diagnostic findings. Unexpectedly, the abdominal radiography disclosed severe gastrointestinal distension, which suggested digestive tract obstruction since the intestinal tract was filled throughout with gas (Fig 1A).

The dog was hospitalized in the intensive care unit. Given the severe hypocalcemia, calcium supplementation was initiated. Calcium gluconate (10% Calcium gluconate, Daihan Pharm Corp. Ltd, Seoul, Korea) was injected slowly at 9.3

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Table 1. Selected data from the blood analyses

Variable	Reference interval	Days					
		0		1		2	3
		AM	PM	AM	PM		
Sodium (mmol/L)	141-152	151	151	152	ND	152	147
Potassium (mmol/L)	4.37-5.35	4.8	4.8	5.7	ND	3.7	3.8
Total calcium (mmol/L)	2.25-2.83	1.15	1.58	1.43	1.68	2.50	2.83
Ionized calcium (mmol/L)	1.12-1.42	0.69	ND	0.75	ND	0.82	1.16
Phosphorus (mmol/L)	0.84-2.00	5.17	2.68	1.78	1.58	1.97	0.81
Total protein (g/L)	54-71	69	56	55	ND	74	67
Albumin (g/L)	26-33	38	32	26	ND	32	31
Glucose (mmol/L)	3.61-6.55	2.61	7.66	5.83	5.49	7.88	6.22

ND, not done

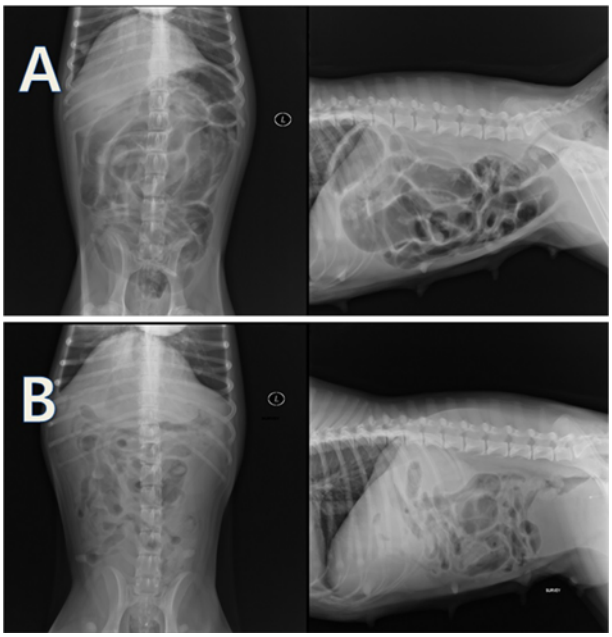

Fig 1. Abdominal radiographs obtained from a dog with primary hypoparathyroidism. A: Ventrodorsal and lateral radiographs before the initiation of treatment with calcium gluconate. Note the generalized gastrointestinal dilation. B: Ventrodorsal and lateral radiographs at 4 h after treatment with calcium gluconate.

mg/kg (IV) over 20 min, followed by a constant rate infusion of 3.5 mg/kg/h. Three hours after the initiation of treatment (day 0), the serum total calcium concentration was elevated but still lower than the RI (Table 1). The patient's tetanic signs were alleviated considerably after the initial calcium treatment. However, ataxia still remained, although continuous calcium infusion was performed. The gastrointestinal gas passed out of the anus continually and most of the gastrointestinal distention disappeared in the abdominal radiography (Fig 1B). A barium contrast study was performed to definitively rule out mechanical ileus, and abdominal radiography showed no obstructions. Oral administration of calcium gluconate (16 mg/kg, tid) and calcitriol (0.03 μg/kg, sid; Bonky soft capsule, Yuyu Pharma Inc., Seoul, Korea) was initiated the following day (day 1). On day 2, the serum total calcium level was elevated relative to the RI, and the ionized calcium concentration had also increased, although it was still lower than the RI (Table 1). The dog was able to stand up, but it still exhibited a gait disturbance.

Three days after presentation (day 3), the ionized calcium concentration was normalized and the ataxia had disappeared entirely. Based on a consideration of hypercalcemia, the doses of calcium gluconate and calcitriol were reduced. The dog was discharged from the hospital and treatment with calcitriol and calcium gluconate was continued at home. The 1-month follow-up examination revealed no abnormal findings, so oral administration with calcitriol alone was prescribed. Five months after initial presentation, the dog had maintained its normal activity, with no further episodes of collapse or other clinical signs associated with hypocalcemia or hypercalcemia.

Discussion

In the present case, the radiographs detected severe intestinal distention at presentation, which was highly suggestive of mechanical ileus. Fortunately, the intestinal distention was resolved rapidly after the initial administration of calcium gluconate, which suggested that the distension was a consequence of hypocalcemia. Gastrointestinal motility depends on the contractile activity of smooth muscle cells in the tunica muscularis (1). Calcium ions are also essential for the contraction of smooth muscle. Unlike striated muscle, where calcium is derived solely from internal stores, intestinal smooth muscle cells also depend on extracellular calcium stores for excitation-to-contraction coupling (10). Therefore, it is possible that hypocalcemia can cause diminished gastrointestinal smooth muscle contraction, thereby leading to gastrointestinal dysmotility (3).

Recently, a retrospective study of 17 dogs with primary hypoparathyrodism described the perceived abdominal pain in four dogs (8). In contrast to the present case, it was sug-

gested that the abdominal pain might have resulted from increased gastrointestinal motility or spasm, or increased nervous tissue excitability. However, hypocalcemia has been implicated in the pathophysiology of intestinal dysmotility in humans and other animals, although little or no information is available on dogs. Human studies show that electrolyte imbalances, including hypocalcemia and hypokalemia, are associated with the occurrence of acute colonic pseudo-obstruction (5). Hypocalcemia is also believed to be a predisposing factor in ruminal tympany and abomasal displacement in post-parturient dairy cattle (2). Experimentally induced hypocalcemia in cows was also accompanied by a decrease in intestinal mechanical activity (3). For these reasons, hypocalcemia may reduce the motility of intestinal smooth muscles and could have been responsible for severe intestinal distension in the present case. However, further studies will be necessary to clarify the underlying mechanism.

In the present case, the severe intestinal distension was similar to the characteristics of intestinal pseudo-obstruction syndrome. In human studies, many etiologies and associations that underlie intestinal pseudo-obstruction have been described for the acute and chronic forms. Post-surgical conditions are the most common causes of acute intestinal pseudo-obstruction, while also common are medical conditions such as infections, cardiac diseases, neurological problems, respiratory diseases, and electrolyte imbalance (6). In the chronic form, visceral myopathy and neuropathy are the two main causes of primary intestinal pseudo-obstruction (also called idiopathic intestinal pseudo-obstruction). The secondary causes of chronic intestinal pseudo-obstruction include progressive systemic sclerosis, immune-mediated diseases, endocrine diseases, and pharmacological factors (7). To the best of our knowledge, however, all previously reported cases of intestinal pseudo-obstruction in small animals were of the chronic form and were associated with visceral myopathy. In the present case, it appears that the gastrointestinal symptoms were induced only by hypocalcemia without visceral myopathy, although an intestinal biopsy was not performed. It is uncertain whether the intestinal pseudo-obstruction was acute or chronic, because the actual period that the patient had a hypocalcemic status was unknown. However, the etiology of the pseudo-obstruction, where hypocalcemia resulted in gastrointestinal dilation, is likely to be distinct from previous reports, regardless of their chronicity.

In conclusion, persistent hypocalcemia caused by primary hypoparathyroidism might result in reduced gastrointestinal motility in dogs. However, gastrointestinal dilation has not been considered in the differential lists in the majority of reports about primary hypoparathyroidism. Clinically relevant decreases in intestinal motility caused by hypocalcemia are probably rare, but possibly under-recognized. Thus, clinicians who treat dogs with gastrointestinal dilation should be aware of this condition.

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일차성 부갑상샘기능저하증에 이환된 개에서 장확장증 발생 증례

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요 약 : 1세, 암컷, 17 kg의 잡종견이 갑작스러운 허탈증상으로 본원에 내원하였다. 혈액 검사 결과에서 심각한 저칼슘혈증을 보였으며, 방사선 사진 영상에서는 장 폐색을 의심하게 하는 가스로 가득 찬 장확장증 소견을 나타내었다. 칼슘 글루코네이트의 정맥 내 투여를 시작한 후 4시간째에 장 확장 소견은 경감되었다. 혈청 부갑상샘호르몬 농도는 저칼슘혈증에 비해 정상 범위 아래로 측정되었으며, 이를 기초로 하여 환견은 일차성 부갑상샘기능저하증으로 진단되었다. 칼슘 글로코네이트와 칼시트리올의 투여 3일 후, 환견의 임상증상과 전해질 불균형은 개선되었다. 본 보에서는 일차성 부갑상샘기능저하증으로 진단된 개에서 일반적으로 관찰되지 않는 증상인 심각한 위장관 확장증의 발생 예를 보고한다.

주요어 : 개, 저칼슘혈증, 장폐색, 일차성 부갑상샘기능저하증