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Short Communication

PANCREATIC ELASTASE-1 ELISA FOR THE DIAGNOSIS OF EXOCRINE PANCREATIC INSUFFICIENCY AND ITS MANAGEMENT WITH ENZYME REPLACEMENT THERAPY: A CASE REPORT

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SUMMARY

Exocrine pancreatic insufficiency (EPI) is a mal-absorptive syndrome caused by insufficient secretion of digestive enzymes from pancreatic acini. The most common causes of EPI in dogs and cats are pancreatic acinar atrophy and chronic pancreatitis. The primary cause of EPI in canines is pancreatic acinar atrophy (PAA), an immune-mediated condition that results in the progressive loss of pancreatic acinar cells responsible for enzyme secretion. A four-year old intact male German shepherd dog (GSD) was presented with the complaints of progressive weight loss, lethargy, polyphagia, abnormal stools with soft consistency and flatulence. The complete blood count, hepatic and renal biochemical tests, along with abdominal ultrasound and fecal elastase testing, were performed. Based on the clinical signs, physical examination, hematology, biochemical finding and fecal elastase levels, the condition was diagnosed as Exocrine Pancreatic Insufficiency (EPI). Pancreatic Enzyme Replacement Therapy (PERT) was initiated, accompanied by cobalamin supplementation. Within two weeks, there was a noticeable reduction in clinical signs, and over the subsequent eight weeks, the dog gained 6 kg of body weight, with fecal consistency returning to normal.

Keywords: Elastase, ELISA, Exocrine pancreatic insufficiency, German Shepherd

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Exocrine pancreatic insufficiency (EPI) is characterized by inadequate production of digestive enzymes by the pancreatic acini, resulting in poor digestion, mal-absorption, and mal-nutrition (Soetart *et al.*, 2019, Westermarck *et al.*, 2012).

Exocrine pancreatic insufficiency has a worldwide distribution. The most well-documented breed pre-disposition is the German Shepherd Dog (GSD), and while any breed can be affected, Rough-coated Collies, Cavalier King Charles Spaniels, Cairn Terriers, Chows, Cocker Spaniels, Eurasier dogs, and West Highland White Terriers are reported to be at increased risk of EPI (Batchelor *et al.*, 2007; Moeller *et al.*, 2002; Proschowsky *et al.*, 2007; Wiberg *et al.*, 1999). Clinical signs associated with EPI are caused by defective digestion and absorption of dietary macromolecules (protein, fat, and starch) (Pongprasobchai 2013, Owens *et al.*, 2007).

The most observed clinical signs are polyphagia, parorexia, coprophagia, soft stools, steatorrhea, increased fecal volume, borborygmus, flatulence, weight loss, seborrhea, and muscle atrophy (Westermarck *et al.*, 2012). EPI is diagnosed based on measuring decreased pancreatic secretion capacity by pancreatic function tests. (Freudiger 1971). The exocrine pancreas has a large reserve secretory capacity, and clinical signs of maldigestion do not occur until 90% of secretory capacity is lost (Dimagno *et al.*, 1973). In dogs with EPI, the hydrolase activities of the pancreas are only 1/50,000th of those of healthy dogs (Westermarck *et al.*, 1980). The diagnosis of EPI is most accurately determined through the measurement of serum trypsin-like immunoreactivity (TLI), with lower levels indicating the presence of the condition. However, in specific situations, such as in developing countries like India where financial constraints may limit access to TLI testing, an alternative diagnostic approach like patient-side ELISA for fecal pancreatic elastase 1 can be used as a supportive tool, which is also internationally accepted as a diagnostic tool in diagnosing EPI.Management of EPI typically involves oral supplementation with pancreatic enzymes to compensate for the deficient digestive enzymes, thereby promoting better nutrient absorption. Additionally, dietary adjustments-such as providing a highly digestible, low-fiber diet-can further improve therapeutic outcomes.

A four-year-old intact male German Shepherd Dog (GSD) was presented with a history of progressive weight loss, lethargy, polyphagia, abnormal defecation characterized by soft, voluminous stools (Fig. 1.1) and excessive flatulence. Vital parameters, including mucous membrane color, capillary refill time, rectal temperature, heart rate, and respiratory rate, were within normal physiological limits. Routine fecal examination was negative for endoparasitic infections. On physical examination, the patient was alert and responsive, exhibiting a body condition score indicative of emaciation to cachexia, with evidence of mild muscle wasting, brittle hair coat, mild dehydration, and signs of abdominal discomfort. Palpation of the right epigastric region elicited borborygmi.

Complete blood count, serum biochemistry, fecal

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elastase-1 assay, and abdominal ultrasonography were performed. The hematological and biochemical alterations included mild neutrophilic leucocytosis, macrocytic anemia suggestive of nutritional deficiency, hypoproteinaemia and hypophosphatemia. Ultrasonographic examination revealed hepatomegaly and a mildly enlarged pancreas with increased echogenicity (Fig. 1.2), indicative of pancreatopathy. Exocrine pancreatic insufficiency (EPI) is diagnosed primarily by measuring species-specific serum trypsin-like immunoreactivity (cTLI), which directly correlates with the functional mass of pancreatic acinar tissue. Currently, the only validated alternative to cTLI for diagnosing EPI in dogs is a canine-specific patient-side ELISA for fecal pancreatic elastase-1 (cE1; ScheBo Biotech AG, Germany). This assay, validated for canine fecal samples, interprets concentrations >20 µg/g as excluding EPI, while concentrations <20 µg/g are diagnostic of EPI in dogs exhibiting compatible clinical signs (Spillmann *et al.*, 2000).

Based on the patient's clinical history, breed predisposition, clinical examination findings, ultrasonographic abnormalities, hematobiochemical profile, and markedly reduced fecal pancreatic elastase-1 concentration (Fig. 1.3), a definitive diagnosis of Exocrine Pancreatic Insufficiency was established.

EPI is treated by Pancreatic Enzyme Replacement Therapy (PERT), nutritional management (low-residue diets with moderate fat content) and supplementation of cobalamin. Pancreatic enzymes in the form of coated capsules (Tab Pancresolve-DS [1-0-0], Vivaldis, India) were provided 20 minutes before food in the initial phase of treatment along with proton pump inhibitor, Pantoprazole (Tab Acidfre-20 mg [1-0-1], Freossi, India). The proton pump inhibitors were used only for first 14 days as gastric pH decreases the enzymatic activity of lipase and trypsin.

To control the small intestinal bacterial overgrowth, antibiotic Metronidazole (Metrogyl Suspension, JB Chemicals & Pharmaceutics, India) was used. For supplementation of vitamins and minerals, multivitamin syrup (Multistar Pet [5 mL-0-5 mL], Mankind Pharma, India) and gastrointestinal diet was added along with pre & probiotics.

Over a period of 8 weeks, digestion, the consistency

Fig.1.1. Voluminous stool due to indigestion before Pancreatic Enzyme Replacement Therapy

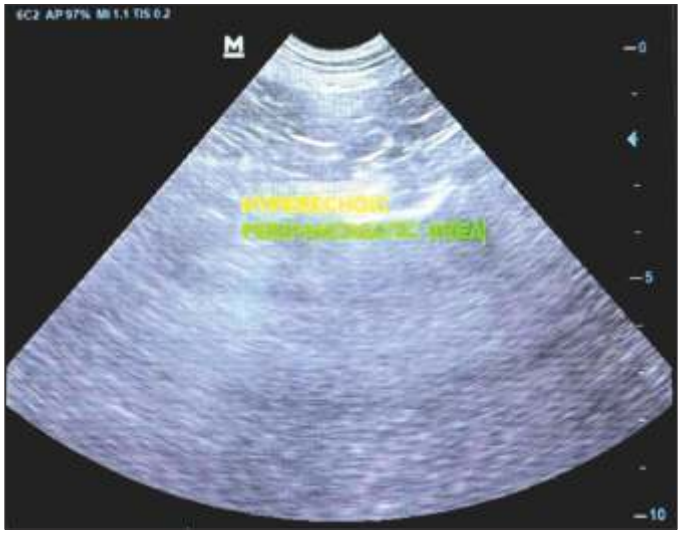

Fig.1.2. Hyperechoic Per-pancreatic region

Fig.1.3

Fig.1.4

Fig.1.5

Fig.1.3. ScheBo PE-1 Quick ELISA Kit [No line seen in "T" indicates: means no detectable pancreatic elastase-1 → suggests exocrine pancreatic insufficiency (EPI)]. Fig.1.4. ScheBo® Pancreatic Elastase-1 (PE-1) stool test Kit. Fig. 1.5. Stools after PER Therapy

of faeces and weight gain was assessed and the dog gained 6 kg of weight during this period. The owners were advised to avoid feeding any junk food products and to maintain strict monitoring of the feeding schedule. Over time, the fecal consistency improved from a 'cow dung-like' appearance to a normal, brown-coloured, segmented form (Fig 1.4).

Underlying pathologic processes that may result in clinical signs of EPI in dogs are pancreatic acinar atrophy (PAA), chronic pancreatitis and pancreatic neoplasia. PAA is reported to be by far the most common cause of severe EPI in dogs. Characteristic of PAA is a selective destruction of the digestive enzyme–producing acinar cells, which may lead to almost complete loss of secretory capacity. Chronic pancreatitis is a common cause of EPI in cats and human beings (Westermarck *et al.*, 2003). Trypsin-like immunoreactivity measures serum trypsin and trypsinogen concentrations and is considered the test of choice for EPI. Serum TLI assays are highly sensitive for the diagnosis of EPI (Williams *et al.*, 1988).

Animals with EPI do not produce sufficient trypsin, and as a result, diagnosis is made through the measurement of serum trypsin-like immunoreactivity (TLI), a test with high specificity and sensitivity. For dogs, the reference value of TLI is between 5.0 and 32.0 ng/mL and values below 2.5 ng/ mL confirm the disease (Matilda *et al.*, 2011, Westermarck *et al.*, 2012, Nelson *et al.*, 2015, Soetart *et al.*, 2019).

Gastric pH decreases the enzymatic activity of lipase and trypsin. Therefore, the administration of proton pump inhibitors such as omeprazole, one to two times a day for continuous use, is necessary (Nelson *et al.*, 2015; Matilda *et al.*, 2011). The use of antibiotics such as metronidazole aims to prevent intestinal bacterial overgrowth caused by the loss of antimicrobial factors in pancreatic juice or the increased availability of undigested substrates in the intestine (Nelson *et al.*, 2015, German 2012). Decreased or absent intrinsic factor causes hypocobalaminemia, so Vitamin B12supplementation should also be part of the treatment (Nelson *et al.*, 2015, Soetart *et al.*, 2019).

The dietary recommendation for dogs with EPI consists of diets based on hydrolysed protein with high digestibility, low fiber content, and a higher quantity of B-complex vitamins (Nelson *et al.*, 2015). Low-fat content was previously indicated, but it has been found to directly interfere with the animal's weight gain and is therefore only used as a last resort to control steatorrhea (German 2012). Lifelong adherence to enzyme supplementation and dietary management is typically necessary to maintain the dog's health and quality of life.

CONCLUSION

This case report describes the clinical presentation of exocrine pancreatic insufficiency in a German shepherddog, highlighting the diagnostic application of patient-side ELISA for fecal pancreatic elastase-1, and evaluating the treatment response and effectiveness of oral pancreatic enzyme replacement therapy.

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