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Non-communicating small intestinal duplication in a dog: a case report

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ABSTRACT: Enteric duplication is rare in dogs. Here, we report the rarest form of duplication in which two segments are parallel and share a wall for most of their lengths. A nine-year-old spayed female Yorkshire terrier was referred to the Veterinary Medical Teaching Hospital at Gyeongsang National University due to anorexia and diarrhoea. Physical examination, haematological examination, radiography, and ultrasonography were performed. On physical examination, dry, pale mucous membrane was identified. Moderate anaemia with decreased packed cell volume was detected in complete blood count. Serum urea nitrogen and creatinine levels were mildly increased. Radiographic images revealed no significant findings. On ultrasonographic examination, a multi-layered appearance of a focal small intestinal segment was identified in the left mid abdomen. Following the lesion, it was divided into two small intestinal segments. Based on imaging findings, intussusceptions or enteric duplication were suspected. To resect the abnormal small intestinal segment, enterectomy was performed. Follow-up was not performed because the patient expired during the postoperative recovery time. The histopathological diagnosis was non-communicating small intestinal duplication. Non-communicating intestinal duplication is related to embryologic abnormalities and is usually concurrent with other anomalies such as vertebral malformations and urogenital duplications. However, this case had no other anomalies associated with the malformation of the intestine.

Keywords: intestine; enteric duplication; ultrasonography malformation; surgery

Duplications of the alimentary tract are tubular or spherical structures lined by epithelium similar to intestine that are firmly attached to or share a wall of the alimentary tract (Srivastava et al. 2009). The aetiological mechanism of enteric duplication is currently unknown. There have been several theories proposed to explain the occurrence of enteric duplication (Jakowski 1977; Macpherson 1993). Enteric duplication may be accompanied by urogenital tract anomalies, other gastrointestinal tract anomalies or concurrent vertebral anomalies (Jakowski 1977; Lo et al. 2004). Clinical signs of duplication depend on location, size, compression of adjacent structures and communication with the gastrointestinal tract (Spaulding et al. 1990). Lesions may be a subclinical, incidental finding or may gradually expand producing recurrent abdominal pain, abscess formation, signs of partial intestinal obstruction, ulceration and perforation (Grosfeld et al. 1970; Bower et al. 1978).

Abdominal radiographic findings are frequently non-specific, but a soft-tissue mass may be seen within the abdomen. Ultrasound is the preferred initial diagnostic modality for the diagnosis of enteric duplication (Spaulding el al. 1990). The duplicated luminal border is hyperechoic, the muscular wall is hypoechoic and the serosal layer and adjacent fat are hyperechoic (Simonovsky 1996). Potentially purulent cellular debris and haemorrhaging in the enteric duplication produce an echoic appearance contrasting with that of the mobile luminal contents. Real-time imaging may detect peristaltic

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activity in the enteric duplication (Spaulding el al. 1990). Other abnormalities that may initially have some sonographic characteristics similar to a duplication can include obstructed bowel, intramural haematoma, intramural tumour and intestinal wall abscess (Lee et al. 1977; Moore and Carpenter 1984). Due to some similarities in appearance, a careful ultrasound and a histological evaluation are necessary to prevent a misdiagnosis. Diagnosis of enteric duplication also requires histological evaluation to differentiate aberrant tissue from a diverticulum. The tissue of the enteric duplication must be attached to the gastrointestinal tract and must include both alimentary epithelium and a central layer of smooth muscle (Espalieu et al. 1985).

Non-communicating enteric duplications in the abdomen are extremely rare (Srivastava et al. 2009; Kim et al. 2013). We here report the case of a non-communicating small intestine duplication that was described in a dog on the basis of sonographic, radiographic, gross and histopathological findings.

Case description

A nine-year-old 1.4-kg spayed female Yorkshire terrier was referred to the Veterinary Medical Teaching Hospital at Gyeongsang National University with anorexia and diarrhoea. The patient had a history of anorexia, which had started about one month previously. In the preceding week, the diarrhoea had been accompanied with melena. Mild depression and dry pale mucous membrane were identified in physical examination. A small nodule (5 × 5 mm) on the right caudal mammary gland was also detected. Laboratory evaluation, complete blood count, as well as serum chemistry and antigen detection using the parvoviral antigen kit were conducted. Moderate anaemia with decreased packed cell volume (19.4%, reference range: 35.0–55.0%) was detected in the complete blood count. An increase in BUN (21.42 mmol/l, reference range: 2.5–8.9 mmol/l) and creatinine (129 μmol/l, reference range: 22.88–106.75 μmol/l) levels but a decrease in albumin (22 g/l, reference range: 25–44 g/l) and total protein (48 g/dl, reference range: 54–82 g/dl) levels were found in serum chemistry. Parvoviral antigen results were negative. Radiographic examination did not reveal any remarkable findings. On ultrasonographic examination, a multi-layered small intestine wall was detected in the left mid abdomen. Corrugated small intestine was connected to the lesion. A moderate amount of fat was identified between the walls. Following the lesion, the segment was divided to two small intestine segments (Figure 1). Differential diagnoses included intussusception and small intestine duplication.

For gross diagnosis, exploratory laparotomy was conducted. The small intestinal lesion was markedly extended. The extended small intestine was adherent to surrounding peritoneum, the remaining uterus and the urinary bladder (Figure 2A). To treat the intestinal lesion, the extended intestinal segment was resected and anastomosis performed. Two tubular-shaped parallel lumens were lined up inside of the resected tissue. One of the lumens was considered to be the normal intestine. The other was suspected to be the duplicated intestine (Figure 2B). On gross examination, haemorrhaging was identified in the lumen of the duplicated intestine (Figure 2C). The intestinal lesion was confirmed as non-communicating intestinal duplication based on histopathological examination. Histopathologically, the cross section of the small intestine contained two intestinal lumens. One was a duplicated intestinal lumen; the other was a normal intestinal lumen. The septum between the intestinal lumens included mucosa and muscularis mucosa separated by connective tissue-like submucosa. Normal and duplicated areas were covered with normal intestinal mucosa, villi and crypts. However, some of the mucosa in the duplicated intestine was composed of squamous epithelium. Severe multifocal haemorrhage and focal ulceration were observed in squamous foci in the duplicated intestine. In addition, many chronic inflammatory cells such as lymphocytes and macrophages had infiltrated the lamina propria and submucosa of the squamous duplicated intestine (Figure 3). Postoperative follow up could not be performed because the patient expired during the postoperative recovery time.

DISCUSSION AND CONCLUSIONS

Enteric duplication is a rare developmental malformation in veterinary medicine (Radlinsky et al. 2005 ; Kook et al. 2010). Grossly, duplicated intestinal segments are usually blind sacs or cysts that share a wall with the normal intestine (Spaulding et al. 1990). Gastrointestinal tract duplications can

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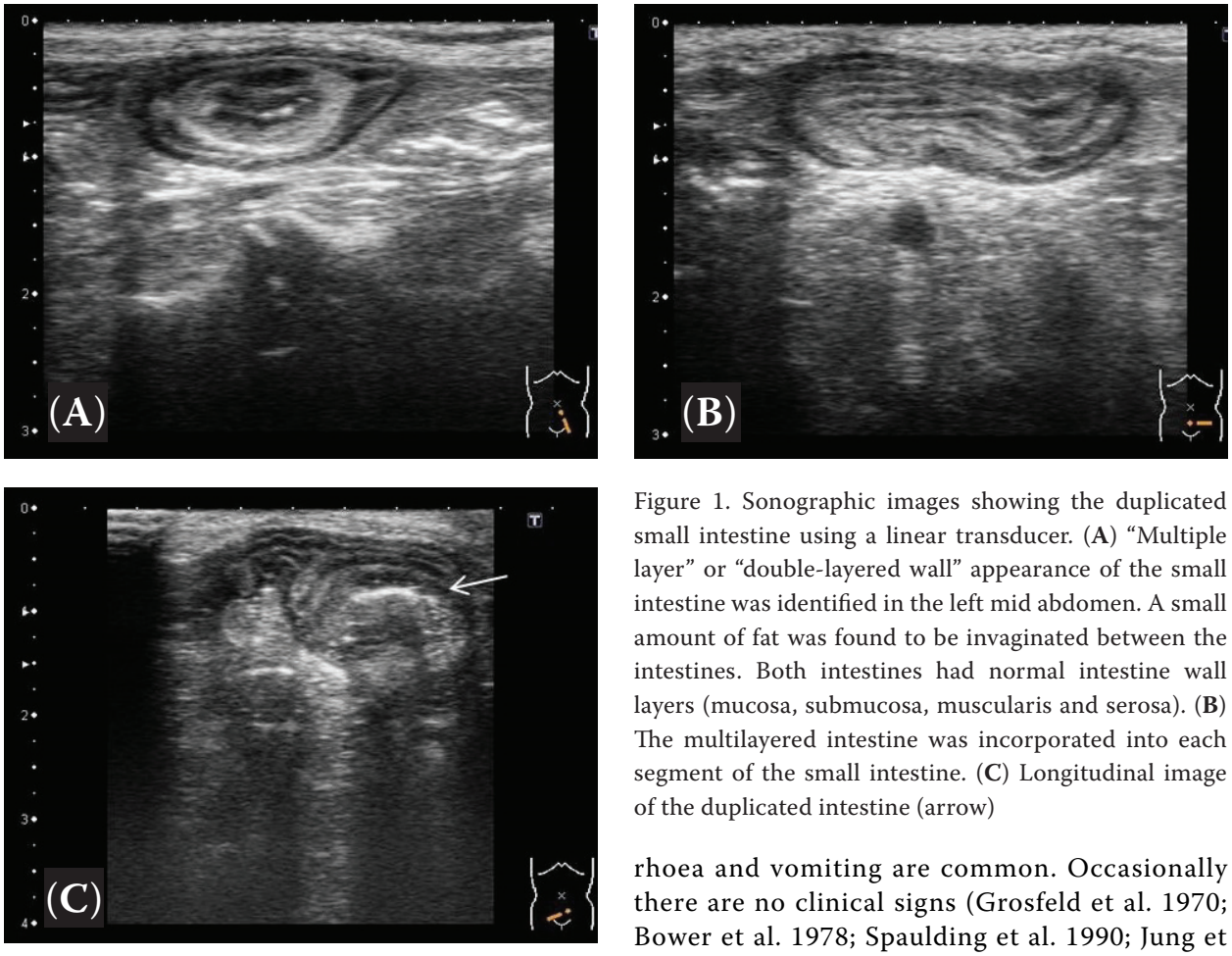

Figure 1. Sonographic images showing the duplicated small intestine using a linear transducer. (**A**) "Multiple layer" or "double-layered wall" appearance of the small intestine was identified in the left mid abdomen. A small amount of fat was found to be invaginated between the intestines. Both intestines had normal intestine wall layers (mucosa, submucosa, muscularis and serosa). (**B**) The multilayered intestine was incorporated into each segment of the small intestine. (**C**) Longitudinal image of the duplicated intestine (arrow)

be found anywhere along the alimentary tract (from the oesophagus to the anus). Enteric duplications are commonly diagnosed in young animals, up to 85% of cases by the age of two years old (Grosfeld et al. 1970; Macpherson 1993). However, a significant number of patients may not be diagnosed until adulthood. In this case, the patient was nine years old. There were no clinical signs associated with enteric duplication until one month previously. Thus, the patient might have been asymptomatic for up to nine years.

Clinical signs of enteric duplication depend on the type, location of duplication, communication with the gastrointestinal tract and compression of adjacent structures. Clinical signs usually begin in early childhood. Specifically, enteric duplication has been reported to cause anorexia, depression, abdominal pain, abdominal distension and vomiting in animals. These duplications have also been described as incidental findings in asymptomatic adults. In humans, abdominal distension, palpable abdominal mass, intermittent pain, nausea, diarrhoea and vomiting are common. Occasionally there are no clinical signs (Grosfeld et al. 1970; Bower et al. 1978; Spaulding et al. 1990; Jung et al. 2009). This patient had a history of anorexia and diarrhoea. Melena was observed several times. Anaemia and dehydration were found in laboratory examination, which were considered to be caused by several episodes of melena. There were no significant findings in physical examination.

The classification system for duplication that is used in human medicine can also be applied to enteric duplication in dogs. Partial duplication without other anomalies is classified as Type I. Complete duplication usually accompanied with other anomalies such as vertebral deformities and urogenital duplications is classified as Type II. Type I is subdivided into type IA, IB, IC, ID and IE: Type IA is spherical and non-communicating; Type IB is tubular and non-communicating; Type IC is tubular and communicating; Type ID is loop with separate blood supply; Type IE has multiple duplications (Kottra and Dodds 1971; Arthur et al. 2003). Enteric duplication can also be classified as parallel or intramesenteric based on vascular supply. In the parallel type, the artery of the duplication is separated from the artery to the normal

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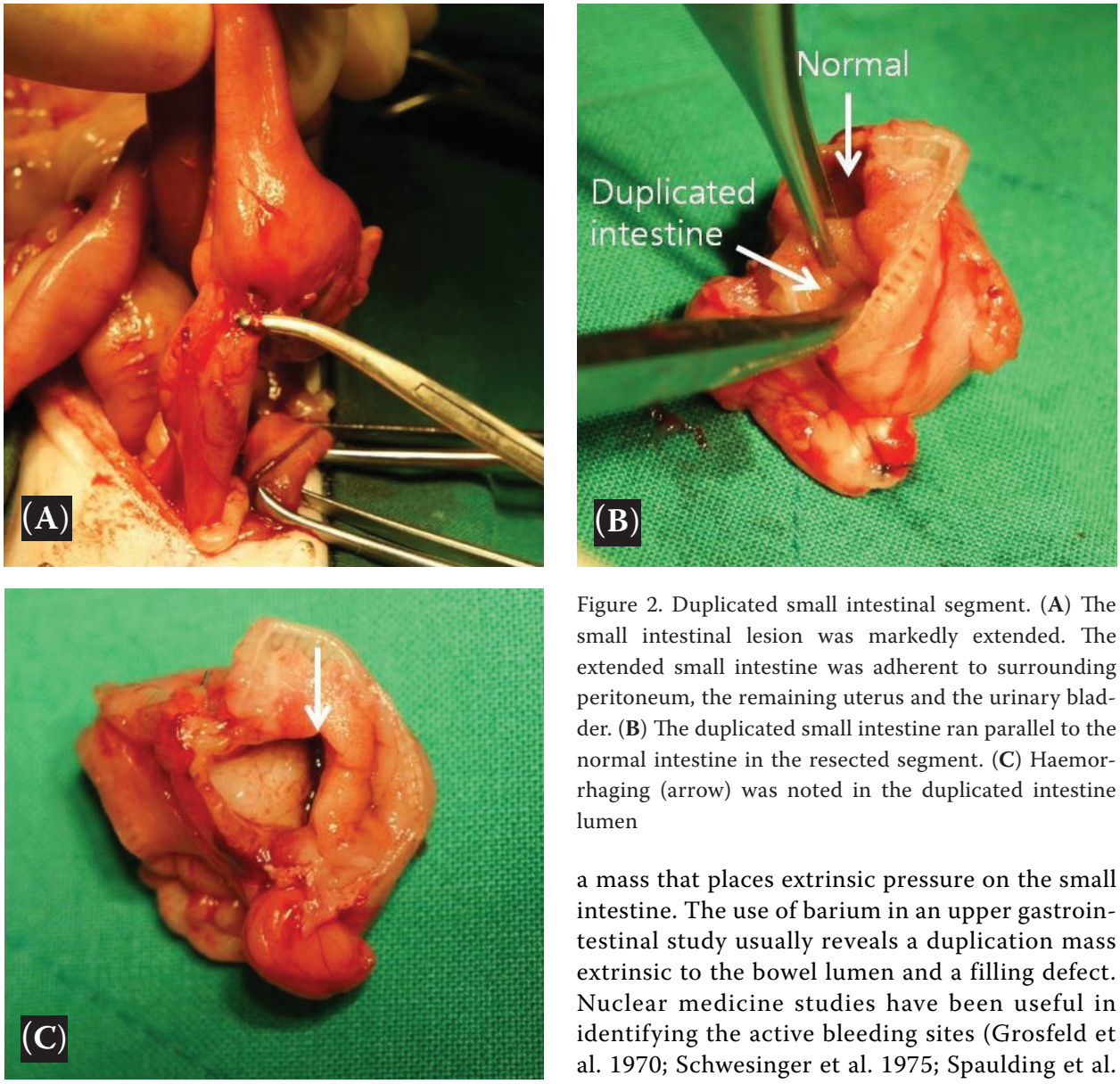

Figure 2. Duplicated small intestinal segment. (**A**) The small intestinal lesion was markedly extended. The extended small intestine was adherent to surrounding peritoneum, the remaining uterus and the urinary bladder. (**B**) The duplicated small intestine ran parallel to the normal intestine in the resected segment. (**C**) Haemorrhaging (arrow) was noted in the duplicated intestine lumen

bowel with the duplicated intestine on the border of one leaf of the mesentery. In the intramesenteric type, the arteries pass over both surfaces of the duplication and reach the bowel with the duplication located between both leaves of the mesentery (Landon et al. 2007; Jung et al. 2009). In the dog of this report, the enteric duplication was Type IB, because the segment was a non-communicating tubular loop duplication of the normal intestine. A separate, or parallel blood supply was not evident.

In radiographic examination, enteric duplication is frequently non-specific. A soft tissue mass may be seen within the abdomen and mass-displacing viscera may also be observed. An appearance of partial small intestine obstruction can be caused by a mass that places extrinsic pressure on the small intestine. The use of barium in an upper gastrointestinal study usually reveals a duplication mass extrinsic to the bowel lumen and a filling defect. Nuclear medicine studies have been useful in identifying the active bleeding sites (Grosfeld et al. 1970; Schwesinger et al. 1975; Spaulding et al. 1990; Deftereos et al. 2004). In this case, there was no significant finding on radiographic examination, nor were other deformities such as vertebral dysplasia confirmed. An upper gastrointestinal study was not conducted. Ultrasonographic examination was performed to find the cause of the melena.

Ultrasonography plays a critical role in the diagnosis of alimentary tract enteric duplication. The ultrasonographic appearance of an enteric duplication is a "double-layered wall" or "muscular rim sign". The four layers (mucosa, submucosa, muscularis and serosa) of the normal intestine wall are usually evident in the wall of the enteric duplication. A common muscular layer is present between the duplicated intestine and its adjacent bowel (Spaulding et al. 1990; Deftereos et al. 2004;

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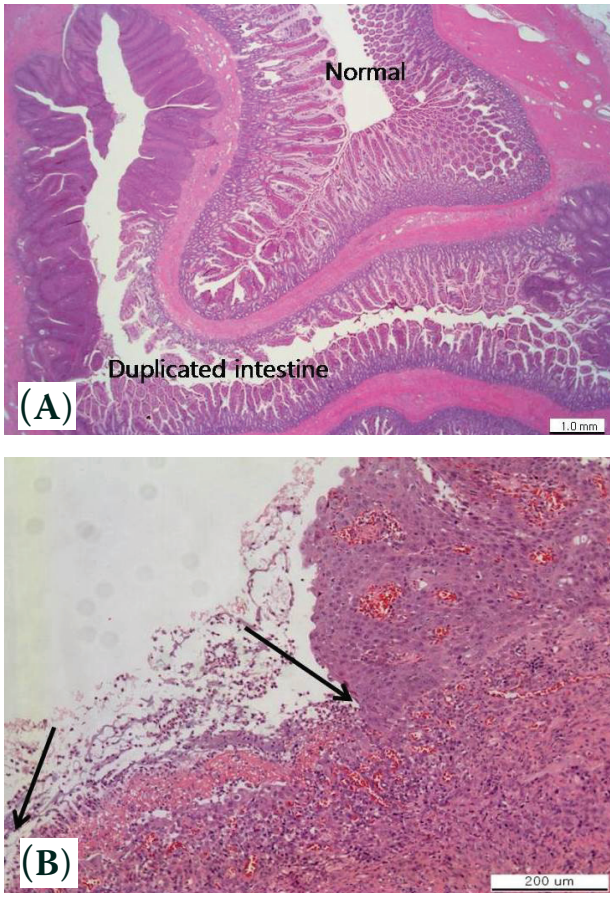

Figure 3. Histopathological findings of the resected small intestine. (**A**) The duplicated intestine shared an intestinal wall barrier with the normal intestine. The lining of the duplicated intestine had a normal mucosa layer. The septum between the normal and duplicated intestine consisted of mucosa, submucosa and muscularis layers. (**B**) Multifocal haemorrhaging and focal ulceration (arrow) was identified in the duplicated intestine

Cheng et al. 2005; Radlinsky et al. 2005). In the present case, a multi-layered appearance of the small intestine was identified. Both intestines had the normal four layers of the intestinal wall (mucosa, submucosa, muscularis and serosa). They were divided into two intestinal segments. A small amount of fat was found to be invaginated between the layers as well as partial corrugation of the small intestine. Intussusception and enteric duplication were the differential diagnoses. However, it was difficult to discriminate these two possibilities using ultrasonography.

Diagnosis of enteric duplication requires histopathological examination in order to differentiate the phenomenon from enteric diverticulum. Diverticulum is caused by a defect to the tunica muscular layer, allowing mucosa to bulge outward (Arthur et al. 2003). Histopathological examination revealed that the duplicated small intestine was composed of normal intestinal layers (mucosa, submocosa and muscularis). A diverticulum could be ruled out because the presence of the muscularis layer was confirmed. The normal small intestine was covered with simple columnar epithelium. However, some of the mucosa in the duplicated intestine was composed of squamous epithelium, indicating that severe squamous metaplasia was caused by chronic irritation. Severe multifocal haemorrhaging and focal ulceration were observed in squamous foci in the duplicated intestine. Anaemia was suspected as a result of haemorrhaging in the duplicated intestine. Because of the non-communicating nature of the duplicate intestine, haemorrhaging in the duplicated intestine did not appear to be the cause of the melena. We suspect that this symptom may have been caused by the presence of an unidentified lesion such as a gastrointestinal ulceration.

The standard treatment for enteric duplication is surgical excision. Complete excision should be performed whenever possible because complications can ensue from residual unresected duplications. Minimal resection of the adjacent normal intestine may be executed to prevent the risk of complications caused by obstruction or perforation. The common blood supply to the bowel must be preserved to avoid ischaemic necrosis (Kraft 1962; Grosfeld et al. 1970; Patenaude et al. 1993; Lo et al. 2004; Jung et al. 2009). Segmental excision of the involved bowl including the duplicated intestine with end-to-end anastomosis was conducted for this patient. The lesion appeared to be an extended intestine. The duplicated intestine was identified to be the jejunal section. The lesion had adhesions to the peritoneum, the remaining uterus and the urinary bladder. However, other deformities such as urogenital duplication were not observed. No procedures were carried out to determine the cause of the side effects such as ischaemia or obstruction. The prognosis could not be evaluated because the patient expired during the postoperative period.

In conclusion, surgical inspection, ultrasonographic and histopathological examinations played important roles in the diagnosis of this enteric duplication. The case was finally diagnosed as non-communicating intestinal duplication without other anomalies.

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