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Genus - Strongylus

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Species

Strongylus vulgaris

Strongylus edentatus

Strongylus equinus

Host : Equine

Location : Large intestine

Character	*S. vulgaris*	*S. edentatus*	*S. equinus*
Size	1.5-2.5 cm	2.5-4.5 cm	2.5-5 cm
Teeth in buccal capsule	two ear shaped dorsal teeth	No tooth	3 teeth
Leaf crowns	Both external & internal leaf crowns are present	Both external & internal leaf crowns are present	Both external & internal leaf crowns are present
Bursa	Present	Present	Present

Life-cycle:

- Life-cycle : Direct
- Host : Equine
- Location : Large intestine
- Infective stage : L3
- Transmission of infection : Ingestion of infective larvae along with feed and water.

Strongylus vulgaris

- The eggs are passed out in the faeces and they hatch under suitable conditions of moisture and temperature.
- The hatched out first stage larvae are rhabditiform.
- They grow and moult to second stage larvae which are less rhabditiform.
- The second stage larvae after performing the second moult, develop to third stage. This stage is infective sheathed strongyliform larva.
- Infection of the final host occurs by ingestion of infective larvae along with feed or water. The ingested larvae undergo exsheathment in the intestine and penetrate into the intestinal wall.

- After about a week, they migrate to the anterior mesenteric artery (cranial artery) through the wall of the submucosal arterioles.
- Here they remain associated with formation of thrombi and aneurysms of blood vessel (**verminous aneurysm**).
- After about 45 days of infection, they migrate back to the wall of the caecum and colon via the arterial system. Subsequently, the larvae enter into the lumen of caecum and colon and attain maturity.
- Females start laying eggs in about **6-7 months** after the infection.

Strongylus equinus

- The preparasitic development is similar to that of *S. vulgaris*. Infection is by ingestion of infective larvae.
- The L3 penetrate the large intestinal wall and form nodules in muscular and subserosal layers. Within the nodules, they moult to L4 which then migrate to the peritoneal cavity and then into the liver where they remain for about 2-3 months.
- From liver, they reach again to the peritoneal cavity via the pancreas and moult to L5 which reach to the large intestine by penetration and become adult.
- Pre patent period : **8-9 months**

Strongylus edentatus

- The preparasitic life-cycle is similar to that of of *S. vulgaris*. Infection is by ingestion of infective larvae.
- The L3 penetrate the large intestinal wall and reach to the liver portal circulation. There, they moult to L4 about 6-8 weeks after infection.
- These L4, then migrate under the peritoneum via the hepatorenal ligament and reach to the right abdominal flanks where they form haemorrhagic nodules. In these nodules, they remain for about 3 months and moult to L5 which migrate towards the wall of large intestine where they again form haemorrhagic nodules which finally rupture and release the worms into the lumen.
- Pre patent period : **10-12 months**.

PATHOGENESIS

❖ Larva :

S. vulgaris

- The larvae of *S. vulgaris* are most pathogenic and cause arteritis, thrombosis and **thickening of the cranial mesenteric artery**.
- **Aneurysm**
- The emboli may sometimes break away and lodge in the small blood vessels. As a result, there will be partial or complete ischemia in the intestinal part and produce colic necrosis and gangrene in the parts of bowel.

S. equinus

- The larvae produce haemorrhage and destruction of liver cells. They **form nodules** in the wall of intestine.

S. edentatus

- The larvae produce **haemorrhagic nodules** in the right abdominal flanks under peritoneum and in the wall of intestine. These nodules sometimes burst into the peritoneal cavity and cause peritonitis, secondary bacterial infection and haemorrhage etc.

❖ **Adult** :

- The adult parasites are having well developed buccal capsule with teeth by which the can take the plug of mucosa and also suck blood. There may also be haemorrhage from the feeding points after detachment of worms.
- The tissue feeding may lead to the gross damage of the caecum and colon followed by ulceration which may heal, leaving the small scars. The loss of blood due to sucking and haemorrhage may cause anaemia.

CLINICAL SIGNS

- Colic
- Rough coat
- Diminished appetite
- Diarrhoea
- Edematous swelling on the abdomen and leg
- Emaciation and anaemia
- Adult worm causes heavy blood loss due to blood sucking activity resulting in anaemia. (Normocytic, normochromic anaemia).
- Large number of haemorrhagic ulcers are seen in the intestine which indicates the site of attachment of worm.

DIAGNOSIS

- Clinical signs like anaemia, diarrhoea, poor growth etc.
- Faecal examination for eggs
- **Verminous aneurysm** in cranial mesenteric artery can be detected by rectal palpation.

TREATMENT

- Fenbendazole - 7.5mg / Kg b wt. (oral).
- Thiabendazole - 440mg / Kg b wt. (oral).
- Ivermectin - 0.2mg / Kg b wt s/c.

CONTROL

- General hygienic measures
- Use clean pasture
- Proper disposal of faeces
- Overstocking in stables and on grazing grounds should be avoided.
- Periodic deworming of horse
- Treatment of infected animal
- Providing of parasitic- free fodders
- Alternate grazing on pasture of horses by other species of animals should be followed to reduce the larval burden of strongyles in the pasture.
- Ponies could be protected against *S. vulgaris* by vaccination at the age of 8-10 weeks with irradiated larval vaccine.