

## Uterine horn aplasia with complications in two mixed-breed bitches

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### ABSTRACT

Unilateral segmental uterine horn aplasia was found in 2 mixed-breed bitches. The 1st bitch was presented with clinical signs of acute abdominal pain, and pyometra was suspected. Pyometra was confirmed surgically with rupture of a blind-ending cranial portion of the anomalous right uterine horn, which had resulted in peritonitis. The 2nd bitch was presented for routine ovariohysterectomy. The right uterine horn was affected by segmental aplasia, with mucometra of the cranial portion of the affected horn. Histopathology demonstrated both uteri to have diffuse cystic endometrial hyperplasia. It is postulated that cystic endometrial hyperplasia, together with the congenital anomaly, resulted in pyometra in one case and in mucometra in the other case. This is believed to be the 1st report of uterine horn aplasia in the bitch in association with clinical signs and lesions other than infertility.

**Key words:** cystic endometrial hyperplasia, mucometra, pyometra, segmental aplasia, uterine horn, uterus.

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### INTRODUCTION

A variety of forms of segmental uterine aplasia in several domestic animal species, including cattle, sheep, pigs, horses, cats and dogs, have been described. This congenital anomaly is commonly reported in the sow and the cow<sup>1,6</sup> and arises from defective development of the Müllerian (paramesonephric) ducts during embryogenesis<sup>6</sup>.

There are several reports of *uterus unicornis* and segmental aplasia affecting the uterine horns of the bitch<sup>6,10</sup>. We have found no reports of segmental uterine aplasia associated with clinical signs other than infertility in the bitch<sup>3,10</sup>.

This report describes 2 cases of unilateral segmental uterine horn aplasia. Both were associated with additional uterine pathology in the form of different manifestations of the cystic endometrial hyperplasia-endometritis-mucometra-pyometra complex. The 1st case involves a bitch with severe disease signs probably arising from the underlying congenital uterine anomaly. The 2nd case was an

incidental finding in a bitch presented for routine ovariohysterectomy.

### CASE HISTORIES

#### Case 1

A 19 kg mixed-breed bitch approximately 3 years of age was presented with abdominal pain, pyrexia (40 °C) and vomiting. She was in a collapsed state and 5 % dehydrated. There was no obvious vaginal discharge. Neutrophilia with left shift and monocytosis were noted on blood smear. Abdominocentesis revealed a septic exudate with phagocytosed cocci in degenerate neutrophils. On faecal analysis, eggs of *Ancylostomum* were identified. Abdominal survey radiographs were inconclusive. The bitch was admitted to hospital and immediate therapy was instituted, consisting of intravenous balanced crystalloid fluid administration (Sabax Plasmalyte B, Adcock Ingram Critical Care) and broad-spectrum antimicrobials, amoxycillin (Amocillin, CAPS SA) at 20 mg/kg, p.o., b.i.d. and metronidazole (Zolerol, Rolab) at 20 mg/kg, p.o., b.i.d.. Although the bitch was maintained on broad-spectrum antimicrobials for the following 2 days, the pyrexia and vomiting persisted. On the 4th day she was referred to one of the authors for further investigation. Abdominal ultrasound (Concept 2000,

TGL Medical) with a 7.5 Mhz linear array probe revealed the presence of generalised ascites and moderate fluid dilatation of the uterus. Abdominocentesis confirmed the previous findings of a septic exudate. Vaginoscopy showed the vaginal mucosal features associated with late dioestrus or anoestrus of the ovarian cycle of the bitch. Haematology revealed an inflammatory leukogram characterised by severe leukocytosis, neutrophilia, regenerative left shift, monocytosis and mild lymphopaenia. Mild microcytic hypochromic anaemia was present, possibly associated with the hookworm infestation. Serum electrophoresis revealed hyperglobulinaemia, characterised at electrophoresis by increased acute-phase reactants ( $\alpha$ - and  $\beta$ -globulins) indicative of acute inflammation (TSP 67 g/l, albumin 27 g/l, globulins 40 g/l)<sup>5</sup>. All other serum chemistry and urinalysis were within the normal range. It was decided to perform an exploratory laparotomy. Intravenous antimicrobials, N-benzyl penicillin (Novopen, Novo Nordisk) at 20 000 IU/kg and metronidazole (Sabax Flagyl, Adcock Ingram Critical Care) at 20 mg/kg, and fluids (Sabax Plasmalyte B, Adcock Ingram Critical Care) were administered. Anaesthesia was induced with intravenous 5 % thiopentone-sodium (Intraval-sodium, Rhône-Poulenc Rorer) at 10 mg/kg and maintained with fluothane in oxygen.

On incision, the peritoneal cavity contained approximately 3 litres of mucopurulent exudate. Extensive fibrinous adhesions involving the serosal surfaces of the intestines, bladder, uterus, broad ligament and omentum were noted. Closer inspection of the uterus showed a discrete rupture involving the ovarian pole of the right uterine horn, indicated by leakage of mucopurulent material. Distal to the rupture, the horn consisted of a blind-ending sac 4 cm in diameter and connected to the uterine body by the broad ligament. There were blood vessels but no obvious uterine tissue within this portion of broad ligament. The contralateral horn was approximately 10 cm long with a diameter of 1 cm and was normally confluent with the uterine body. An ovariohysterectomy was performed,

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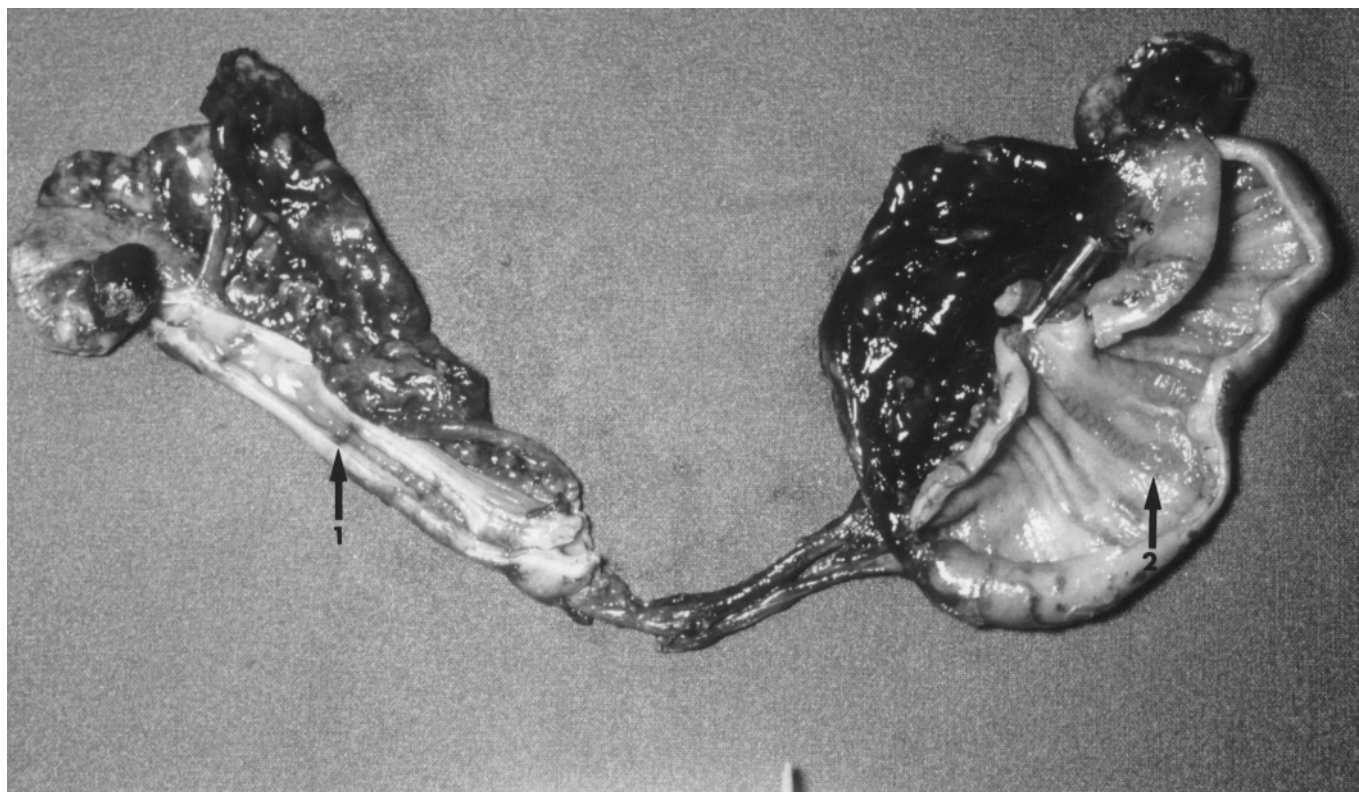


Fig 1: Incised uterus from Case 1, showing the left uterine horn with placental attachment sites (arrow 1). The abnormal right uterine horn has a granular luminal endometrium (arrow 2). Note the perimetritis with inflammatory changes and haemorrhage affecting the broad ligament.

with removal of the ovaries and amputation of the uterine body adjacent to the cervix. The peritoneal cavity was lavaged with copious amounts of warmed fluids (Sabax Ringers lactate, Adcock Ingram Critical Care) and the effluent removed by suction. The incision was closed routinely. Post-operatively the bitch received ongoing antimicrobial therapy (amoxycillin and metronidazole), and intravenous crystalloid fluid therapy (Sabax Maintelyte B, Adcock Ingram Critical Care) was continued for 24 h. She recovered uneventfully and was discharged a week later.

The reproductive tract was examined. The broad ligament, ovarian bursae and ovarian surfaces were haemorrhagic with fibrinopurulent exudate. The right horn ended blindly on the broad ligament, which connected with the uterine body. On opening the uterus (Fig. 1), the dilated right horn contained mucopurulent exudate overlying a hyperaemic and faintly granular endometrium. The endometrial surface of the contralateral horn was macroscopically normal and had 4 previous placental attachment sites, representing an earlier (probably unilateral) pregnancy.

A swab of the exudate from the right uterine horn was submitted for aerobic and anaerobic bacterial culture. No

growth was obtained after 48 h of incubation.

Histopathological investigation confirmed unilateral partial segmental aplasia involving the caudal portion of the right uterine horn. There was severe diffuse purulent endometritis with diffuse cystic and hyperplastic changes of the right uterine horn. The cranial extremity of the right horn was also affected with a locally extensive metritis. Fibrinopurulent peritonitis involving the broad ligament and peri-ovarian structures was present. Bilaterally there was ovarian folliculogenesis, with rete cysts present as an incidental finding.

## Case 2

A clinically normal, small mixed-breed bitch of uncertain age was presented for routine ovariohysterectomy. On opening the abdomen a problem was encountered in adequately identifying the reproductive tract owing to an anomalous right uterine horn. The middle section of the right horn appeared to be absent, with the cranial and caudal portions of the right uterine horn blind-ending and connected by intervening broad ligament (Fig. 2).

All portions of the uterine horns were 1.5 cm in diameter and normal in gross appearance. On incision (Fig. 3), the cranial portion of the right uterine horn

contained a moderate amount of mucoid, brown fluid. The endometrium was uniformly thickened and had multiple raised fluid-filled cysts ranging from 1 to 3 mm in diameter. Both the cranial and caudal portions of the right uterine horn ended blindly at the junction with the intervening broad ligament. The luminal surface of the caudal portion of the right uterine horn was granular and covered with scant clear mucus. On transverse section the endometrium was mildly thickened. The left uterine horn was similar in appearance to the distal portion of the right uterine horn.

A swab of the fluid from the proximal portion of the right uterine horn was submitted for aerobic and anaerobic bacterial culture. No growth was obtained after 48 h of incubation.

Histopathology of the portion of broad ligament between the blind-ending portions of the right uterine horn confirmed that neither uterine nor uterine-scar tissue were present. A diagnosis of partial unilateral segmental aplasia associated with mucometra of the cranial portion of the anomalous uterine horn was made. Diffuse cystic endometrial hyperplasia affected all portions of the uterus. Multiple *corpora lutea* were present on both ovaries. This is in concordance with the progestational morphology of the



Fig 2: Intraoperative view of the reproductive tract in Case 2 showing the cranial (long arrow) and caudal (short arrow) blind-ending segments of the right uterine horn.

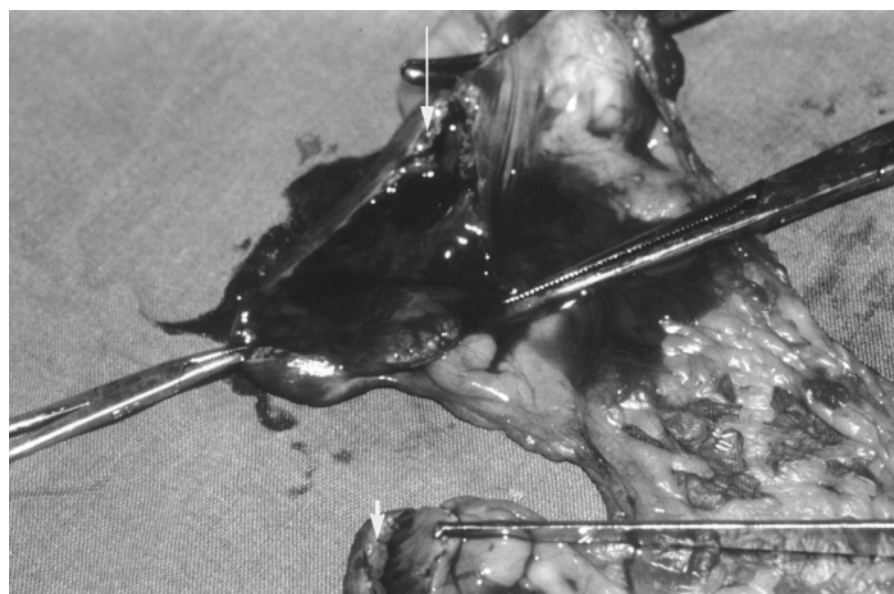


Fig 3: Incised right uterine horn segments of Case 2. The cranial segment (long arrow) has mucometra and cystic endometrial hyperplasia (CEH). This segment is separated from the caudal blind-ending segment (short arrow) by broad ligament without evidence of any intervening uterine tissue.

surface endometrium, which was diffusely hypertrophic and vacuolated. These histological features are consistent with dioestrus in the bitch.

## DISCUSSION

Various forms of segmental aplasia of the uterine horn have been reported in many animal species. This anomaly is more frequently seen in cattle and pigs<sup>1,6</sup>. It is known as 'white heifer disease' in white Shorthorns, but also occurs in other breeds. Segmental aplasia in the bovine has been associated with clinical signs of

anoestrus, abnormal oestrus, infertility, and difficulty encountered when performing artificial insemination<sup>12,13,14</sup>. Clinical findings of mucometra, or occasionally pyometra via haematogenous spread of infectious agents have been reported<sup>6,12,13,14</sup>. Complete aplasia of the left uterine horn was recorded as an incidental finding in a 3-year-old Morgan filly<sup>1</sup>. This anomaly has also been reported in various forms in 2 Clydesdale mares and 1 Shetland pony<sup>1</sup>. It was found that 0.5 % of non-pregnant ovine uteri examined at an abattoir had unilateral

horn aplasia<sup>9</sup>. Segmental unilateral aplasia of the uterine horn with mucometra and infertility were recorded in a Himalayan cat<sup>8</sup>. Segmental aplasia of a uterine horn was found in a domestic longhaired cat associated with intermittent abdominal distension<sup>7</sup>.

*Uterus unicornis*, fusion of the horns and decreased length of horns are congenital defects most commonly affecting the uterus in dogs<sup>6,10</sup>. Segmental aplasia of a uterine horn in dogs is described, but has not been recorded in association with any clinical abnormality other than infertility<sup>1,4,10</sup>.

Segmental aplasia of the uterine horns arises as a result of defective development of the Müllerian (paramesonephric) ducts during embryogenesis. Various irregularities in the embryological development of the tubular genitalia result in a variety of abnormal manifestations. These may affect the oviducts, the uterine horns and body and the cranial vagina. Abnormalities include segmental aplasia of the left or right uterine horn. These congenital anomalies are usually incidental findings encountered at either surgery or *post mortem* examination. Segmental aplasia may be associated with females presented with a history of 'infertility'<sup>3,4,7,10</sup>.

In this report, 2 cases involving unilateral segmental aplasia of a uterine horn in the bitch are described. Cystic endometrial hyperplasia was diffusely present in the uteri of both bitches. Segmental uterine aplasia together with cystic endometrial hyperplasia, in these 2 cases, was seen to predispose to uterine pathology, characterised by mucometra and pyometra. Endometrial hyperplasia (including cystic changes), endometritis, mucometra and pyometra are a pathophysiological continuum<sup>2,4,10,11</sup>. Cystic endometrial hyperplasia may reflect an underlying hormonal condition that together with additional predisposing factors may lead to a variety of inflammatory and infectious changes<sup>2,4,6,10</sup>.

This continuum resulted in severe clinical disease in 1 bitch, with pyometra and rupture of the blind-ending uterine sac. Pyometra generally arises following uterine contamination through a patent cervix (normally associated with pro-oestrus and oestrus) by opportunistic pathogens originating from the vaginal vault<sup>2,4</sup>. Pre-existing cystic endometrial hyperplasia of the uterus creates a favourable milieu for these microorganisms to become pathogenic. Inadequate patency impeding uterine drainage leads to complications associated with intra-uterine mucus and pus accumulation. Impeded uterine drainage is usually associated

with complete or partial cervical closure, which may be functional or anatomical in nature. These complications more commonly produce clinical signs during dioestrus in the bitch<sup>2,4,6</sup>.

Ascending uterine infection through the cervix was unlikely in the 1st case because of the anomaly preventing communication of the cranial uterine segment with the uterine body. The most probable route of infection was haematogenous<sup>6</sup>. This bitch had also had a previous pregnancy indicated by the presence of numerous placental attachment sites within the contralateral uterine horn. This clearly illustrates that neither segmental aplasia nor cystic endometrial hyperplasia are invariably associated with infertility<sup>4</sup>. In the 2nd bitch, the blind-ending portion of the aplastic horn was dilated with a sterile fluid consistent with mucometra<sup>6</sup>. In both cases partial segmental aplasia was confirmed histopathologically, with the absence of any uterine remnants or uterine-scar tissue in the aplastic segments.

The presence of mucometra or pyometra in the blind-ending segments of the grossly abnormal uterine horns is significant. This supports the hypothesis

that inadequate patency impeding uterine drainage contributes to the development of complications of a pre-existing cystic endometrial hyperplasia<sup>2</sup>.

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