Assessing the mare for breeding: the warning signs we would like to ignore

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Introduction

Selection criteria for horse mating commonly include performance, pedigree, potential commercial value of offspring, conformation but hardly ever fertility. Our task, as theriogenologist is to identify conditions that may negatively impact on fertility and apply correction where appropriate. Subtle signs of reproductive derangement should never be ignored, as they may rapidly escalate into more serious and persisting conditions, compromising the ability of the mare to get pregnant and carry to term.

Ovarian Abnormalities

Ovarian abnormalities are often detected on US, and may include tumours, ovarian hematomas, cystic structures, persisting luteal structures and anovulatory follicles. The most common ovarian tumours found in the mare include: Granulosa Cell Tumor (GCT), Cystadenoma and rarely Teratoma and Dysgerminoma. Only Dysgerminomas may potentially turn malignant. GCT are the most frequently encountered ovarian tumours in the mare and are usually unilateral, slow growing, and benign. According to the different prevailing hormonal profiling (inhibin vs testosterone), affected mares may exhibit behavioural abnormalities like anestrus or aggressive behaviour. Diagnosis is based on changes in behaviour, ovarian US appearance and hormonal profiling, including progesterone, inhibin, testosterone. Recently, the anti-Müllerian hormone (AMH) has become the most sensitive biomarker in the diagnosis of the condition.
Small cystic structures within the ovulation fossa may sometimes be observed on US and raise concern as they can potentially interfere with oocyte release at ovulation time. Paraovarian and oviductal cysts are incidental findings, but are not associated with reduced fertility. They usually arise from structural remnants of the müllerian and wolfian ducts. Pedunculated paraovarian and oviductal cysts may sometimes resemble follicular structures on US, but careful palpation will discriminate, due to the location and excessive mobility of the cystic structures.
Ovarian hematomas result from excessive hemorrhage into the follicular lumen and may resemble in US appearance to GCT. The controlateral ovary is fully functional and no abnormalities are found in hormonal profiling. The condition does not require treatment and hematomas gradually reduce in size during the course of several weeks. Occasionally, very large hematomas may disrupt ovarian tissue and result in a non functional ovary. Haemoperitoneum has been reported as an infrequent complication.
Small inactive ovaries (usually a bilateral condition) are a common feature during debilitating medical disease, malnutrition, Cushing's disease, advanced age, anestrus (both seasonal and lactational), karyotype abnormalities and exogenous hormone therapy.
Corpora lutea that fail to regress at the normal time (14-15 days) in the non pregnant mare are considered to be pathologically persistent. The lifespan of a corpus luteum may be prolonged by a variety of causes including failure of secretion of prostaglandins in amounts adequate to induce lysis, presence of an immature CL from a diestral ovulation that occurred shortly before prostaglandin release, early embryonic death after maternal recognition of pregnancy and chronic uterine infection, resulting in endometrial disruption and a decreased prostaglandin production. If untreated the corpus luteum may persist for 2-3 months.

Ovulation failure is a physiological event during the spring and autumn transitional period and early pregnancy (40-150days gestation), but mares may develop follicles that do not ovulate during the physiologic breeding season. These structures are referred to either as anovulatory, hemorrhagic, luteinising or persistent follicles. Anovulatory follicles maybe quite large, persist for up to 2 months and result in abnormal estrus behaviour. The cause of ovulation failure has been suggested to be endocrine in nature, either from a lack of sufficient pituitary gonadotropin stimulation to induce ovulation or from insufficient estrogen production from the follicle itself. The majority of persistent follicles will regress in 1-4 weeks, but they are usually unresponsive to administration of ovulatory agents. The condition will occur in approximately 11% of cycles and is more common in mares >16 years of age. About 45% of affected mares will exhibit another anovulatory follicle in a subsequent estrous cycle. Finally, about 65% of anovulatory follicles produce progesterone and will respond to exogenous prostaglandin.

Uterine Abnormalities

The uterus can be readily examined by US for abnormal content: endometrial fluid, cysts, foreign bodies (including retained endometrial cups), tumours , inspissated mucus, biofilm, air and intraluminal adhesions. Excessive endometrial edema, abnormal shape of endometrial folds and blood flow abnormalities may also be observed on US and further clarified by the use of Doppler technology. Endometrial cultures and cytology, biopsies and hysteroscopy are additional tools to complete the investigation.

Fluid Accumulation

Causes of intrauterine fluid accumulation include impaired cervical function, persistent mating induced endometritis (PMIE), a dependent, pendulous uterus, impaired lymphatic drainage, angeosis, infection or a combination of the above. The location of fluid accumulation within the uterine lumen, whether it is visualized in the uterine body, near the cervix or within the uterine horns, provides a clue as to the cause. Mares with primary cervical incompetence or cervical adhesions tend to retain fluid just cranial to the cervix. In addition, mares susceptible to persistent mating-induced endometritis accumulate fluid in the uterine horns and body as a result of impaired myometrial contractions, poor lymphatic drainage, excessive glandular secretions, angeosis, a dependent uterus and possibly abnormalities in hormonal and neurological signalling. Mares with poor perineal conformation may aspirate air, fecal material, environmental contaminants and/or urine into the vagina and uterus, resulting in pneumovagina and pneumometra. On US air appears as a series hyperechoic dots and lines throwing strong artifactual echoes (acoustic shadowing) , to obliterate the view of deeper tissues. Urine may also be observed pooling in vagina, bathing the vaginal cervical os, as hyperecoic fluid. Vestibular varicosities may cause intermittent vaginal bleeding in a small percentage of aged barren and pregnant mares.

Infectious endometritis

Infectious endometritis is most commonly seen in older (> 12 yr of age), pluriparous mares with defective perineal conformation. It may also occur in mares that are susceptible to PMIE and are not treated after mating, in embryo donor mares that are repeatedly manipulated for embryo recovery and in mares with cervical incompetence. Etiology includes a variety of bacterial and fungal microorganisms. Clinical signs include: reduced pregnancy rates, “shortened inter-ovulatory period”, vulvo-vaginal discharge, excessive or persisting endometrial edema and intrauterine fluid accumulation associated with positive endometrial cultures and cytology. On vaginoscopy the cervical os appears reddened and exudates may extrude from it, accumulating in the anterior vagina. Cytology may display different features depending on the responsible microorganism. Recovery of beta-hemolytic *Streptococcus*,

Staphlococcus, or *Klebsiella* are more likely to be associated with a positive cytology (defined as having > 2 neutrophils/field) than recovery of *E.coli*, *Pseudomonas*, or *Enterobacter cloaca*. Laboratory data should always be interpreted in relation to clinical signs as false positives and false negatives occur and correlation between cytology and culture results varies between organisms recovered. Localised area of infection may occur and present a real diagnostic challenge. Hysteroscopy may help in the diagnosis and treatment.

Angiosis/Uterine Cysts

Adequate blood flow to the uterus is needed for normal function. Age related changes and "inflammatory vascular alterations" within the walls of uterine arteries have been reported. The incidence as well as the severity of vascular lesions increased with the number of pregnancies and with aging. Angiosis appears to indirectly reduce fertility through a reduction in endometrial perfusion, and through disturbances in uterine drainage caused by reduced venous function.The most obvious clinical finding in mares with angeosis is the presence of cysts and the persistence of endometrial edema after ovulation. Endometrial lymphangectasia develops physiologically during estrus, resulting in the typical estrous edema of the uterine wall. The edema disappears rapidly after ovulation, providing the drainage mechanisms are functionally intact. If they are not, the result is a pathological endometrial edema, morphologically characterized by persistent lymphangectasia. A ventrally, dependent uterus in older multiparous mares appears to contribute to the problem. Findings show a positive association between uterine cysts and disturbed uterine hemodyamics: the larger the lymphatic cyst the greater the perturbation of blood flow.