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FUNCTIONAL FORMS OF INFERTILITY IN CATTLE

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Introduction

Infertility denotes a degree of reduced fertility which results in failure to produce or delay in producing the annual live calf. Infertility is one of the important economic losses in high producing herds and that modern feeding and management practices in large herds may accentuate the problem. Various causes of cattle infertility can be broadly grouped as anatomical factors, functional abnormalities, management problems and infectious agents (Arthur et al., 1989). Recently there has been a noticeable change in the causes of infertility in cattle. Wide spread use of artificial insemination reduced the infectious forms of infertility. Infertility due to anatomical factors are comparatively less. The demands put up on the dairy cow to produce more milk and the genetic selection for the high yield have resulted in functional aberrations of the reproductive and endocrine systems, as a serious concern. The objective of this article is to explain the causes, diagnostic methods and treatment regimen of functional forms of infertility in cattle.

Classification of functional infertility

As a rule the functional forms of infertility tend to affect individual animals within a herd but in the aggregate they constitute an important causes of infertility. Causes for functional forms of infertility are endocrinological abnormalities, inherited factors, nutritional deficiencies/ excesses, social influences and stress of production. This form of infertility can be broadly divided as no observed oestrus, ovulatory defects, luteal deficiency, hormonal imbalance and fatty liver syndrome.

1. No observed Oestrus

Causes for this condition are true anestrus, suboestrus/silent heat, non-detected oestrus (come under management causes of infertility), persistent CL and Ovarian cysts.

1.a. True anestrus: Ovaries are quiescent and inactive. Reason for the failure of normal activity may be insufficient production of gonadotrophins to cause folliculogenesis. Main feature will be the absence of a CL either developing, mature or regressing. Milk or blood progesterone determinations are helpful in confirming diagnosis, two samples can be taken at 10day intervals or a single sample ten days before ractal palpation.

True anestrus is most frequently seen in high-yielding dairy cows and in firstcalf heifers, which are still growing . The anterior pituitary appears to be refractory to stimulation with GnRH in the immediate postpartum period. The refractory period is probably due to the duration of progesterone-induced negative feed back during pregnancy. The act of suckling stimulates bursts of prolactin secretion which may be responsible for the extension of the period of anestrus. The duration of anestrus in cows nursing calves is longer than in similar cows milked twice daily; this suggests that nursing or frequency of milk removal may influence the pituitary gonadotropic activity. Deficiencies of phosphorus, copper, cobalt and manganese, ingestion of phyto-oestrogens and diseases which cause severe weight loss and debility or metabolic disturbances, such as ketosis can cause anoestrus.

Treatment: Improve feeding (energy intake should be improved), PMSG (Folligon) 2000 IU, S.C. or I.M, GnRH, (Receptal, Fertagyl) 0.5 mg IM, SC or IV, PRID (Insert into vagina and left insitu for 13-14 days, with PGF2" administer 24 hours before removal, oestrus occurs 2-5 days after withdrawal) and oestrogens (primarin).

1.b. Suboestrus: Internal oestrus changes without external heat signs. Suboestrus can be due to genetic predisposition, over weight and nutritional deficiencies of $-carotene, phosphorus, copper or cobalt.

Treatment : Nutritional deficiency has to be corrected. If corpus luteum is present, give PGF2" or analog (Dinoprost 25mg (lutalyse) cloprostenol (estrumate), fenprostalene (Synchrocept B), Luprostiol (prosolvin), Tiaprost (iliren) followed by fixed time insemination. If CL is at a refractory stage a double injection prostaglandin regimen at an 11-day interval could be used.

1.c. Persistent CL: Due to uterine infection, there will be interference with release of luteolysin and CL will persist. The only sure way of reaching a true diagnosis would be sequential rectal palpation or the use of repeat milk or blood progesterone determination. Treatment is effective with PGF2" or analogue.

2. Ovulatory defects

Ovulatory defects are mainly delayed ovulation, anovulation and cystic ovaries. Ovulation in the cow is atypical since it occurs 10-12 hrs after the end of behavioral oestrus and 18-26 hrs after the ovulatory LH peak. Ovulatory defects are due to two causes- Endocrine imbalance and Mechanical factors.

2.a. Delayed Ovulation: Diagnosis can be made if the same follicle can be detected in the same ovary on two successive examination, one at peak oestrus and another 24-36 hrs later. If ovulation had not occurred by 24 hrs after service, the cow should be reinseminated.

Treatment: (i). GnRH - GnRH administration causes a rapid rise in FSH & LH concentrations which peak within 30-60 minutes and return to pre-injection values within 4 hrs. (ii). Double AI - 2nd AI 24 hrs after 1st AI

2.b. Anovulation: In this case follicle regresses and become atretic. Sometimes follicle luteinizes. Diagnosis can be made by noting on rectal palpation that a follicle persists longer than one would have suspected. In the case of the luteinized follicle it will remain for 17-18 days before regressing; the ovary containing it will be rounded, smooth and fluctuating rather than irregular and solid as it is with CL.

Treatment is effective with GnRH or hCG (chorulon, corion, profasi 1500-3000 I.U.I.V. or I.M.) If ovarobursal adhesions are present there is no treatment

2.c. Cystic Ovaries: Ovaries are said to be cystic, when they contain one or more persistent fluid-filled structures larger than a mature fallicle ie. >2.5 cm in diameter in one or both ovaries. The follicle increases in size, there will be degeneration of the granulosa cell layer and it persists, usually for at least 10 days. The consequence of this is to alter the normal cyclical activity of the cow. So that it becomes either acyclic or nymphomaniacal.

Pre disposing factors are genetic selection for high yield and feeding of high protein diet. Etiological factors are endocrine abnormalities and mechanical factors.

Traditionally ovarian cysts have been classified as either follicular cysts which are thin-walled, frequently multiple and with little or no luteal tissue in the cyst wall., or luteal or luteinized cysts which are thick-walled, more usually single and with a large quantity of luteal tissue present in the cyst wall. Cows with follicular cysts are usually nymphomaniacal and with luteal cysts are in anestrus.

Diagnosis can be made from history, clinical signs, rectal palpation and by measuring progesterone in blood or milk.

Treatment: (i) Follicular Cysts: In some cases spontaneous recovery can occur; hCG, GnRH and PRID are effective. (ii) Luteal cysts: hCG, GnRH and PGF2"

Both GnRH and hCG causes luteinization of the cyst, as a consequence plasma progesterone concentrations increases. This has a negative feedback on the pituitary, then causing a decline in endogenous LH, which is necessary for the maintenance of the cysts, resulting in its demise.

Prevention: By careful genetic selection improvements can be made by eliminating bulls that have sired daughters which have subsequently suffered from cystic ovarian disease. Ideally, cows should not be treated for cystic ovaries and certainly the progeny should not be used for breeding.

Prophylactic use of GnRH has shown some success in reducing the prevalence of cysts. (100-200 microgram GnRH at 12 to14 days post partum)

3. Luteal Deficiency

Embryonic mortality after artificial insemination accounts for the majority of reproductive failures in the bovine, with a mortality rate of 40% of all fertilized ova. In cattle, because most embryonic deaths occur between days 8 and 16 during hatching of the blastocyst and implantation, cycle lengths are unaffected (Jainudeen and Hafez. 1987). Progesterone is necessary for the maintenance of pregnancy. The main source of the hormone is the corpus luteum. The maintenance of progesterone secretion by viable corpus luteum is vital to early pregnancy. So that if CL is not completely formed or it is not functioning adequately then insufficient progesterone is produced and pregnancy fails. Luteal deficiency is a cause for repeat breeding in cattle.

It is impossible on rectal palpation to differentiate between a normal and abnormal CL. While determining progesterone concentrations in blood or milk it has been possible to make some assessment of luteal function. Low progesterone values were obtained during early luteal phase in repeat breeders. Diagnosis of luteal deficiency using single blood or milk samples is unreliable as there are wide fluctuations in the same animal. The conception rate will be higher in animals treated with progesterone within 3 weeks after artificial insemination (Srivastava and Kharche, 2001)

Treatment: (i). Injection of hCG or GnRH after ovulation. It will stimulate the development and function of the CL or induce accessory CL formation.

(ii). HCG or GnRH 14-17days after A.I. If the stimulus for the maternal recognition of pregnancy is weak, it will prevent the CL regressing and provide sufficient time for pregnancy to establish.

(iii). Progesterone injections (250-500 mg) at 7^{th}, 14^{th} and 21^{st} day of A.I.

4. Hormonal imbalance

Most hormonal disturbances causing infertility in cows are secondary to basic nutritional, hereditary and stress factors. Occasionally hormonal disturbances may be due to the ingestion or injection of exogenous steroids or other hormones (Roberts, 1986). The time of transport of the Oocyte and Zygote along the uterine tube is under the influence of oestogens and progesterone. Thus if there is an incorrect balance of these hormones there may be accelerated or retarded passage of the zygote, so that it reaches the uterus at a time when the environment is hostile to its survival. Even if an imbalance could be demonstrated, it is not easy to correct it.

5. Fatty liver syndrome

This condition is due to mobilization of body fat reserves, especially subcutaneous, to meet the energy deficit which occurs in milk production.

The consequences of high fat mobililization are ketosis, post partum metritis and retension of foetal membranes. In this syndrome, animal will be infertile. In animals with severe fatty liver the mean calving intervals and services per conceptions will increase.

Evidence of impaired liver function can be obtained by measuring blood cholesterol and the enzyme aspartate aminotransferase in cows 8 weeks before calving. The cholesterol will be lowered and aspartate aminotransferase elevated.

Attempts to prevent this condition can be made by ensuring that cows are not excessively fat at calving and receive adequate energy thereafter to exclude the need for excess fat mobilization. Prevent excess energy intake during the end of the lactation and dry period.

Conclusion

As we proceed with genetic selection of dairy cows for the high yield, the functional forms of infertility is a serious concern, that to be handled scientifically and economically. By timely preventive and treatment measures, most of the functional aberrations of reproduction can be avoided. For proper diagnosis, detailed examination by a professional more than once is needed.

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