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ReviewPaper

Recent Advances in the Incidence, Etiological Factors andManagementofUrolithiasisinBovines

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ABSTRACT
Urolithiasis condition can cause significant economic losses due to urethral obstruction; rupture of the urethraor bladder; or death from the buildup of toxic substances that are usually removed through the urine in bovines.It is a multifactorial disorder, diagnosed by laboratory findings and ultrasonography. Uroliths can be removedby surgical methods. Post-scrotal urethrotomy, tube cystotomy, cystorraphy is the common surgical methodsusedinmanagementofurolithiasisinbovines.Itmaybepreventedbydietarymodificationandurineacidification
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KEYWORDS:*Calculi,Sigmoidflexure,Urolthiasis,Urethrotomyetc.*

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I. INTRODUCTION

In cattle, the tubes that carry urine from the kidneys to the bladder (the ureter), or from the bladder tothe penis (the urethra), can become blocked by stones (uroliths or calculi) produced in either the kidneys or thebladder.Thispreventstheanimalfrompassingurineandproducestheconditionknownasobstructiveurolithiasis.
Urinary calculi, or uroliths, are concretions of solid mineral and organic compounds that causedisease through direct trauma to the urinary tract and obstruction of urinaryoutflow. Urinary calculi form inboth castrated and uncastrated males and also in females but obstructive urolithiasis is primarily a problem ofcastrated adultmales[3].

Calculi are mostly lodgeddistal to sigmoid flexure but may also lodged between the ischial arch andthe sigmoid flexure, may obstruct neck of bladder and in some cases a large calculus obstructs the urethra incattleandatglanspenisoratthesigmoidflexurebutmayalsoobstructtheneckoftheurinarybladderinbuffalo [3].The number of affected animals is usually low but can be as high as 10% annually in exceptionalcircumstances. An overall incidence of 5.04 percent in animals has been reported in India. The species wiseincidencehasbeen reportedas: goats 49.83percent,cattle 32.87 percent,dogs 14.53 percent,horses 1.38percent sheep 1.04 percent and cats 0.34 percent [2]. Higher incidence reported in buffalo calves as compare tocattle [3].The condition occurs more frequently in feedlot situations. Urolithiasis with intact urethra and ruptureurethra hasbeenreported89.48%and10.52%respectively [11].

POSSIBLEPREDISPOSINGFACTORS

Theetiologyiscomplexandmultifactorial.Althoughurolithiasisisknowntohavenumerouspredisposing etiology factors, but exact mechanism of stone formation and growth is not fully known [13].Urinarycalculiformationusuallyresultsfromacombinationofvariousphysiological,nutritionalandmanagementa l factors [3]. It may occur due to excessive or imbalanced intake of minerals in feedlots whilefattening cattle receive rations high in cereal grain and oil meals [7]. Ingestion of certain plants containing highlevels of oxalate, estrogen or silica. Diets high in magnesium content. Increased presence of mucoprotein in thediet caused by feeding high concentrate low roughage rations, pelleted rations, or rations high in phosphate.Concentrated urine, which is produced when there is no drinking water available or when water is of poorquality. Water deprivation can be exacerbated by heavy fluid loss from in hot conditions. An imbalance in thecalciumtophosphorusratiooftheDiet.Normally,aruminantremovephosphorusfromitsbodybyexcretingit

into saliva and then out through the feces (manure). High grain, low roughage diets decrease the formation ofsaliva,soextraphosphorusmustberemovedfromthebloodbythekidneysandthenexcretedintheurine.Whendietsaret oohighinphosphorus,theurinephosphoruslevelsbecomeexcessivelyhigh,andthephosphorussettlesandconsolidatesi ntostone-likepelletsthatcanbetoolargetopass[1]Obstructiveurolithiasis is found most common in the age group of 0-6 months [11]. Urolithiasis in castrated beef cattle hasbeen reported to be associated with di ethyl stilbestrol (DES) implants. Geographical and seasonal influencesplay an important role for range herds in semi-arid areas. In addition, the anatomy of the male ruminant urinarytract also contributes due to the potential narrowness of the passage and tortuous route. Thedistal end ofsigmoid flexure is a common site for uroliths to lodge in all ruminant species [4; 13; 9 and 11]. Prevalence ofurolithiasis is highest in winter and spring season; struvite (ammonium magnesium phosphate) and calciumphosphatecrystalsarethemostcommontype ofurolithinurolithiasisinbovines[11].

CLINICALSIGNS

The major clinical signs reported in partial obstruction include anorexia, suspended rumination anddecreased water intake. Animals suffering from partial obstruction dribble blood tinged urine after prolonged,painful attempts of urination, as the disease progress the symptoms depicted are abdominal bilateral distention,tenesmus, colic, and weight shifting, and grinding of teeth, urethral pulsation. In case of total obstructionsymptoms are as above but more painful, animals may strain to urinate but are not able to pass any urine andmay have a stretched out stance or go down and tendency of rectal prolapsed. In advance cases the animal mayseem more comfortable again for a while but will develop swelling under the skin of the belly, the sheath, underthe tail, and will gradually deteriorate over a couple of days in case of rupture of the urethra. Less specific signsinclude rectal prolapse, rumen stasis, tachycardia and tachypnea. There is severe damage to the bladder andurethral mucosa by uroliths which leads to haematuria, oligouria and dysuria. Complete urethral obstructionresultsindeathdue touraemia.

Animal may seem more comfortable initially in rupture of the bladder (cystorrhexis), but one to twodays after the rupture its belly will start to fill up with urine (a fluid thrill) on abdominal ballottementis noticedand the animal will become depressed and weak [3]. Then it will go down and die fairly quickly. If there is anobstructionand itisnotrelieved,abladder or urethralrupturewilloccurusually within48 hours.

PATHOGENESIS

Formation of calculi and development of urolithiasis is a complex process and occurs in a series ofphases from formation of nidus, concentration of urine and lastly the precipitation of various salts from urine.Formation of urinary calculi is dependent on super saturation of urine with soluble ionized minerals. Crystalformation occurs when the inhibitory capacity of mucopolysaccharides, ions, and organic acids is exceeded.Factors further predisposingsupersaturation of urine includes increased insensible water loss, urine stasis,increases in urine pH allowing precipitation of phosphate solute, decrease production and secretion of mucusfromthebladder.VitaminAdeficiencyandhighestrogenintake haveallbeenimplicatedasrisk factors.

Calculus composition is directly related to the type of diet; for example silicate urolithiasis is commonwhen the diet is composed of grass hay or cereal grass hays as the majority of the diet. Hay high in oxalatespromotes oxalate stone formation. Diet composed of high calcium containing grains or high legume or cloverhay promotes the formation of calcium carbonate stones. Cereal grains that are typically fed to ruminants andmay make up part of the diet for pet goats are high in phosphorus and lead to an imbalance or inversion of thecalcium/phosphorus ratio,thus causing theformation oftriplephosphatestones.Urolith formation can befurther exacerbated if there is a significant amount of magnesium in the diet leading to magnesium ammoniumphosphate stone formation. The formation of urinary calculi has been evaluated in multiple nutritional studies invarious species and continues to be a significant cause of morbidity and mortality in production food animals aswellastheevergrowing pet ruminantpopulation. The role of nutrition and over conditioning of thepetruminantpopulation hassignificantlyincreased thepresenceofurolithiasisinthispopulation.

LABORATORYFINDINGS

Common routine blood work consisting of a complete blood count (CBC) and serum biochemicalanalysis (SBA) are warranted in the suspected urolithiasis case. The CBC is typically within normal limitshowever, evidence of mild to moderate chronic non regenerative (normocytic normochromic) anemia may bepresent along with evidence of an inflammatory leukogram consisting of increased percent band neutrophils,decreased segmented neutrophils, decreased neutrophil to lymphocyte ratio (1:1), and increased production offibrinogen.TheSBA typically haselevationsinthebloodureanitrogenandcreatinine.Thepresenceofazotemia may be pre-renal, renal, or post-renal. The azotemia can become quite severe in the ruptured case dueto a larger amount of soft tissue for equilibration of the BUN and creatinine [5 and 11].The ruptured bladder orurethraresultsinmoreseverealterationsofthebloodelectrolytes:hyperkalemia,hyponatremia,hypoglycemia,

hyperphosphotemia, and hypochloremia; the presence of the hypochloremia lead to a hypochloremic metabolicalkalosis.Urinalysis and sediment examination should be included as part of the initial examination of thepatient. Marked crystalluria supports the diagnosis of obstructive urolithiasis. Further examination of the urinefor color, clarity, specific gravity; the biochemical evaluation assesses for the presence of ketones, occult blood,urobilirubinogen, pH, glucose, and bilirubin. Alkaline pH of urine favours the formation of phosphate andstruvite calculi [12]. Ultrasonography is very useful imaging technique in the diagnosis of urolithiasis to assesstheurinarybladdercondition[11].

MEDICINALANDSURGICALTREATMENT

If the animal is diagnosed with urolithiasis pursuant of medical therapy depends on the severity of thedisease, the stage of the disease, the nature and extent of the uroliths, the intended long term use of the animal,thefrequencyofthedisease,andmostimportantlythefinancialconstraintsoftheowner[10].Medicalmanagemen t can only be pursued in the affected animal if the bladder wall is intact. In mild cases, the animalscan be treated by using tranquilizers, antispasmodic drugs and litholytic drugs like cystone. Diuresis should notbeusedbeforethe removal ofcalculi.

The treatment of obstructive urolithiasis is primarily by surgical intervention. Recurrent urolithiasis,calculi atmultiple sites, badly damagedurethra, atonic bladder or severe cystitis leads to failure of surgicalrepair in obstructive urolthiasis. Urethrotomy, either post scrotal or post ischial at the site of calculi lodgment iswidely recommended and practiced to relieve the obstruction from sigmoid flexure [3]. Other surgical methodsinclude penile catheterization, bladder fistulation (bladder marsupialisation), intra pelvic cystic catheterization,cystorrhaphyandtubecystotomyasdemandedbyclinicalsituation.Thetechniqueoftubecystotomyisa method of fixation of tube in the urinary bladder for the free passage of urine, followed by chemical dissolutionofcalculiwhichshowsexcellentresults.Cystotomytubesbypassurinaryoutflowobstructionsorasanalternat ive to the urethral catheterization [8]. Tube Cystotomy become popular as a treatment for obstructiveurolithiasis in goats and buffalo calves with subsequent medical dissolution of the urolith [6 and 9]. Severaldifferent types of tubes are available, including Foleys catheters, Mushroom tip catheters and percutaneouscatheters; more recently low profile gastrostomy tubes have been adapted for the use in cystotomy tubes.Cystotomy tubes should remain in place for at least 14 days; before removal to ensure adequate adhesionsbetween the bladder and the body wall to reduce the possibility of urine leakage or peritonitis [14]. Overallsuccess rate with tube cystotomy is around 73.33% and it depend upon post operative care by owners, differenceinthenatureandseverityof lodgment ofcalculi[6 and11]inthemanagementofobstructive urolithiasis.

CONTROLANDPREVENTION

Whetherthepatienthasclinicalevidenceofurolithiasisorapreventionprogramisbeinginstitutedboth strategies must focus on dietary modification and urine acidification. The ration should be modified, whichincludes elimination of alfa-alfa feeding, reduction or elimination of grain feeding, a change to grass hay asprimary forage, encouragement of grazing and ammonium chloride supplementation. Ruminant urine is highlyalkaline favoring formation of calculi. Acidification of urine can increase the solubility of the uroliths and it iscomposed of magnesium ammonium phosphate (struvite), calcium phosphate (apatite), and calcium carbonateand thereby inhibits precipitation in the urine. Urine acidification can be accomplished through the addition ofammonium chloride (Nausadar) salt to the ration at a daily dose of 0.5% to 1% of the total dry matter intake, 2%of concentrate ration, or @ 225– 500mg/kg/b.wt./day [3].The palatability of ammonium chloride is poor makingfree choice feeding difficult to maintain; most times it must be mixed with some form of concentrate feedrememberingthatthistypeoffeedpromotescalculogenesisorsomeformofatreatforconsumption.Alternativelyitca nbemixedintoasugarsolution(mixedwithjaggaryorsyrup)anddrenchedorallyorfrozenin the freezer and fed as a treat. Continuous therapy with ammonium chloride can lead to refractoriness to urineacidification therefore current recommendations are to continue the drug for 3 consecutive weeks and thendiscontinue for 1 week and then start back on the medication. Regular monitoring of the urine pH is necessaryfor monitoring the effectiveness of the drug, with the ideal pH of the urine being <6.5. Dietary management is akey step for dissolution of calculi, prevention, and management of the disease by decreasing the amount ofexcretedsolutesintheurine.Dietarymodificationneedstobecorrelatedtothetypeofcalculipresentandalsoto the area ofthe countrythatthe affectedanimalislocated.

REFERENCES

[1]. Ahmed,A.S.,Amer,H.A.andIbrahim,I.M.InfluenceofdietarymineralimbalanceontheincidenceofurolithiasisinEgyptiancalves. *Arch.Exp.Veterinarmed*,1989.**43(**1**):**73-77.

[2]. Amarpal,Kinjavdekar,P.,Aithal,H.P.,Pawde,A.M.,Tarunbir,Singh,Pratap,K.andSingh,T.Incidenceofurolithiasis:aretrospectivestudyo ffiveyears.*IndianJournalofAnimalSciences*,2004.74**(2)**:175-177.

[3]. Amarpal,Bishnoi,A.K.,Sharma,S.N.andSingh,Jit.In:RuminantSurgeryatextbookofthesurgicaldiseasesofcattle,buffalo,camel, sheep

andgoat.2nd ed.CBSPublisherand Distributors, NewDelhi.2020:380-392.
[4]. Clotide,E.M.P.,Cappelaro,D.M.,Nobre,DandCampedelli,P.Urolithiasisinbovines.Biologico,Sao.Panlo.1980.**46**:77-80.

[5]. Donecker,J.M.andBellamy,J.E.C.Bloodchemicalabnormalitiesincattlewithrupturedbladdersandrupturedurethras.*Can.Vet. J.*,1982.**33**:355-357.

[6]. Ewoldt,J.M.,Anderson,D.E.,Miesner,M.D.andSaville,W.Shortandlongtermoutcomeandfactorpredictingsurvivalaftersurgicaltube Cystotomyforthetreatment ofobstructiveurolithiasisin smallruminants.*Vet.Surg*.,2006.**35**:417-422.

[7]. Hesse,A.T.,Tiselius,H.G.andSiener,R.UrinaryStones,Diagnosis,TreatmentandPreventionofrecurrence.**3rd**edn.Basel.S.KargerAG.,20 09.ISBN978-3-8056-9149-9.

[8]. Kalim,M.O,Zaman,R.andTiwari,S.K.SurgicalManagementofObstructiveUrolithiasisinamaleCowcalf.*Vet.World*,2011. **4(**5**)**:213-214.

[9]. Kushwaha, R.B., Amarpal, Aithal, H.P., Kinjavdekar, P. and Pwde, A.M. Clinical appraisal of 48 cases of obstructive urolithiasis inbuffalocalvestreatedwithtubeCystotomyand Urethrotomy.*Adv.Anim.Vet.Sci*.2014.**2(2):**106-110.

[10]. Larson,B.L.Identifying,treating,andpreventingbovineurolithiasis.*Vet.Med*.,1996.**91(4):**366-377.

[11]. Mahajan,A.,Gupta,A.K.,Bhadwal,M.S,Bhat,M.A.andBhardwaj,H.R.OccurrenceandmanagementofobstructiveurolithiasisinRuminan ts.*JournalofAnimalResearch*,2017.**7(4**):723-731.

[12]. Pugh,D.G. In:SheepandGoatMedicine,1st ed.ElsevierPublishingCo.Philadelphia,U.S.A.,2002.

[13]. Radostitis, O. M., Blood, D.C., Gray, G.C. and Hinchcliff, K.W. Veterinary Medicine a text book of the disease of cattle, sheep, pig,goat andhorse.BailloereTindells,London,2009.

[14]. Singh,K.,Gopinathan,A.,Shakya,P.,Bodh,D.andSangeetha,P.EfficacyoftubeCystotomyfortreatmentofobstructiveurolithiasisingoatsw ithintact UB.*Ind.J.SmallRumin*.2014.**20(2**):74-78.