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SYSTEMATIC REVIEW OF CONGENITAL ANOMALIES IN CALVES AND KIDS REPORTED DURING THE PERIOD FROM 1975 TO 2021 IN BANGLADESH

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ABSTRACT

Background: Congenital malformations are structural or functional anomalies that occur during intrauterine life and can be identified prenatally, at birth or sometimes only later during life. These congenital defects can evolve from abnormal genetic or environmental factors or a combination of both but the causes of many cases are unknown. The risk factors are associated with congenital defects varies depending on species, breed, body system, geographical location and season. Some congenital malformations can cause death of newborn, while others can only create a structural defect without affecting vital functions and responsible for slowing down of genetic progress and economic loss for the breeders. Congenital defects constitute the at least fifth largest cause of neonatal morbidity and mortality but national estimate of the prevalence of these anomalies are lacking in inland literature. Thus, it is important to estimate the prevalence with identify the causes and risk factors of congenital defects for their possible prevention to minimize the economic losses in animal industry.

Objective: The objective of the review was to derive an estimate of the birth prevalence and associated host risk factors of congenital anomalies reported in calves and kids in Bangladesh.

Materials and Methods: The occurrence of congenital anomalies in new born farm animals has been reviewed from the first report published in 1975 up to 2021 in Bangladesh. The search was carried out in Google, PubMed, Banglajol and also the original inland journals.

Results: All types of search identified 33 inland articles and 35 articles published from foreign nations. Of the 33 available inland articles of which most of the articles based on either single clinical report or data abstracted from veterinary hospital records. Approximately 36 types of congenital malformations were recorded, of which 31 types in cow calves and only five types in kids. Higher occurrence of congenital defects reported in calves (96.95%; n = 1746 cases) than kids (3.05%; 55 cases), but both the calves (56.25%; n = 923) and kids (67.27%; n = 37 cases) affected with atresia ani comparatively higher than other reported congenital malformations. System-wise analysis on prevalence of congenital anomalies found highest prevalence in digestive system (67.18%), followed by ocular system (17.07%), musculoskeletal system (8.42%), urogenital system (2.58%), integument system (2.52%) and others. Comparatively higher prevalence of congenital defects reported in male (65.57%) than female (34.43%) and crossbred (67.03%) than indigenous (32.97%) calves in Bangladesh.

Conclusion: Calves and kids born with various congenital defects in their different body systems with highest occurrence of atresia ani in both calves and kids have been recorded. Calves have a great variety of defects than kids probably due to genetic causes. Atresia ani and ocular dermoids are the most frequent occurrence but several cases are still not reported due to lack of disease monitoring system, leading to an underestimation of the real weight of congenital malformations especially in cross-bred cattle. Although some congenital defects can be corrected surgically, the majority cases may not be easily corrected surgically moreover surgically corrected animals may not be used for breeding purposes. Therefore, prevention of the occurrence of congenital defects is required based on the identification of causal factors.

Keywords: Review, Congenital anomalies, Calves, Kids, System-wise defects, Host risk factors, Bangladesh

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INTRODUCTION

The hereditary (all genetic) and/or genetic and/or congenital (not all genetic) disorder terms are often confused and used interchangeably. The genetic disease is a condition which occurs due to an abnormality in the individuals genome while a hereditary disease is condition caused by mutation of a gene can be characteristically transmitted from one generation to another. A congenital defect is a malformation present at birth that may be either inherited or arise as a result of environmental factors. Congenital disorders are structural or functional abnormalities present at birth which are of two types: structural and functional disorders. Structural disorders are abnormalities with the shape of the body or a part of the body, whereas functional disorders include metabolic and degenerative disorders. Congenital disorders also result from genetic and chromosomal disorders. Accordingly, genetic disorders are hereditary but not all congenital disorders are hereditary. Therefore, congenital defects are abnormalities of structure or function present at birth and may account for a high percentage of losses of newborn from just before to just after births. Embryonic and fetal development are the result of a complex series of orchestrated events and errors in the sequential steps of development may result embryonic loss, fetal death, fetal mummification, abortion, stillbirth, birth of nonviable neonates and birth of viable offspring with defects.[1] When a developmental disruption results in a deviation from normal that is present and apparent at birth, the defect is said to be congenital.[1] Technologic advances in the field of teratology have identified an increasing number of specific genetic, environmental and infectious agents as etiologic determinants of certain cases of defective fetal development. Teratogens are agents or factors that cause development of physical defects in the embryo or fetus.[1] The congenital malformations may be caused by genetic or environmental factors or a combination of both but the causes of many cases are unknown. These defects can result from either defective gene or from a genetic insult or agent that's associated with the fetal environment or from their interaction and these defects are classified as lethal, sub-lethal and non-lethal.[2] Specific causes may be difficult to identify but often follow seasonal patterns associated with growth characteristics of toxic plants or availability of suitable vectors of arthropod-borne viruses. Although congenital defects may follow maternal disease due to plant intoxication or viral infection but such defect sometimes may occur in the absence of clinical signs in the dam. The sporadic occurrence of congenital defects in calves[3-10] and kids[11-14] during the twentieth century have been reviewed in the livestock research report published from Bangladesh.[15] In addition to these research reports on congenital defects recorded in this review, a considerable number of research reports on congenital anomalies in calves and kids have been published during the period from 2001 to 2021 in Bangladesh. Therefore, this review describes an overall up-to-date analysis of the available research reports on congenital defects in calves and kids published from Bangladesh.

MATERIALS AND METHODS

The research reports on the occurrence of congenital anomalies in newborn domestic ruminants published from Bangladesh during the twentieth century have been reviewed in an earlier report.[15] In addition, the research reports on congenital malformations in cow calves and goat kids published during the period from 2001 to 2021 from Bangladesh have been reviewed

from Google search and original journals in which these articles published. A total of 33 inland research articles published in both inland and elsewhere and 35 articles published from foreign countries have been reviewed and analyzed.

RESULTS AND DISCUSSION

Congenital defects specially caused by genetic defect represent a hidden danger for animal production. These malformations are responsible for economic losses either because they reduce the productivity or because their spread in the population would decrease the productivity of that species or breed. The occurrences of congenital defects in cow calves and goat kids in Bangladesh during the period from 1975 to 2021 are presented in Table 1 and Table 2, respectively.

Table 1. Congenital defects in calves during the period from 1975 to 2021

SN	Types of defects	Calves No. (%)	Ref. No.	Photo presented	District
A. Digestive system					
①	Atresia ani				
	Atresia ani	28 (01.60)	16	No	Mymensingh
	Atresia ani	08 (00.46)	8	No	Mymensingh
	Atresia ani	87 (04.98)	17	No	Mymensingh & Patuakhali
	Atresia ani	36 (02.06)	18	No	Mymensingh
	Atresia ani	693 (39.69)	19	No	Bangladesh
	Atresia ani	15 (00.86)	20	No	Mymensingh & Rajshahi
	Atresia ani	17 (00.97)	21	No	11+ districts
	Atresia ani	01 (00.06)	22	No	Dinajpur
	Atresia ani (Photo 1)	21 (01.20)	23	Yes	11 districts
	Atresia ani	15 (0086)	24	No	Khulna, Satkhira & Bagherhat
	Atresia ani	02 (00.11)	25	No	Sirajgonj
	Sub-total	**923 (56.25)**			
②	Atresia ani et recti	39 (02.23)	17	No	Mymensingh & Patuakhali
	Atresia ani et recti	05 (00.29)	20	No	Mymensingh & Rajshahi
	Sub-total	**44 (02.52)**			
③	Umbilical hernia (Photo 2)	03 (00.17)	8	No	Mymensingh
	Umbilical hernia	25 (01.43)	20	No	Mymensingh & Rajshahi
	Umbilical hernia	66 (03.78)	16	No	Mymensingh
	Umbilical hernia	31(01.78)	17	No	Mymensingh & Patuakhali
	Umbilical hernia	39 (02.23)	24	No	Khulna, Satkhira & Bagherhat
	Sub-total	**164 (09.39)**			
④	Cleft palate	19 (01.09)	17	No	Mymensingh & Patuakhali
⑤	Epulis	17 (00.97)	17	No	Mymensingh & Patuakhali
⑥	Gum carcinoma (Photo 3)	02 (00.11)	23,26	No	Mymensingh & 11 districts
⑦	Tongue deformity (Photo 4)	04 (00.23)	21,23	Yes	11 districts & 11+ districts
	Sub-total	**42 (02.41)**			
	Total (Digestive system)	**1173 (67.18)**			
B. Musculo-skeletal system					
①	Polymelia (accessory limbs)	17 (00.97)	17	No	Mymensingh & Patuakhali
	Pygomelia (Photo 5)	01(00.06)	27	Yes	Chittagong

Table 1. Congenital defects in calves (Contd.)

SN	Types of defects	Calves No. (%)	Ref. No.	Photo presented	District
②	Bent leg (Photo 6)	10 (00.57)	21,23	Yes	11 districts & 11+ districts
	Knuckling of the limbs	12 (00.69)	16	No	Mymensingh
	Knuckling of the fetlock	01 (00.06)	24	No	Khulna, Satkhira & Bagherhat
	Sub-total	**41 (02.35)**			
③	Contracted tendon	11 (00.63)	8	No	Mymensingh
	Contracted tendon	04 (00.23)	20	No	Mymensingh & Rajshahi
	Sub-total	**15 (00.86)**			
④	CML & ankylosis of limbs	48 (02.75)	17	No	Mymensingh & Patuakhali
⑤	Schistosomus reflexus	02 (00.11)	24	No	Khulna, Satkhira & Bagherhat
	Schistosomus reflexus	35 (02.00)	17	No	Mymensingh & Patuakhali
	Schistosomus reflexus (Photo 7)	01(00.06)	28	Yes	Chittagong
	Sub-total	**38 (02.18)**			
⑥	Scoliosis & spondylolisthesis	05 (00.29)	17	No	Mymensingh & Patuakhali
	Total (Musculo-skeletal system)	**147 (08.42)**			
	C. Ocular system				
①	Neonatal blindness (Photo 8)	05 (00.29)	23	Yes	11 districts
	Neonatal blindness	01 (00.06)	21	No	11+ districts
	Sub-total	**06 (00.34)**			
②	Dermoid cyst (bilateral) (Photo 9)	01(00.06)	29	Yes	Mymensingh .
	Dermoid cyst (Photo 10)	03 (00.17)	23	Yes	11 districts
	Dermoid cyst	19 (01.09)	17	No	Mymensingh & Patuakhali
	Dermoid cyst	198 (11.34)	19	No	BD?
	Dermoid cyst (ocular)	13 (00.74)	20	No	Mymensingh & Rajshahi
	Ocular dermoids	54 (03.09)	30	No	Mymensingh
	Dermoid cyst (ocular)	03 (00.17)	21	No	11+ districts
	Dermoid cyst	01 (00.06)	24	No	Khulna, Satkhira & Bagherhat
	Sub-total	**292 (16.72)**			
	Total (Ocular system)	**298 (17.07)**			
	D. Integument system				
①	AHF & DHK	11 (00.63)	17	No	Mymensingh & Patuakhali
②	Congenital skin outgrowth (CSO)	18 (01.03)	17	No	Mymensingh & Patuakhali
	CSO (Photo 11)	01 (00.06)	21	No	11+ districts
	CSO (Photo 12)	01 (00.06)	23	Yes	11 districts
	Sub-total	**20 (01.15)**			
③	Inherited congenital hypotrichosis	01 (00.06)	23	Yes	11 districts
	Inherited congenital hypotrichosis	01 (00.06)	21	No	11+ districts
	Sub-total (Photo 13 & Photo 14)[26]	**02 (00.11)**			
④	Epitheliogenesis imperfect (EI)	06 (00.34)	10	Yes	Mymensingh
	EI (Photo 15 & Photo 16)	05 (00.29)	26	Yes	Mymensingh
	Sub-total	**11 (00.70)**			
	Total	**44 (02.52)**			

Table 1. Congenital defects in calves (Contd.)

SN	Types of defects	Calves No. (%)	Ref. No.	Photo presented	District
E. Nervous system / Craniofacial abnormalities					
①	Hydrocephalus	14 (00.80)	17	No	Mymensingh & Patuakhali
	Hydrocephalus	01 (00.06)	24	No	Khulna, Satkhira & Bagherhat
	Sub-total	**15 (00.86)**			
②	Neonatal neck paralysis	01 (00.06)	23	Yes	11 districts
	Neonatal neck paralysis	01 (00.06)	21	No	11+ districts
	Sub-total	**02 (00.11)**			
	Total	**17 (00.97)**			
F. Urogenital system					
①	Aplasia in female genitals	07 (00.40)	17	No	Mymensingh & Patuakhali
②	Congenital Urinary tract obstruction	14 (00.80)	17	No	Mymensingh & Patuakhali
③	Cryptorchidism	05 (00.29)	17	No	Mymensingh & Patuakhali
④	Hypoplasia of testes & scrotum	09 (00.52)	17	No	Mymensingh & Patuakhali
⑤	Persistent urachus	06 (00.34)	17	No	Mymensingh & Patuakhali
	Persistent urachus	04 (00.23)	20	No	Mymensingh & Rajshahi
	Sub-total	**10 (00.57)**			
	Total (Urogenital system)	**45 (02.58)**			
G. Dual system					
①	Recto-vaginal fistula (Photo 17)	07 (00.40)	17	No	Mymensingh & Patuakhali
	Recto-vaginal fistula	06 (00.34)	20	No	Mymensingh & Rajshahi
	Sub-total	**13 (00.74)**			
②	Supernumerary limb (Photo 18)	01(00.06)	6	Yes	Mymensingh
	Total	**14 (00.80)**			
H. General anomalies					
①	Ascites	03 (00.17)	17	No	Mymensingh & Patuakhali
②	Hydrothorax	05 (00.29)	17	No	Mymensingh & Patuakhali
	Total	**08 (00.46)**			
	Overall	**1746**			

CML = Contracture of muscles, ligaments AHF & DHK = Abnormal hoof formation and defects of hoods' keratin. 11 districts = Rangpur, Dinajpur, Barishal, Chittagong, Mymensingh, Noakhali, Khulna, Gazipur, Sunamgonj, Tangail & Cox's Bazar. 11+ districts = Dinajpur, Thakurgaon, Gopalgonj, Mymensingh, Chittagong, Noakhali, Gazipur, Sathkhira, Sunamgonj, Tangail and Jhalakati

Most of the published research reports on the congenital anomalies in the newborn animals are based on either the data collected from the veterinary hospital records or studied as clinical cases by the veterinary clinician at the hospitals. However, animal farmers are usually brought newborn animals affected with congenital defects at the hospital those are alive but not reported dead such cases. The actual prevalence rate of congenital anomalies cannot be either determined at the population level where animals are maintained at smallholder farmers at rural levels or based on data maintained all the clinical cases in the hospital patient registered book. However, an overall 0.51% (54 cases out of 10509 all types of clinical cases of cattle) prevalence of congenital defects based on hospital records has been reported earlier from

Bangladesh.[26] Similarly 0.08 to 6.9% prevalence of congenital defects in calves of different breeds and geographical location have been reported elsewhere.[2] Therefore, the prevalence of the different congenital defects in neonatal calves and kids has been analyzed on the basis of total number of neonatal animals affected with congenital defects reported in the different published reports.

Analysis on the occurrence of congenital defects based on different body systems of affected neonatal animals showed significantly highest prevalence rate in digestive system (67.18%) in comparison to ocular system (17.07%), musculoskeletal system (8.42%), urogenital system (2.58%), integumentary system (2.52%) and nervous system (0.97%) of calves in Bangladesh (Table 1). These findings support with the 36.6% highest prevalence of congenital defects affected with digestive system in calves has been reported from India.[31] However, higher involvement of musculoskeletal and nervous systems than digestive and uro-genital systems have been reported in developed nation.[2] These variations on the prevalence of congenital malformations in different body systems between developing and developed countries might be due to differences of animal breeding program and management of environmental risk factors.

Table 2. Occurrence of congenital defects in Black Bengal goat kids

SN	Types of defects	Kids affected No. (%)	Ref. No.	Photo presented	District
A. Digestive system					
①	Atresia ani (male = M)	02 (03.63)	20	No	Mymensingh & Rajshahi
	Atresia ani	04 (07.27)	23	No	11 districts
	Atresia ani	03 (05.45)	21	No	11+ districts
	Atresia ani	28 (50.91)	24	No	Khulna, Satkhira & Bagherhat
	Sub-total	**37 (67.27)**			
②	Umbilical hernia	11 (20.00)	24	No	Khulna, Satkhira & Bagherhat
	Total (Digestive system)	48 (87.27)			
B. Ocular system					
①	Dermoid cyst	03 (05.46)	24	No	Khulna, Satkhira & Bagherhat
C. Urinary system					
①	Urethral diverticulum (M)	02 (03.64)	20	No	Mymensingh & Rajshahi
D. Nervous system					
①	Double monster	01 (01.82)	12	Yes	Mymensingh
②	Monster kid	01 (01.82)	14	Yes	Chittagong
	Sub-total	**02 (03.63)**			
	Overall	**55**			

11 districts = Rangpur, Dinajpur, Barishal, Chittagong, Mymensingh, Noakhali, Khulna, Gazipur, Sunamgonj, Tangail and Cox's Bazar 11+ districts = Dinajpur, Thakurgaon, Gopalgonj, Mymensingh, Chittagong, Noakhali, Gazipur, Sathkhira, Sunamgonj, Tangail and Jhalakati

Review of the comparative analysis on the overall occurrence of congenital anomalies between cow calves and goat kids found extremely higher occurrence of congenital defects in calves (n = 1746; 96.95%) in comparison to kids (n = 55; 3.05%) (Table 1 and 2). This

observation supports the higher overall occurrence of congenital malformations in calves (0.80%) than kids (0.21%) reported earlier in an individual report from Bangladesh.[23] In addition, congenital defects have been recorded in eight body system in calves whereas only five systems of goats affected with congenital malformations (Table 1 and Table 2).

The higher rate of occurrence of congenital anomalies in calves than goat kids relates with the existing breeding method and program of cattle and goats in Bangladesh. Cattle cross-breeding program has been lunched in Bangladesh since 1970s and currently approximately 33.66% indigenous cattle exist.[32] Within the existing cattle breeding services with AI, dairy farmers have little or no idea of the merit and quality of the semen being offered by the breeding service providers. In addition to locally produced semen, importation of inappropriate genetic material with inappropriate and indiscriminate cross-breeding program between unrelated genetic materials without any national cattle breeding policy might have been thought to be the major cause of genetic erosion of indigenous cattle associated with high occurrence of congenital anomalies in neonatal calves. Whereas approximately 90% goat population is indigenous Black Bengal breed of which 83 to 92% goat farmers are usually used village buck for natural breeding service to their goats and only 17% farmers uses AI system which are very limited in some research station for breeding goats in Bangladesh.[33] As a result chance of genetic erosion and occurrence of congenital anomalies is very limited in goat kids in Bangladesh.

Host risk factors of congenital defects in calves

The classification of diseases, even congenital anomalies can help to ferret out an important, frequently causal relationship. The most widely used classification of disease are ① topographic, by bodily region or system, ② anatomic, by organ and tissue, ③ physiological, by function or effect, ④ pathological, by the nature of the disease process, ⑤ etiologic (causal), ⑥ juristic, by speed of advent of death, ⑦ epidemiological and ⑧ statistical. Any single disease or anomaly may fall within several of these classifications. In the topographical classification, diseases or anomalies can be subdivided into different systems like digestive system, musculo-skeletal system and others (Table 3). Congenital anomalies comprise a wide range of abnormalities of body structure or function that are present at birth and are of prenatal origin. For efficiency and practicality, the focus is commonly on major structural anomalies (Table 3).

Table 3 presents higher prevalence of congenital anomalies in male (65.57%) than female (34.43%) calves and cross-bred (67.03%) than indigenous (32.97%) calves. These findings support the earlier report on higher prevalence of congenital anomalies in male (70.7%) than female (29.3%) calves in India.[31] Higher prevalence of congenital malformations in cross-bred (67.03%) than indigenous (32.97%) claves recorded in this study are in confirmatory of earlier reports.[26] However, the 5:2 ratio of the occurrence of atresia ani between male and female and higher prevalence in indigenous (71.74%) than crossbred (28.26%) calves have also been reported.[34]

Description of some bovine congenital anomalies in Bangladesh

Congenital malformations involving the digestive tract (mouth to anus) can be broadly divided into upper part (mouth and head regions) and lower part (anus) abnormalities. Four cases of congenital cleft lip (harelip) in cross-bred calves in which unilateral (Photo 19) as well

Table 3. Some host risk factors on the occurrence of congenital defects in calves

SN	Body system-wise congenital defects	Gender of calves			Breed of calves		Photo presented	Ref. No.
		Male	Female	Total	Cross	Local		
A.	**Digestive system**							
①	Cleft lip (Photo 19 &20)	03 (01.10)	01 (00.37)	04 (01.47)	04 (01.47)	0	Yes	26
②	Gum carcinoma(Photo 3)	01(00.37)	01 (00.37)	02 (00.73)	02 (00.73)	0	Yes	26
③	Atresia ani (AA)	09 (03.30)	03 (01.10)	12 (04.40)	12 (04.40)	0	Yes	26
	Atresia ani (Photo 1)	19 (06.96)	09 (03.30)	28 (10.26)	10 (03.66)	18 (06.59)	No	16
	Atresia ani	20 (07.33)	08 (02.93)	28 (10.26)	06 (02.19)	22 (08.06)	No	34
	Sub-total	**48 (17.58)**	**20 (07.33)**	**68 (24.91)**	**28 (10.49)**	**40 (14.65)**		
④	Atresia ani et recti	06 (02.19)	02 (00.73)	08 (02.93)	02 (00.73)	06 (02.19)	No	34
⑤	Rectovaginal fistula (Photo 17)	00	01 (00.37)	01 (00.37)	01 (00.37)	00	No	34
⑥	Atresia ani et double urethra	00	01(00.37)	01 (00.37)	01 (00.37)	00	No	34
⑦	Atresia ani et vulvi	00	01(00.37)	01 (00.37)	01 (00.37)	00	No	34
⑧	AA et recti without urethra	00	01(00.37)	01 (00.37)	01 (00.37)	00	No	34
⑨	Atresia ani et two tails	01 (00.37)	00	01 (00.37)	00	01 (00.37)	No	34
⑩	AA with recto-vaginal fistula	00	01 (00.37)	01 (00.37)	01 (00.37)	0	Yes	26
⑪	Umbilical hernia	01 (00.37)	00	01 (00.37)	01 (00.37)	0	No	26
	Umbilical hernia (Photo 2)	49 (17.95)	17 (06.23)	66 (24.18)	32 (11.72)	34 (12.45)	No	16
	Sub-total	**50 (18.32)**	**17 (06.23)**	**67 (24.54)**	**33 (12.09)**	**34 (12.45)**		
	Total (Digestive system)	**109 (39.93)**	**46 (16.85)**	**155 (56.78)**	**74 (27.11)**	**81 (29.67)**		
B.	**Ocular system**							
①	Ocular dermoid cyst (Photo 9)	01(00.37)	01 (00.37)	02 (00.73)	02 (00.73)	0	No	26
	Ocular dermoids (Photo 10)	30 (10.99)	24 (08.79)	54 (19.78)	54 (19.78)	0	No	30
	Ocular dermoids	05 (01.83)	03 (01.10)	08 (02.93)	04 (01.47)	04 (01.47)	No	16
	Sub-total	**36 (13.19)**	**28 (10.26)**	**64 (23.44)**	**60 (21.98)**	**04 (01.47)**		
②	Congen blindness(Photo 21)	01 (00.37)	01 (00.37)	02 (00.73)	02 (00.73)	0	Yes	26
	Total (Ocular system)	**37 (13.55)**	**29 (10.62)**	**66 (24.18)**	**62 (22.71)**	**04 (01.47)**		
C.	**Integument system**							
①	Congenital skin outgrowth	01 (00.37)	01 (00.37)	02 (00.73)	02 (00.73)	0	Yes	26
②	Congenital hypotrichosis	01 (00.37)	03 (01.10)	04 (01.47)	04 (01.47)	0	Yes	26
③	Epitheliogenesis imperfecta	06 (02.19)	05 (01.83)	11 (04.03)	11 (01.87)	0	Yes	26
	Total (Integument system)	**08 (**0.93)	**09 (03.30)**	**17 (06.23)**	**17 (06.23)**	**0**		
D.	**Haematopoietic system**							
①	Congenital perineal hematocele (Photo 22)	01(00.37)	00	01 (00.37)	01(00.37)	0	Yes	26
E.	**Musculoskeletal system**							
①	Congenital rickets (Photo 23,24)	01(00.37)	00	01 (00.37)	01 (00.37)	0	Yes	26
②	Brachygnathia (Photo 25)	01 (00.37)	00	01 (00.37)	01 (00.37)	0	No	26
③	Bowie or Bent legs (Photo 26)	09 (03.30)	05 (01.83)	14 (05.13)	10 (03.66)	0	Yes	16,26
④	Arthrogryposis (Photo 27-30)	05 (01.83)	02 (00.73)	07 (02.56)	07 (02.56)	0	Yes	26
⑤	Shortened legs (Photo 31)	01 (00.37)	01 (00.37)	02 (00.73)	02 (00.73)	0	No	26
	Total (Musculoskeletal)	**17 (06.23)**	**08 (02.93)**	**25 (09.16)**	**21 (07.69)**	**4 (01.47)**		
F.	**Uro-genital system**							
①	Agenesis of urethra	02 (00.73)	00	02 (00.73)	01 (00.37)	1 (00.37)	No	34

Table 3. Some host risk factors on the occurrence of congenital defects in calves (Contd.)

SN	Body system-wise congenital defects	Gender of calves Male	Female	Total	Breed of calves Cross	Local	Photo No.	Ref. No.
G.	**Nervous system**							
0	Hydrocephalus	01 (00.37)	0	01 (00.37)	01 (00.37)	0	No	16
0	Double monster fetus	01 (00.37)	-	01 (00.37)	01 (00.37)	0	Yes	5
	Total (Nervous system)	**02 (00.73)**	**0**	**02 (00.73)**	**02 (00.73)**	**0**		
H.	**General**							
①	Weakness and paresis	03 (01.19)	02 (00.73)	05 (01.83)	05 (01.83)	0	Yes	26
	Overall (%)	**179 (65.57)**	**94 (34.43)**	**273**	**183 (67.03)**	**90 (32.97)**		

as entire involvement of lips (Photo 20) has been recorded in Bangladesh.[26] Modes of inheritance are monogenic autosomal recessive or incomplete dominant in several breeds of cattle. In addition, environmental factors during the early gestation period include hypervitaminosis A, folic acid deficiency and administration of griseofulvin for the treatment of ringworm are considered as risk factors. Gum carcinoma in two neonatal calves (Photo 3) has been reported and the affected calves were not able to suckle milk properly.[11] Post-operative observation recurrence of the similar growth at the site was recorded.[26]

Atresia ani (imperforated anus)

Imperforate anus is a congenital anomaly defined as the failure of development of anal opening of proper size and location. It is a congenital malformation of the anorectum due to the failure of the urorectal fold to divide the cloaca completely or of the failure of the perforation of the fetal anal membrane that divides the rectus and anus during fetal development. As a consequence, the anal opening is closed and feces considered important contributing factors in the pathogenesis of anorectal malformation.[35] Anatomically, atresia ani has been classified into four types, namely I, II, III and IV.[36] Type I atresia ani is defined as a congenital stenosis of a patent anus. Type II has a persistent complete anal membrane alone or a combination of an anal membrane with the rectum ending as a blind pouch cranial to the membrane. Type III has an imperforate anus with rectum terminating further cranially. Type IV has a normal ending of the terminal rectum and anus, while the cranial rectum terminates as a blind pouch within the pelvis. The highest occurrence of the congenital anomalies recorded in the digestive system (67.18%) of calves, of which these calves affected with highest percentage of atresia ani (56.25%) malformation (Table 1).[8,16-22] It is most frequently encountered congenital defect of the lower gastro-intestinal tract reported in mammals[37] especially in calves and kids in Bangladesh (Table 1 & 2). Higher prevalence of atresia ani was recorded in male (17.58%) than female (7.33%) calves, whereas higher in indigenous (14.65%) than crossbred (10.49%) calves (Table 3).[16,26,34] These cases are characterized by the absence of anal opening but bulge at its site.[20] The calves exhibited symptoms of great discomfort making violent efforts to defecate.

Atresia ani et recti

Congenital absence of anal opening and rectum is known as atresia ani et recti in newborn animals. If the rectum ends blindly as a cul-de-sac a short distance cranial to the anal

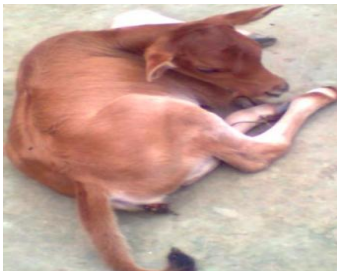

Photo 1. Atresia ani in a crossbred calf.[23]

Photo 2. Umbilical hernia in a calf.[38]

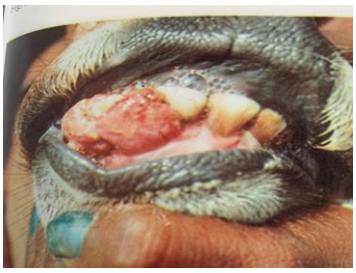

Photo 3. Congenital gum carcinoma in a crossbred calf.[26]

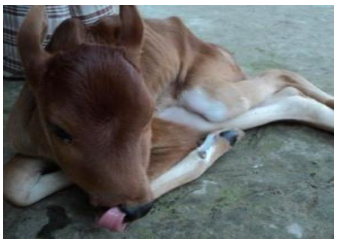

Photo 4. Tongue deformity in a calf.[23]

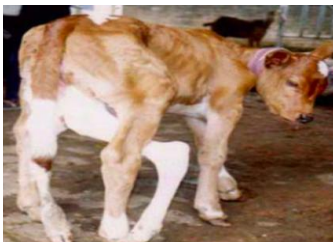

Photo 5. Pygomelia in a cross-bred calf.[27]

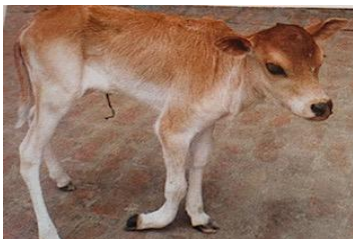

Photo 6. Congenitally elongated right radio-ulna with bent metacarpus of a calf.[26]

Photo 7. Schistosomus reflexus bovine fetus.[28]

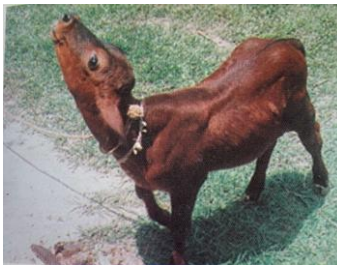

Photo 8. Congenital blindness in calf with encephalopathy signs.[26]

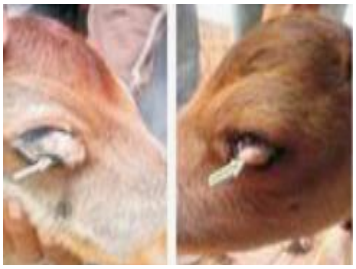

Photo 9. Bilateral dermoid cyst in a calf.[39]

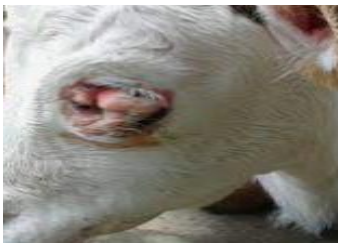

Photo 10. A calf affected with a severe dermoid cyst in the eye.[29]

Photo 11. A neonatal calf congenitally affected with a drum-stick like skin outgrowth on the hump.[26]

Photo 12. A neonatal calf congenitally affected with sac-like skin outgrowth on the pole.[26]

Photo 13. Inherited con-genital hypotrichosis (ICH) affected newborn calf showing absence of hair on the whole body surface.[26]

Photo 14. ICH affected newborn calf showing absence of hair with thickened, wrinkled, greasy and erythematous skin.[26]

Photo 15. A calf affected with epitheliogenesis imperfecta (EI) showing absence of skin layer on all the four legs.[10]

Photo 16. EI affected calf showing epithelial defects on muzzle, lips and palate.[10]

Photo 17. Atresia ani with recto-vaginal fistula in a neonatal heifer calf.[26]

Photo 18. A neonatal calf showing seven legs with three extra supernumerary legs on the back.[40]

Photo 19. Congenital unilateral macrostoma with complete cleft in a neonatal calf.[26]

Photo 20. Cranio-facial anomalies in a crossbred calf with defects of mouth, lips, muzzle and eyes.[26]

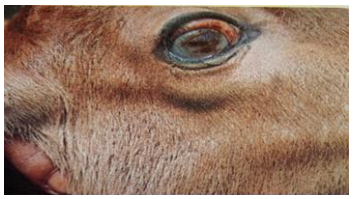

Photo 21. Congenital blindness in a neonatal calf with congestion on the conjunctiva and cornea.[26]

Photo 22. A neonatal calf affected with congenital perineal haematocele.[26]

Photo 23. A neonatal calf affected with congenital rickets showing curvature of all the four legs.[26]

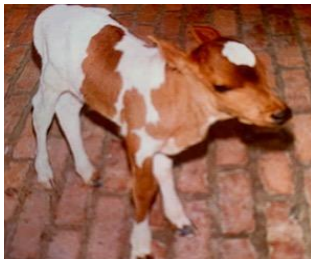

Photo 24. Post-treatment status of congenitally rickets affected calf showing straight all four legs.[26]

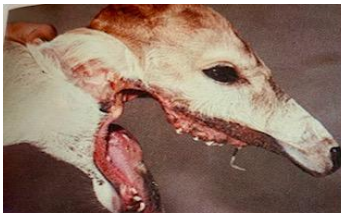

Photo 25. A neonatal calf affected with congenital brachygnathia showing longer upper than lower jaws with defective teeth and oral cavity. [26]

Photo 26. A neonatal calf affected congenitally with bowie or bent leg of both the distal parts of the hind legs.[26]

Photo 27. Arthrogryposis-congenital fixation of cervical vertebrae and left front knee joint of a neonatal calf.[26]

Photo 28. Arthrogryposis-congenital ankylosis of the left hock joint of neonatal calf.[26]

29. Multiple ankylosis of cervical and lower hind limb joints of a neonatal calf.[26]

Photo 30. Arthrogryposis-congenitally bent fetlock joint of all the four limbs of a calf.[26]

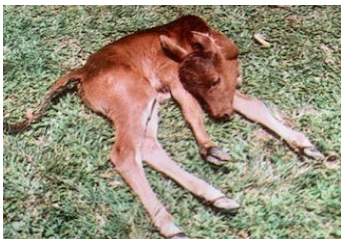

Photo 31. A cross-bred calf with congenitally shortened right fore leg.[26]

Photo 32. A congenitally weak paretic calf showing unable to stand.[26]

Photo 33. A congenitally weak calf showing inability to maintain a normal posture and gait.[26]

Photo 34. A Bhutti breed cow delivered a dwarf heifer calf in Bangladesh which is recorded as the smallest cattle in the world.[67]

Photo 35. Girolando calf showing congenital bifid tongue and changes in the fusion of the mandibula.[41]

Photo 36. A newborn human baby like face delivered by a Black Bengal doe in Assam, India.[42]

membrane, the condition is called rectal atresia or atresia ani et recti. Atresia ani et recti occurs the urorectal fold to fails to divide the cloaca completely or can also occur due to the failure of the fetal anal membrane to breakdown that divides the rectum and anus during fetal development.

Out of 1746 calves recorded with congenital defects, of which only 44 (2.52%) calves affected with atresia ani et recti (Table 1)[17,20] and these cases are characterized by neither anal opening nor bulge at the anal site even with hand pressure on the distended abdomen.[20] Higher prevalence of atresia ani et recti was found in male (2.19%) than female (0.73%) calves, whereas it was higher in indigenous (2.19%) than crossbred (0.73%) calves (Table 3).[34]

Recto-vaginal fistula

Recto-vaginal fistula is an abnormal opening between ventral wall of rectum and dorsal wall of vagina in female calves due to increased fecal pressure inside the rectum due to absence of anal opening (Photo 17). It is considered an embryologic failure of the urorectal septum to separate the cloaca into urethra-vesicle and rectal segments. Of the 1746 calves affected congenitally, of which 13 (00.74%) had defect of dual systems with recto-vaginal fistula anomalies (Table 1).[17,20] A fistulous tract may form between rectum and vagina called recto-vaginal fistula. It appears uncommon in the general population but with a relatively high incidence in certain crossbreds.[17,20,35]

Persistent urachus

Of the 1746 recorded cases of congenital malformations in calves, only10 (00.57%) cases found affected with persistent urachus (Table 1).[17,20] Persistent (patent) urachus is a congenital or acquired defect that results when the embryonic connection (called urachus) between the urinary bladder and allantoic sac of a fetus fails to close after birth causing dribbling of urine from the umbilical region of the newborn.[43] It is frequently accompanied by omphalitis, omphalophlebitis and urachitis with inflammation and infection extending to the abdominal structures. Persistent (pervious, patent) urachus is often conceived to be the result of a failed occlusion due to some congenital defects of the urachus or of the umbilical ring.

Normally the urachus closes and degenerates along the border of the median ligament of the bladder. If it remains open, urine will be excreted from this tube at the umbilicus and the condition is called persistent urachus or urachal fistula.[43]

Umbilical hernia

This study recorded 164 (9.39%) cases of congenital umbilical hernia affected calves (Table 1).[8,16,17,20,24] Congenital umbilical hernia (omphalocele) is the protrusion in a newborn, of the peritoneal sac and eventually of other abdominal organs that are covered by skin and subcutaneous tissue through the abdominal wall at the umbilical level.[44] It is due to failure of the closure of the umbilical ring at birth and its severity varies in relation to the extent of the umbilical defect and the amount of the protruding abdominal organs. This malformation is the most frequently noted congenital disorder in calves[45] and it seems to be associated with genes located at the centromeric end of BTAB.[46] Overall 9.39% prevalence of umbilical hernia in neonatal calves was recorded. However, higher prevalence of this malformation was found in

male (18.32%) than female (6.23%) but no differences was found between crossbred (12.09%) and indigenous (12.45%) calves (Table 3).[16,26] Umbilical hernia is a congenital defect in umbilicus in which eviscerated abdominal organs are covered by amnion rather than skin. Due to improper closing of umbilicus after birth, the abdominal organ particularly intestine protrude through that opening and covered by skin.

Ocular dermoids

Out of 1746 cases of calves affected with congenital defects, of which 298 (17.07%) had ocular dermoids (Table 1).[17,19,20,21,23,29,30] Both the male (13.19%) and female (10.26%) calves affected with ocular dermoids but the higher prevalence recorded in crossbred (21.98%) than indigenous (1.47%) calves in Bangladesh (Table 3).[16,26,30] The clinical cases of either unilateral[23] or bilateral[29] dermoid cysts have been reported. However, analysis of hospital records showed higher prevalence of bilateral (n = 37) than unilateral (n=14) dermoid cysts in calves.[30]

Ocular dermoid is a skin or skin-like appendage usually arising on the limbus, conjunctivae and cornea that they can be unilateral or bilateral and may be associated with ocular or other manifestations. It is due to heritable autosomal recessive and polygenic trait.[47] Hair from the lesions is predominantly responsible for the associated irritation resulting in chronic inflammation of the conjunctivae and cornea and may cause visual impairment.[29,48]

Ocular dermoids are usually congenital or hereditary in nature. They are formed due to defective epidermal closure along embryonic fissures, which isolates an island of ectoderm in the dermis or sub cutis. The cyst usually contains hair, keratin and sebum and these materials may produce progressive enlargement of the structure so that it becomes clinically apparent.[49]

The characteristic appearance of a dermoid is a mass of skin-like tissue that has very coarse hairs emanating from the surface. Dermoid may occur on the conjunctiva, cornea, limbus, eyelids or third eyelid. Limbal dermoids that involve both cornea and adjacent conjunctiva occur much more commonly than pure corneal dermoids. They may have a pendulous appearance or be flat that can be either be very small lesions or be large enough to inhibit eyelid closure. Dermoids are classified either as cystic or solid, based on the number and size of intraepithelial keratinaceous cysts that are common to most dermoids. The hair that grows from the dermoid cause's severe irritation resulting in epiphora, ocular discharge, blepharospasm and corneal ulceration.[50] Ocular dermoid cyst occurs congenitally in several forms leading to corneal erosion and ulceration.

Congenital tendon contracture

The three types of congenital defects first recoded from Bangladesh in 1975.[8] Diagnosed in 11 (00.63%) newly born calves; in all of them the fetlock joint of either left or right front leg was fixed in flexion due to contracture of the flexor tendons. The calves were normal in size and in good physical condition but could not get up unaided nor walk properly. Eight such calves lived more than a year after which they were sacrificed by the owners on humanitarian grounds.[8] However, out of 1746 reported cases, 15 (0.86%) had congenital defects as contracted tendon in calves (Table 1).[8,20] Etiologic factors for the anomalies include inherited factors, in

utero-malpositioning, large size of fetus, arthrogryposis syndrome, toxic food, mineral and vitamin deficiency during pregnancy have been reported elsewhere.[51] Calves having contracted tendon are characterized clinically by unable to walk due to bend legs.[20]

Polymelia

A cow gave birth to a seven-legged calf recently in the Feni district at her 4th parity in Bangladesh (Photo 18). In addition to four normal legs, three extra legs on the back of the calf which may be called notomelia.[40] In addition, out of 1746 cases of congenital anomalies, 17 (0.97%) recorded as polymelia cases (Table 1).[17]

Polymelia is a congenital anomaly which defined as the presence of accessory limbs (supernumerary legs) attached to various body regions and could be classified as cephalomelia (extra limbs attached to the head), notomelia (extra limb attached to the back bone region), thoracomelia (extra limb attached to the thorax region) and pygomelia (extra-limbs attached to the pelvis region), according to the body region the accessary limb is appearing. Amongst polymelia, notomelia is most common whereas pygomelia condition is a rare occurrence in which additional limb is attached in the pelvic region.[52]

A cross-bred (Sindhi × Indigenous) calf born with congenital defect with two accessary hind limbs attached to the pelvic region in between the hind legs which is clinically identified as a congenital anomaly popularly called pygomelia (Photo- 5).[27] The pygomelia affected calf treated successfully with surgical excisions.[27]

Arthrogryposis (Rigid joints)

Arthrogryposis is ankylosis of the limbs, usually combined with a cleft palate and other growth deformities. It is a congenital malformation characterized by non-progressive joint contractures that can affect upper or lower limbs and/or the vertebral column, leading to various degrees of flexion or extension limitations, evident at birth. Arthrogryposis multiplex is a lethal autosomal recessive genetic defect that originated in Angus cattle. It has been reported in livestock and pets and often associated with muscle atrophy or other malformations. It has more than one etiological factor, including physical limitation of in utero movement causing fetal Akinesia / hypokinesia syndrome, maternal illness, intrauterine viral infection (e.g. Akabane virus), toxin exposure and genetic disorders affecting the fetus.[53] Arthrogryposis multiplex congenital has been associated with a genomic deletion encompassing ISG15, HES4 and AGRN genes in the American Angus breed of cattle whereas in Red Dairy cattle it is associated with a deletion in the first exon of CHRNB1.[54] Out of the 1746 cases of congenital defects, 48 (2.75%) calves had contracture of muscles, ligament and ankylosis of limbs (Table 1).[17] This study recorded higher prevalence of arthrogryposis in male (1.83%) than female (0.73%), also higher in crossbred (2.56%) than indigenous (0%) calves (Table 3).[26]

Schistosoma reflexum syndrome

Schistosoma reflexum or schistosomus reflexus (SR) is congenital syndrome briefly characterized by visceral eventration (exposed abdominal and sometimes thoracic, viscera), failure or incomplete closure of abdominal wall and severe dorsoflexion and ankylosis of the spine and arthrogryposis.[55,56]

Out of 1746 cases of congenital anomalies recorded in calves, of which only 38 (2.18%) recorded as Schistosomus reflexus (Table 1).[17,24,28] The Schistosomus reflexus (SR) in calves are clinically characterized by angulation of limbs and no abdominal closure in Bangladesh.[17]

These cases have been diagnosed as SR syndrome but etiology not diagnosed and no genetic analyses has been performed but it has been reported that an autosomal recessive mode of inheritance for SR exists in cattle.[55]

Hydrocephalus

Overall 15 (0.86%) cases of hydrocephalus was recorded in this study (Table 1).[17,24] Hydrocephalus is a multifactorial, congenital or acquired disorder characterized by an abnormal accumulation of cerebrospinal fluid (CSF) within the cranial cavity. When the CSF is accumulated in the ventricles which is called internal hydrocephalus and when the CSF is accumulated in the subarachnoid space which is called external hydrocephalus. The increase of CSF is, above all, related to its abnormal reabsorption or defective lymphatic drainage and rarely to its production and it induces progressive enlargement of the head. Etiology includes-genetic factors, developmental anomalies, intrauterine or prenatal infection, in utero teratogens exposure, dietary deficiencies (vitamin A deficiency in the rabbit) or tumors or bleeding in the brain.[57] Polycephaly is a congenital malformation in which an individual with two or more heads, probably due to partial or total union of two developing embryos or to a partial duplication of a body or to the antero-posterior compression of the embryonic disk.[57,58]

Blindness

Out of 1746 calves affected with congenital defects, only 6 (0.34%) affected with congenital blindness,[21,23] of which only two such calves (one male and one female) were available for host risk factor analysis but both the calves were crossbred (Table 3).[24] Blindness with no other clinical signs in neonatal calves may be caused from a vitamin A deficiency. Vitamin A is important for retinal formation, which is how the brain receives light information and creates images in the brains. Deficiency of vitamin A is well-known nutritional cause of blindness in cattle at any age. It occurs in cattle when plasma and liver levels fall below 0.7 μmol/L (0.2 ppm) and 2 μg/g respectively, and is often due to suboptimal amounts of green forage intake for a prolonged time. Vitamin A deficiency can lead to calf loss from abortion and stillborn calves in pregnant cows. Calves that are born alive from a cow with low vitamin A levels may be blind from micropthalmia or constriction of optic nerve.[59,60]

Inherited congenital hypotrichosis

Only two (0.11%) cases of inherited congenital hypotrichosis in calves have been reported from Bangladesh.[21,23] In addition two more cases of inherited congenital hypotrichosis in calves have also been reported.[26] Bovine hypotrichosis is a congenital condition characterized by partial or complete absence of hair coat (follicles), with or without accompanying developmental defects. Calves are often born with no hair but will grow a short curly coat of hair with age. Skin in affected areas may be scaly and feel thinner than normal. It is a non-lethal defect with a simple autosomal recessive mode of inheritance which is often referred to

in the literature as viable hypotrichosis, congenital hypotrichosis or semi-hairless. Affected calves are prone to environmental stress (cold and wet) and skin infections are more prevalent.

Epitheliogenesis imperfecta

Out of 11 (0.70%) Epitheliogenesis imperfecta (EI) cases recorded in crossbred calves, of which six cases were recorded during 1988 to 1999[10] and five cases recorded during 1999 to 2001[26] periods (Table 1). Of the 11 recorded cases, six calves were male and five were female, whereas all the 11 calves were crossbred (Table 3).

The EI is a rare autosomal recessive skin defect which is clinically characterized by the congenital missing of epithelium of skin and oral mucosa that vary in size and location. Most often, these lesions consist of irregular patches of discontinuity of hair and squamous epithelium of skin usually on the distal extremities. Absence of epithelium over extensive areas of limbs, muzzle, nostrils, tongue, hard palate, cheeks and esophagus have been reported in calves.[61] History of all the 11 cases were the progeny of the same Sahiwal cross bull and semen of this bull was used for artificial insemination for crossbreeding program.[10] The occurrence of EI in Sahiwal cross-bred calves support the earlier report on the occurrence of EI in Sahiwal cross calves elsewhere.[62]

Congenital epulis

A total of 17 (0.97%) cases of epulis have been recorded as a congenital defect in calves (Table 1).[17] Congenital epulis is a benign tumor of the oral cavity, which may lead to mechanical obstruction resulting in respiratory distress and difficulty in feeding.

Bovine dwarfism

Dwarfism as a heritable condition has been reported in many mammals including multiple breeds of cattle including Angus, Brown Swiss, Danish Red, Dexter, Hereford, Holstein, Japanese Brown and Shorthorn breeds.[63] Several types of inherited chondrodysplasia have been reported in cattle which are characterized by systemic skeletal disorders including shortness and deformity of limbs, head and vertebrae. No single gene or mutation is responsible for all reported cases of bovine dwarfism. Bulldog dwarfism of Dexter cattle is caused by two discrete mutations in the aggrecan (ACAN) gene and also described as autosomal recessive, whereas dwarfism in Aberdeen Angus calves is caused by autosomal recessive.

Congenital bovine chondrodysplasia, also known as bulldog calf syndrome, is characterized by disproportionate growth of bones resulting in a shortened and compressed boy, mainly due to reduced length of the spine and the long bones of the limbs. Abnormalities in the collagen type II alpha 1 chain (COL2A1) gene causing bulldog calf syndrome in cattle have been reported.[64]

Dwarf heifer Rani is the smallest in the world in Bangladesh

A Bhutti breed cow delivered a dwarf heifer calf in 2019 at Charigram in the district of Manikganj, Bangladesh and called her as Rani (Queen). The 23-month-old Bhutti or Bhutanese, heifer stands just 51 cm (20 inch) and weight 28kg / 62 lb. (Photo-34). The Bhutti or Bhutanese heifer Rani, which found fame in Bangladesh, died of overeating and gas accumulation in her stomach on August 19, 2021.[65-67]

With a length of 27 inches, a height of 20 inches (51 cm) and a weight of 57 pounds, the 23-

month-old white cow is at least four inches shorter than the current title holder for the world's shortest bovine- Manikyam, a 24 inch (61.1 cm)-tall Vechur cow from India that set the record in 2014.[66]

The Guinness World Record has declared Rani, the Bhutti heifer who was being raised as a farm at Savar in the outskirts Dhaka, as the shortest cow ever, over a month later after her death.[65]

Congenital defects are structural and functional disorders that occur in newborns under the influence of genetic and environmental factors during pregnancy. Environmental or non-genetic causes have the same economic results as genetic causes but are far easier to rectify. Simply correcting the environment will remove the problem. Certain conditions show that an abnormality is likely to be environmental in nature: (a) The abnormality coincided with an environmental factor and was absent upon removal of the factor, (b) The abnormality occurred in groups of non-related individuals, and (c) The symptoms are similar to those of an abnormality known to result from environmental factors.[68]

Genetic abnormalities occur when genes are missing, in excess, mutated or in the wrong location (translocation). A few genes can directly cause an abnormality, however, these are rare. Usually these genes are recessive, meaning two must be present to cause an abnormality. Both parents must be carrier of the gene for a calf to be abnormal. Two will be carriers and one will be normal. Certain conditions show that an abnormality is likely to have a genetic origin: (a) The abnormality is more common in a group of related animals, and (b) The symptoms are similar to those of an abnormality identified through test mattings. Study of an animal's chromosomes using blood samples can identify several genetic defects.[68]

Some rare congenital anomalies reported in calves and kids elsewhere

Bifid tongue, also called glossoschisis is a rare congenital anomaly in any species and is characterized by incomplete fusion of the lateral tongue buds, resulting in a deep groove in the midline of the tongue. Such an important rare congenital anomaly has been reported in Girolando calf with bifid tongue and changes in the fusion of the mandibular (Photo 35)[41]

A goat gave birth to a human-like face in the Gangapur village of Assam's Cachar district on 29 December 2021. The goat gave birth to a deformed hairless baby that looked like human features-eyes, nose and mouth but its ears were like those of a goat. It had only two limbs and no tail (Photo 36).[42] The baby goat died shortly after birth. The goat also gave birth to another baby that was normal and healthy.[42]

The timing of teratogenic exposure influences the eventual outcome. The susceptibility to an injurious environmental or to genetic agents varies with the stage of development and between species but in general susceptibility decreases with advancement of gestation age. The cells resulting from the union of gametes known as zygotes (< 14 days of gestation, period of pre-attachment) are relatively resistant to the effects of most teratogens but they may be affected by chromosomal alterations or aberrations that occur during the process of gametogenesis or fertilization, as well as genetic mutations that may be passed from one or both parents.[1] During the progresses of development of the zygote into the embryo and organogenesis (14-42 days in bovine) susceptibility to environmental teratogens and teratogenic infectious agent increases.

As the fetus ages increase further (> 42 days in bovine), the fetus becomes increasingly resistant to environmental teratogens, except the late-differentiating structures such as the cerebellum, palate and uro-genital system remain at risk well into the fetal period.[1]

Chromosomal abnormalities occurring during gametogenesis or fertilization may result in embryo lethal anomalies or occasionally in abnormal but viable offspring. Chromosomal errors such as trisomy, aging of gametes after suboptimal timing of AI represents chromosomal abnormality leading to errors in embryonic and fetal development. These errors in development and placentation can result in fetal death, abortion, abnormally large or small birth weights or birth of defective neonates and are often associated with dystocia.[1]

Inherited defects resulting from mutant genes present in breeding lines or families have been reported in all breeds of domestic animals. They may be expressed in typical patterns of inheritance such as the common simple autosomal recessive pattern typified by the recently described arthrogryposis multiplex anomaly of Angus cattle.[1] Genetic factors are inherited defects resulting from mutant genes or chromosomal abnormalities are seen in families in typical intergenerational and intra-generational patterns of inheritance such as Schsrosomus reflexus the common simple autosomal recessive e.g. syndactyly in cattle.

Deficiency of monophosphate synthase (DUMPS) is a lethal autosomal recessive trait formerly widely dispersed in Holstein cattle. When breeding of two DUMPS carriers results in a homozygous embryo, apparently normal fertilization and embryonic development is followed by death of the fetus in early gestation. Screening of sires destined for use in AI has successfully reduced the incidence of DUMPS.[1]

Included in the list of recognized environmental teratogens are maternal nutritional deficiencies, teratogenic drugs or chemical exposure, mechanical interferences with the fetus, some viral infections, plant toxins, trace elements, physical agents like irradiation, radiology, hyperthermia, uterine positioning and perhaps pressure during rectal palpation for gestational diagnosis and toxic effects of any kind that dam would be exposed to during the early stage of organogenesis.[1]

Congenital defects and abnormalities are recorded as sporadic cases in Bangladesh and these cases might be associated with genetic and/or environmental factors.[20] Atresia ani, umbilical hernia and dermoid cyst have been recorded in both calves and kids but persistent urachus recorded only in calves and urethral diverticulum only in kids. Treatment of atresia ani by excision of a circular piece of skin facilitates dissection of the blind end of the rectum and its fixation by stay sutures to the skin opening minimizes contamination of the subcutaneous tissue, and ventral colostomy has suggested being an effective treatment for atresia ani et recti.[20]

Feeding of several species of lupines in cattle has resulted in ‘crooked calf disease’ characterized by joint contractures, torticollis, scoliosis or hyposis, cleft palate or combinations of these defects. Prenatal viral infections may be teratogenic in domestic animals except horses. The stage of fetal or embryonic development at the time of exposure determines the type and extent of the anomalies. Viral infection late in gestation may result in fetal infection and sero-conversion without observed clinical signs, while exposure during earlier stages may induce pregnancy loss or induce congenital defects.[1]

Pestivirus infections are teratogenic in many species including cattle and ewes e.g. prenatal infection of Bovine virus diarrhea virus (BVDV) in cattle can cause a variety of congenital defects in survivors including cerebellar hypoplasia, brachygnathia, alopecia, ocular defects, internal hydrocephalus and impaired immunocompetence.[1]

Nutritional factors especially iodine deficiency may cause congenital goiter or cretinism in all species. Copper deficiency is a cause of enzootic ataxia in lambs. Manganese deficiency can result in congenital limb deformities in calves. Vitamin D deficiency may cause neonatal rickets and vitamin A deficiency may cause eye defects or harelip.[1]

CONCLUSIONS

There are 24.7 million cattle and 26.774 million goats in Bangladesh but higher occurrence of congenital malformations has been reported in calves than kids. Cross-breeding program in cattle with exotic bull semen has started since 1970s and cattle are mainly breeding by using AI at national level throughout the country. Although there is a cross-breeding of goats with exotic buck at limited in research station level but not encouraged at national levels to save the only one existing Black Bengal goats of Bangladesh. As a result majority of the congenital malformations have been reported in cross-bred calves. The occurrence of congenital defects in calves may be associated with a slowing of the genetic progress and economic loss for the breeders due to the death of affected calves, damaged to their reproductive ability and reduction of milk production. To avoid this problem, it is necessary not only set up cross-breeding plans that avoid the use of semen of problem bulls but also to know genetic conditions underlying the diseases and exclude the semen of carrier bull from AI. Semen should be purchased reputable breeders, produced by parents who are not known to carry undesirable genes. However, modern genetic technology can greatly speed up the process of identifying which bulls are carriers and provide techniques for elimination of the carrier bulls with defective genes from the breeders used for semen collection. Clinical diagnosis of congenital defects as in animals could be made early as possible and surgical correction of the malformed neonates offer the best chance for survival. Most breed associations have procedures to report congenital anomalies and work with pathologists, geneticists, and molecular biologists to identify emerging genetic defects.

This review has identified the need for future studies using standard definitions and methodology so that the data would be globally comparable. In terms of hospital versus population based surveillance, the analysis suggested the need for establishing population based registries with active surveillance for birth defects and maternal risk exposures from carefully selected populations.

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