Chemical rumenitis and Septicemic Colibacillosis causes death in a Shoka deer calf (*Capreolus capreolus capreolus*)

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Abstract:

In May 2007, a suborder Shoka deer calf about one month of age was found in a preserved area of Arasbaran in the East Azerbaijan province. From an ecological view point, the Arasbaran area is uniquely labelled as a reserve biosphere. he calf had diarrhea that recovered after treatment. During rearing, The calf received pasteurized- homogenized milk from market as well as milk of a goat. After 3 weeks, the animal suddenly showed acute depression, anorexia and hypothermia. Antimicrobial and supportive therapy was ineffective and the calf died 12 hours after showing clinical signs. At necropsy, chemical rumenitis and hemorrhagic enteritis was observed. Pure *Escherichia coli* was isolated from liver, lungs, rumen and small intestine contents. Histopathologic examination revealed severe chemical rumenitis and necrohemorrhagic enteritis. Absence of mother, intake of milk from other species and stress of capture were predisposing factors. Entry and fermentation of milk in rumen provoked chemical rumenitis and stasis of digestive system could be responsible for septicemic Colibacillosis (colisepticemia) and death in this Shoka calf.

Case history

In recent years, increasing human population in residential areas of Arasbaran, changing backwoods, destruction of forests and pastures, increase in and dominance of domestic animals to jungles, irregular hunting with new tools and destroying the eco-systems through the construction of new roads are the main causes that threaten the animal species in the implicit area. For example, not one Shoka deer has been seen in the past 30 years. The mentioned factors not only caused destruction of animal species but also reveal the re-emergence of viral diseases, most of which are zoonosis and are transmissible to man (Clements et al., 2009). Tuberculosis can be easily transmitted from deer to cow and more than 40 agents of infectious disease have been explained which could be transmitted from wild life to human and domestic animals (Bohm et al., 2006; Buxton, 2006; Griffin et al., 2000; Jonson et al., 2008; Menzies, 2008; Sanchez et al., 2009). Most agents of diseases reported in deer were pulmonary worms like *Dictyocaulus* and *Protostrongylus* (Bohm et al., 2006). In the past 50 years applicable studies showed that if the calves were raised in farms, mortality rate will decrease under 20%, otherwise the rate will be 30-50%. The mortality rate in Shoka deer may be changed 10-20% on the farms and 10-90% in the natural condition (Mellor et al., 2004). Now, in New Zealand, more than 2 million deer are raised in 5700 farms and the first deer raising farm was officially established in 1970 (Mellor et al., 2004). Based on unofficial reports from Iran Department of the Environment (D.O.E) our country has more than 2000 yellow and red deer in the preserved ecosystems. On the date 20/5/2007 in Darana village-

in the west of the preserved area of Arasbaran in the East Azerbaijan province-a suborder Shoka deer calf (Capreolus capreolus capreolus), the local and Azeri name was Elik, about one month of age was found. The mentioned deer calf had diarrhea that recovered after treatment and clinical attention. During husbandry the calf received pasteurized-homogenized milk from the market and from a goat bought for this purpose. Unfortunately, after 3 weeks the deer calf suddenly showed acute depression, anorexia and hypothermia. Antimicrobial and supportive treatment included: Enrofluxacin, 0.5 ml/10kg.b.w (Erfan-Daru, Iran), Oxytetracycline tablet, 10 mg/kg.b.w. (Dam-lorarn, Iran), multi-vitamins 5 mL per case (sheep) (Interchemie, Netherlands) and steroidal anti-inflammatory drugs like Dexamethasone were used 2ml SID (Nasr-Fariman, Iran). Oral rehydration solution (ORS) dissolved in water was used for a short period during therapy (Sepideh-Dasht, Iran). The above-mentioned treatments were not effective and the case died 12 hours after the appearance of clinical signs.

Diagnostic testing

Necropsy was done before 6 hours after death, and samples were taken from rumen, omasum, intestines, liver, lungs, adrenal glands, urinary bladder and content of rumen and small intestine. Then the samples were sent for bacteriological study and histopathological investigations. Pure *E. coli* was isolated from organs with standard bacteriological culture. Histopathological examination revealed chemical rumenitis and necrotic and hemorrhagic enteritis (Figure 1,2). Severe hyperemia was observed in lamina properia and submucosa of the rumen. Basement membrane and basal lamina were intact. Swelling and hydropic change were noticed in stratum spinosum. Numerous focal necrosis and pustules were seen in the upper and middle layers of stratum spinosum without any viral inclusion bodies. Microcolonies of bacteria were found in some areas of stratum corneum. Evidence of growth or invasion of fungi like Candida albicans or Mucor were not observed (Figure 3). Omasal microscopic lesions resembling rumenal ones showed inflammation. Abomasal mucosa was hyperemic. Liver showed severe hyperemia, mild hyperplasia of bile ducts and mononuclear cholangitis. In the heart, severe hyperemia was accompanied by multiple small to large hemorrhages (petechiae to ecchymoses). Hyperemia was observed in kidney and adrenal glands too. Extensive hemorrhage was observed in the small intestinal serosa. Tunica muscularis was hyperemic and submucosa and lamina properia were edematous and infiltrated by mononuclear or polymorphonuclear (PMN) cells. Due to necrosis, mucosa fall down and obvious hemorrhagic exudates with bacterial micro colonies and PMNs were observed. In some areas, necrosis was extended to the deep layers of intestinal wall and necrohemorrhagic enteritis developed (Figure 4). Lungs were severely hyperemic and showed mild lymphocytic perivascular cuffing.

Assessment

Among *E.coli* which have been isolated from wild ruminants such as Shoka in Spain, it is possible to conclude from the genus which produce Shiga toxin (STEC) that wild ruminants are a reservoir of this serotype of *E.coli* and could transmit infection to human (Simpson, 2002; Woodbury et al., 2005). In Japan and the USA, this serotype caused intoxication in humans through food (Simpson, 2002).

On the basis of the Woodbury et al. (2005) study, the main cause of death in red deer is nonspecific trauma to both sexes in all ages in husbandry farms. Diarrhea syndrome in calves is ranked fifth in importance in death due to unknown agents. The absence of mother's milk and calf isolated by mother are other reasons for the calf's death (Woodbury et al., 2005). Mis-mothering due to mother's inexperience in making a relationship with her calf, mother's malnutrition at the late gestation, and dystocia could be responsible for mis-mothering, cachexia and eventually death which may occur in sheep, goat, cow and deer (Mellor et al., 2004). Phenotype, shape of udder and teat in the goat, type of received milk and entry to rumen could decrease milk intake and milk fermentation irritated rumenal mucosa and injured it (Mellor et al., 2004).

In the present study, the Shoka deer calf not only did not have this own parents, but also was fed by a species with completely different properties compared with the milk of goat (Reinken et al., 1990).

Figure 1. Internal organs of the Shoka deer calf, the viscera are severely hyperemic.

Figure 2. Necrotic rumenitis in the Shoka deer calf.

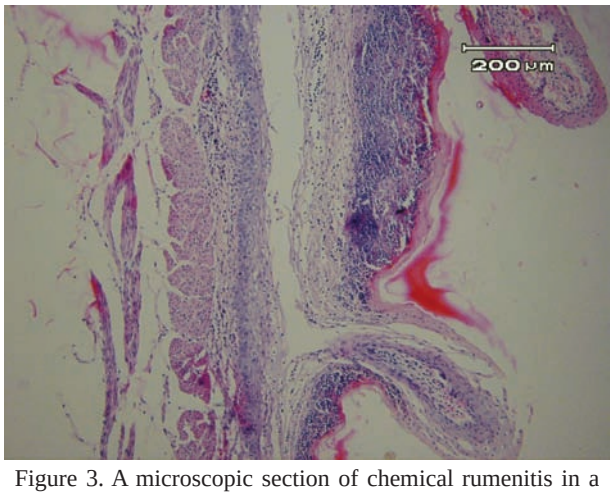

Figure 3. A microscopic section of chemical rumenitis in a Shoka deer calf, severe hyperemia is present in lamina properia and submucosa of the rumen. Basement membrane and basal lamina of stratified epithelium is intact. Swelling and hydropic change in deep layers and pustule formation in upper and middle layers of the stratum spinosum are observed (H&E, × 200).

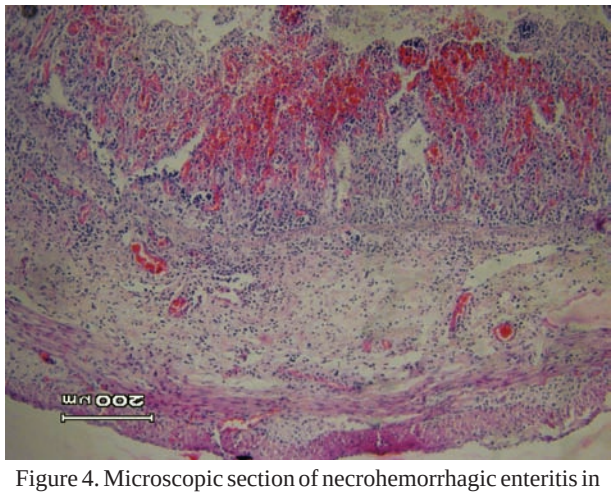

Figure 4. Microscopic section of necrohemorrhagic enteritis in a Shoka deer calf, extensive hemorrhage in the serosa, edema and infiltration of mononuclear or polymorphonuclear (PMN) cells in submucosa and lamina properia are present. Villi are fallen down and obvious hemorrhagic exudates with bacterial microcolonies and PMNs were observed. In some areas, necrosis was extended to the deep layers of intestinal wall (H&E, × 100).

There is one report about allergy in man which appeared due to proteins from milk of goat and sheep which cause eosinophilic esophagitis (Rezazadeh et al., 2009). In addition, Campylobacter jejuni enteritis is found in human after consuming raw milk of goat (Harris et al., 2000; Simpson, 2002). Another report shows the allergy that appeared in human was a cross-action to milk of cow, sheep, wild goat, buffalo, camel and deer (halal meat) (Menzies, 2008). This report shows that the people who had allergies to cow-milk, also have allergies to milk of deer, wild goat and buffalo (Mellor et al., 2004). Mild inflammation of forestomachs could be observed in young calves which used a bucket for milk intake, especially when esophageal groove reflex doesn't work properly, thus a large volume of milk access to rumen and reticulum. A similar problem may occur when the gastric tube is used, so fermented milk could be responsible for mild rumenitis accompanied by edema and infiltration of neutrophils into mucosa (Maxie, 2007). Chemical rumenitis usually occurs after intake of grains or carbohydrates which are rapidly fermented (Maxie, 2007). Hydropic vacuoles, acanthosis and pustule have been discussed as chemical rumenitis due to too many fermentable carbohydrates (Maxie, 2007). The lesions found after intake of fermentable carbo-hydrates include: increased growth of rumenal

papillae, increased cytoplasmic vacuoles in the epithelial tissue (often found in vesicle shape), mild to severe neutrophilic reactions in the mucosa or submucosa, erosion and ulcerations may be found in the mucosa (Maxie, 2007). Rumenal contents seem normal but intestinal contents are usually watery, although it wasn't obvious whether the calf had received colostrum from his mother or not (Rezazadeh et al., 2009). Colostrum deprivation could predispose the appearance of digestive, respiratory and systemic infections which cause diarrhea syndrome, pneumonia, septicemia, inability and eventually death (Mellor et al., 2004). Infection breakout and death depend on the environment of newborns (Mellor et al., 2004).

Many viral diseases cause inflammatory lesions in the forestomachs and digestive system in ruminants. Rumenal necrotic mucosa is found in newborn calves is due to bovine viral herpes 1, bovine papular stomatitis and contagious ecthyma. Rumenal lesion and ulcers may often be accompanied by bovine viral diarrhea and mucosal disease (Maxie, 2007). Severe bleeding and ulcers which were found in the rumenal and omasal mucosa may occur due to bluetongue in sheep. Adenoviral infections often cause multifocal fibrinohemorrhagic rumenitis (Maxie, 2007). No information is available about intake of colostrum so the absence of mother and intake milk from another animal species and the stress of capture could weaken the immune system. In addition to these factors, excessive amount of milk into rumen and chemical rumenitis could predispose growth of pathogens such as *E. coli*. *E. coli* itself may cause hemorrhagic enteritis and eventually death, nevertheless, no virological discovery was found to demonstrate that the virus is responsible for appearance of the disease and eventually, death.

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مجله طب دامی ایران، ۱۳۹۱، دوره ۶، شماره ۳، ۲۰۱-۱۹۷

گزارش مرگ یک رأس گوساله شوکا (*Capreolus capreolus capreolus*) بدنبال التهاب شیمیایی شکمبه و کلی باسیلوز سپتی سمیک

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چکیده

در اردیبهشت ماه ۱۳۸۷ در منطقه حفاظت شده ارسباران در استان آذربایجان شرقی، یک رأس گوساله گوزن زیر گونه شوکا (Shoka deer)، حدوداً یک ماهه شناسایی شد. منطقه ارسباران از نظر اکولوژیکی منطقه ای منحصر بفرد بوده و بعنوان ذخیره گاه زیستکره بشمار می رود. گوساله گوزن مذکور از بدو مشاهده، مبتلا به سندرم اسهال بود که تحت درمان و مراقبت های بالینی قرار گرفته و بهبود یافته بود. در مدت نگه داری، گوساله مذکور از شیر هموژنیزه - پاستوریزه و همچنین شیر یک رأس بزی که به این منظور خریداری شده بود، تغذیه می شد. متأسفانه پس از حدود سه هفته، بطور ناگهانی دچار افسردگی و بی اشتهایی شدید و افت درجه حرارت بدن ($36/5^{o}C$) گردید. اقدامات درمانی از جمله آنتی بیوتیک تراپی، مایع درمانی و استفاده از انواع ویتامین ها موثر واقع نشد و دام ۱۲ ساعت پس از شروع نشانه های بالینی تلف شد. پس از کالبدگشایی، نمونه های مناسب از شکمبه، هزارلا، روده ها، کبد، ریه، قلب، غدد فوق کلیوی، مثانه و محتویات شکمبه و روده ها برای بررسی های میکروب شناسی و آسیب شناسی برداشته شد. از نمونه های ارسالی، با روش های کشت استاندارد، اشریشیاکلی به طور خالص جدا گردید. در کالبدگشایی و مطالعه هیستوپاتولوژیک، التهاب شیمیایی شکمبه و آنتریت نکروتیک هموراژیک جلب توجه می کرد. فقدان مادر، دریافت شیر از گونه حیوانی دیگر و استرس حاصل از اسارت عوامل مستعدکننده بودند. ورود شیر و تخمیر آن در شکمبه، سبب التهاب شیمیایی شکمبه و استاز دستگاه گوارش گشته و علت بروز کلی باسیلوز سپتی سمیک (کلی سپتی سمی) و مرگ گوساله مورد اشاره تشخیص داده شد.

واژه های کلیدی: التهاب شیمیایی شکمبه، آنتریت هموراژیک، اشریشیاکلی، شوکا.

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