Clinical and pathological aspects of osteogenesis imperfecta in newborn Angus calves in Brazil

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Osteogenesis imperfecta (OI) is a genetic disease that affects the connective tissue of bones, tendons, dentin and sclera causing increased bone fragility, joint hyperlaxity, pink teeth, and blue sclerae. Mutations in the genes encoding for type I collagen result in defective formation of this type of collagen, which is the predominant type of collagen in these tissues. OI has been reported in human beings and in a variety of animal species including cattle (in the Charolais, Friesian, Holstein and Holstein-Friesian breeds), sheep, dogs, cats, a llama, a fox, a tiger, and a Rhesus macaque. Transgenic/knockout mice have been used as animal models for this disease. In the State of Nebraska, USA, this disease was observed in Angus calves on one occasion¹, and was presumptively diagnosed in Angus cross calves on another occasion². In Angus/Angus cross calves, however, this condition is not as well characterized as in other bovine breeds in which this disorder is described.

The present report documents the clinical and pathological aspects of a severe form of OI in Angus calves in Brazil. This disease was observed in 12 of 59 newborn purebred Angus calves from a beef herd in the State of Rio Grande do Sul. The calves were derived from 77 unrelated Angus cows all of which were artificially inseminated using semen from the same clinically normal, purebred Red Angus bull born in Brazil. The bull’s dam was derived from an embryo imported from Canada and its sire was also of Canadian origin, the cow having been inseminated with imported semen. Affected calves were observed on the first occasion that semen from the Brazilian bull was used. All clinically affected calves were identified at birth and euthanized 2-20 days after calving. There were also 6 abortions, and 10 animals had bilateral lens opacity since birth. Clinical signs included marked joint laxity, curved hindlimbs, domed forehead, prognathia, inability to stand/walk, and subnormal body size. Post-mortem radiographs of 3 affected calves revealed multiple healing rib fractures, complete long bone fractures, and luxation/subluxation of some limb joints. Necropsy findings included several complete fractures in the axial/appendicular skeleton with hemorrhage/edema of the subcutis/skeletal muscles, multiple bony callouses on the ribs, reduced length of long bones, poorly aligned, fragile, pink incisors, presence of eponychium, and absence of the ligaments and foveae of the femoral heads resulting in hip luxation. Microscopic findings were osteopenia with thin, poorly mineralized trabeculae/mark reduction in the amount of trabecular bone, hypoplastic/hypercellular tendons, and thin dentin. There was also Wallerian degeneration of the spinal cord white matter with axonal spheroid. Immunohistochemistry for BVDV on skin was negative. Semen/blood of the progenitor bull was not available for further investigation. Teratogenic drugs, agrochemical substances, or poisonous plants were not found in this farm. After this diagnosis of OI, the Brazilian bull was removed from artificial insemination. The disease has not been observed in that country since then.

In this study, the diagnosis of OI was based on the history, clinical signs, radiology, necropsy, and histopathology. As in previous reports of OI in cattle/sheep, the inheritance is almost certainly dominant, and the defective gene would have developed as a new germ-line mutation during testicular development of the Brazilian bull rather than having been imported from Canada with genetic material from its sire or dam. This would explain the normal phenotype of this bull and the occurrence of the disease in the offspring of cows unrelated to each other or to the sire/dam.
