Sperm immotility as a cause of infertility in a bull
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A three year old, 800 kg Charolais bull was presented to the Theriogenology Service for a breeding soundness evaluation (BSE) after he failed to produce any pregnancies in a herd of 40 cows and a herd of 30 heifers. The owner claimed that he had purchased the animal with a satisfactory breeding soundness certificate. The bull had never been ill and had been seen to breed many females. During a routine BSE1, > 85% of the bull’s spermatozoa lacked progressive motility. Many spermatozoa were alive and displayed some degree of slow non-progressive motility. Eosin-nigrosin staining confirmed that over 90% of his spermatozoa were alive immediately after collection and that 62% had abnormal midpieces (mitochondrial helix disruptions and abaxially attached flagella).

As the bull was obviously infertile, the client elected to have him slaughtered. The bull’s testes could not be procured, but semen and blood samples were saved for further study. Electron microscopic evaluation revealed spermatozoa with deranged flagellar microtubules and disrupted mitochondrial helix. Further tests identified high incidence of DNA fragmentation detected by TUNEL assay,2 and subcellular defects detected by lectin (PNA and LCA) and ubiquitin labeling.3 Genotyping results are pending, but it is hypothesized that there may be one or more mutations associated with the abnormal maturation of this bull’s spermatozoa.

Sperm immotility has not previously been reported as a congenital defect in bulls. Should this prove to be a genetic defect and a specific mutation can be identified as its cause, other bulls in this pedigree and the breed can be tested. It stands to reason that the defect described here was congenital. Therefore, this case illustrates the grave consequences of an improperly executed BSE prior to the sale of any young bull.

References