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Serum Amyloid A (SAA) Testing to Differentiate Synovial Sepsis from Non-Septic Disease

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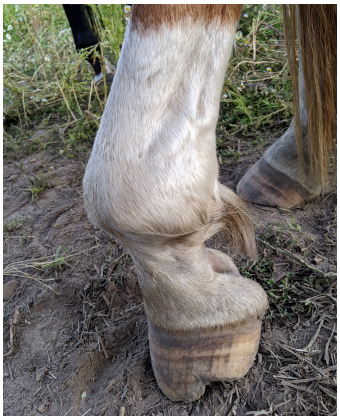
Relevant Species



Early and accurate detection of septic arthritis/synovitus is critical to minimize damage to cartilage and other structures that can ultimately affect the horse's return to function and long-term prognosis. By the time diagnosis is clear cut, a significant amount of damage may be present. Synoviocentesis carries risks, especially when performed in the field, and can complicate standard joint fluid analysis when repeated sampling is required. Additional tools to enhance identification of sepsis and differentiation from non-septic processes are of high value to facilitate appropriate intervention and monitor treatment response.

Blood SAA can be used to identify and monitor synovial sepsis.

In healthy horses with normal joints, serum amyloid A (SAA) is usually undetectable in blood and synovial fluid. With septic pathology however, SAA will increase in both. Interestingly, this increase is often more rapid and dramatic in blood.[1] Blood SAA is also positively correlated with synovial total protein and total nucleated cell count.[2] This allows blood SAA to be used as a surrogate for synovial fluid in many cases, which is particularly advantageous when synoviocentesis is not possible or advisable. Collection of blood is comparatively easy, making it easy to use SAA for monitoring treatment efficacy and resolution of infection, while avoiding the complications of repeated synoviocentesis.[3]

SAA is not elevated with non-septic conditions.

In non-septic synovitis SAA is generally undetectable in both blood and synovial fluid, even when other synovial fluid abnormalities are observed.[1,2,4] Unlike total protein concentration, synovial SAA does not increase significantly with repeated arthrocentesis, through-and-through joint lavage, or with arthroscopy alone.[5,6] It also will not be significantly elevated with low or chronic inflammatory states such as osteoarthritis.[7]

SAA in synovial fluid comes from both the liver and the synovial membrane.

The majority of SAA is produced in the liver, however there is some local production in the synovial membrane.[7] The SAA in synovial fluid results from a combination of both these sources. If blood SAA is elevated due to another systemic inflammatory condition, some systemically produced SAA may filter into the synovial fluid.[4] Infection of other neighboring structures (such as septic osteitis or other septic synovial structures) can also cause a mild increase in synovial SAA.[4] However, barring the presence of other significant disease processes, elevated synovial SAA also supports a diagnosis of septic synovitis.

As with all adjunct diagnostic tests, SAA results must be interpreted in light of other clinical factors and diagnostic results such as joint fluid analysis. Ideally, SAA quantitation should be performed prior to the institution of therapy when possible.[4] If less than 24 hours have passed since the initial injury, SAA in blood and synovial fluid may still be low[3], although some studies have shown an increase in as little as 6-8 hours.[4,7] If SAA concentration is normal and sepsis is still suspected, blood or synovial SAA should be retested 12-18 hours later. Importantly, serial monitoring of blood SAA can be particularly valuable to track clinical progress without subjecting the horse to repeated synoviocentesis.[3]

The VMRD SAA test is fully validated for use with blood, serum, or plasma. Studies with synovial fluid are ongoing–please contact us if you are interested in participating.

References

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