Equine Inflammatory Markers in the Twenty-First Century: A Focus on Serum Amyloid A

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KEYWORDS

• Serum amyloid A • Acute phase protein • Fibrinogen • Inflammation • Equine • Horse

KEY POINTS

- Serum amyloid A (SAA) is the only major acute phase protein in the horse, with concentrations increasing rapidly after an inflammatory stimulus up to high levels compared with baseline, with a subsequent rapid decrease following cessation of the inflammatory process.
- Serum amyloid A is a more sensitive marker of inflammation than other more commonly evaluated laboratory parameters (eg, fibrinogen and white blood cell count).
- Multiple different inflammatory and infectious conditions result in an increase in serum amyloid A levels, including but not limited to colitis, pneumonia, reproductive disease, and septic arthritis.
- Elevations in SAA are not specific for a certain disease process and can increase with inflammation in the absence of infection; elevations in SAA should be evaluated in conjunction with physical examination findings and results of other diagnostic tests.

A continuing challenge faced by veterinary practitioners is early identification of illness, with the goal to initiate early treatment to improve outcome. Patients in early stages of disease can have subtle enough abnormalities that differentiating them from healthy animals is difficult; therefore, sensitive methods to identify diseased patients are desirable. This is particularly true in diseases involving inflammation and infection, where early intervention can affect short- and long-term prognosis. In addition, diagnostic modalities that allow for monitoring of treatment response are extremely useful for guiding therapeutics and assessing prognosis.

Acute phase proteins (APP) are blood proteins synthesized mainly by hepatocytes and are a part of the acute phase response (APR) of the innate immune system.[1–3]

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APP are classified as either negative or positive, depending on whether their serum/plasma levels decrease or increase during an APR, respectively. A notable negative APP is albumin, with positive APP including fibrinogen, haptoglobin, and serum amyloid A (SAA). Serum/plasma levels of positive APP increase in response to a triggering event (eg, infection, trauma) and decrease coinciding with recovery; the magnitude of increase and rate of decline varies between species and the specific APP.[3]

Equine SAA is an apolipoprotein consisting of three isoforms and is complexed primarily to high-density lipoproteins.[4–6] Mainly produced by the liver, extrahepatic secretion of SAA also occurs into colostrum and synovial fluid. SAA is the only major positive APP in the horse. A major APP is defined as one whose concentrations are low or clinically undetectable in normal animals but rapidly increase greater than 10-fold during the APR and rapidly decrease with disease resolution. Relapse or a new/secondary insult result in a return to increased concentrations.[4] This contrasts with moderate APPs, such as fibrinogen and haptoglobin in the horse, which are detectable in serum/plasma of healthy animals, have a slower response to stimuli, and increase 1- to 10-fold during the APR. Fibrinogen has remained the mainstay of blood analysis for inflammation in horses and other large animal species, largely because of the ease and minimal expense of testing. However, fibrinogen concentrations do not increase for 24 hours after the inciting inflammatory event and only peak at 48 to 72 hours.[7] Additionally, the increase in fibrinogen concentrations is small (usually 1- to 2-fold) making it difficult to reliably detect mild inflammation. In contrast, serum/plasma SAA concentrations increase up to 1000 times as soon as 6 hours after stimulation and, once the stimulus is removed, concentrations decrease within 12 hours because of its short half-life (30–120 minutes).[5,8,9]

The widespread use of fibrinogen versus SAA measurement in large animals most likely started as a function of assay availability. Methods of diagnostic evaluation of SAA have greatly improved since its discovery as a major APP, allowing for inexpensive testing in serum or plasma stallside or in laboratories.[10–13] SAA is stable at room temperature and refrigerated, allowing for transport before testing[10] and can be measured in noninvasive samples, such as saliva.[14] Although there are differences in precision and accuracy between assays, most available tests seem accurate enough within clinically relevant ranges.[8,10,11,13] Various publications refer to SAA in mg/L, μg/mL, and ng/mL; the first two of these units are equivalent and the third represents one thousandth the concentration of the first.

SERUM AMYLOID A IN NORMAL HORSES COMPARED WITH HORSES WITH INFECTIOUS OR INFLAMMATORY DISEASE

A commonly accepted reference interval for SAA concentration in horses is 0 to 20 mg/L,[15] with most reports finding normal horses have SAA values at or lower than 12 mg/L.[6,8,16–30] Several studies have examined the utility of SAA for discriminating between horses with or without inflammation. Animals with systemic inflammation had significantly higher SAA concentrations (mean, 1583 mg/L; range, 688–4000 mg/L) than horses with local or no inflammation (mean of 343 mg/L and range of 37–1609, and mean of 5.6 mg/L and range of 1.8–14.5 mg/L, respectively). This discrimination was more distinct than that of fibrinogen, where the mean concentrations of the three groups were 224, 181, and 128 mg/dL, respectively.[31] For differentiating clinically normal horses from those with infectious or inflammatory disease, SAA measurement had a sensitivity of 53%, specificity of 94%, and diagnostic accuracy of 75%, whereas white blood cell count, plasma fibrinogen concentrations, and albumin/globulin ratios had lower diagnostic accuracy (59%–62%).[32] In cases where

serial SAA measurements were obtained, horses with increasing SAA concentrations between 24 and 72 hours of admission were significantly more likely to develop complications or be euthanized, whereas there was no significant difference in outcome in horses with increased SAA concentrations at admission.[25]

SERUM AMYLOID A IN FOALS

Multiple reports have shown foals have similar baseline values of SAA to adult horses and the rate of rise and fall is comparable between foals and adults.[33–36] Foals with sepsis and other bacterial infections have significantly higher SAA concentrations than healthy foals or sick foals without inflammation, although there is enough overlap in the concentrations among these groups that SAA should not be used as a sole means of identifying infection or sepsis.[35–38] In the largest study evaluating SAA in foals, a healthy control group of 226 Thoroughbred neonates had median SAA concentrations of 0.9, 4.5, and 2.5 mg/L on 1, 2, and 3 days old, with the values on Day 2 being significantly higher than on Day 1.[33] In 136 foals with clinical disease, median SAA concentrations of cases with focal infections (eg, omphalitis) were 195 mg/L and those with septicemia higher still at 280 mg/L. Foals with noninflammatory abnormalities, such as failure of passive transfer and noninfectious disease, had low SAA concentrations at 5.1 and 3.1 mg/L, respectively. A separate small study found that foals with positive blood cultures had markedly increased SAA concentrations compared with foals with ambiguous or negative blood cultures, which had moderate or no increases in SAA concentrations, respectively.[35]

Because of the presence of SAA within colostrum, it has been suggested that the increased SAA concentrations in normal foals shortly after birth may be associated with colostral absorption.[39] High fibrinogen levels in neonatal foals can often indicate intrauterine inflammation. Although less is known about SAA levels with intrauterine inflammation, one report assessing the relationship between intrauterine or umbilical cord bacteria with foal health following parturition found no significant difference between postpartum SAA levels and the presence of bacteria in amniotic fluid or venous umbilical blood.[40]

GASTROINTESTINAL DISEASE AND SERUM AMYLOID A

In regards to colic, multiple different APPs have been evaluated for their ability to distinguish surgical from medical causes of colic, and their usefulness in monitoring for complications and predicting prognosis and response to treatment. Most[4,41,42] but not all[43,44] studies have found that SAA concentrations are significantly higher (median, 65[41] to 935 mg/L[42]) in horses with colic-attributable inflammatory causes (eg, enteritis, colitis, peritonitis, or abdominal abscesses) versus surgical and noninflammatory colic (median, 4.8[41] to 228 mg/L[42]). Furthermore, SAA concentrations were increased in horses with equine grass sickness (median, 50 mg/L) compared with those with surgical and noninflammatory colic (median, ~0 mg/L).[45] In contrast to these reports, one prospective study found that 62% of horses with colic requiring surgical intervention had SAA concentrations greater than or equal to 5 mg/L, compared with 19% of medically managed horses, although cases of peritonitis or colitis were excluded.[43] A smaller retrospective study also found that SAA concentrations did not distinguish between surgical and nonsurgical or strangulating and nonstrangulating lesions.[44]

Several studies have shown that peritoneal fluid SAA concentrations are increased in horses with colic compared with control horses, although peritoneal fluid concentrations are not higher than that in serum/plasma.[42,46,47] However, peritoneal fluid SAA concentrations increase more rapidly in horses with strangulating lesions than other

diseases, although it only discriminated between simple obstruction and strangulating or inflammatory colics of greater than 24 hours duration.[47]

It is known that surgery itself, such as exploratory celiotomy for colic, causes an increase in APPs, including SAA.[43,48–50] Compared with more minor surgical procedures, exploratory celiotomy causes a prolonged APR, with serum SAA concentrations increased from 48 to 96 hours after surgery (compared with 12–24 hours with minor procedures).[48,50,51] Horses with postoperative complications had a more pronounced increase in SAA (61.4-fold over baseline) and took longer to achieve peak values (96 hours) compared with horses without complications (29-fold increase; peak at 48 hours).[50] Another prospective study assessing SAA and complications in postoperative colic patients found that the magnitude of SAA increase was greater at 48 hours and 4 to 6 days postsurgery in horses with versus those without complications.[49]

The data are conflicting regarding the accuracy and utility of SAA for determining outcome and prognosis in horses with colic. One study[44] found no significant differences in SAA concentration between survivors and nonsurvivors, whereas another larger study[50] of horses undergoing exploratory celiotomy found that patients with increased SAA concentrations 5 days postoperatively were slightly less likely to survive to discharge (odds ratio, 0.97). Taken together, the current literature with equine colic suggests that SAA alone does not offer clear guidance on differentiating surgical from nonsurgical colics nor should it be relied on for prognosticating survivability when euthanasia is being considered. However, colic cases that are admitted with higher SAA concentrations ($\geq$20, and typically 60–1000 mg/L) should have inflammatory lesions, such as enteritis and colitis, higher on the differential diagnostic list.

There are fewer available studies assessing SAA in gastrointestinal diseases other than colic. A small experimental study found that inoculation with equine *Coronavirus* resulted in SAA concentrations that mirrored naturally occurring disease; although all three challenged horses shed large quantities of virus, only those that showed clinical signs of diarrhea, fever, and anorexia had increased SAA concentrations, which peaked at 200 to 400 g/L.[21]

SERUM AMYLOID A AND EQUINE RESPIRATORY DISEASES

SAA has been investigated mainly for its ability to distinguish infectious from noninfectious causes of respiratory disease and to separate horses with bacterial pneumonia from those with viral infections. Several studies have demonstrated that transport alone, the major risk factor for pneumonia in horses, can cause increased SAA concentrations, ranging from around 30 to 500 μg/mL for 24 to 48 hours after long-distance (1200 km) shipping.[52] This increase was significantly diminished by administration of antimicrobials[52,53] but shorter transport times (4 hours) had no effect.[54]

Horses naturally infected with equine influenza virus A2 (H3N8) had increased SAA concentrations during the first 48 hours of clinical signs. Concentrations then returned to baseline within 11 to 22 days.[55] A useful characteristic of SAA is that it does not increase higher than baseline in horses naturally exposed to but not infected with equine herpes virus type 1 or *Streptococcus equi* subsp *equi*.[29] In bacterial pneumonia, marked increases in SAA concentrations is seen (in the thousands)[32] and one prospective study found that the SAA concentration correlated better with rectal temperature and clinical resolution of disease than fibrinogen concentrations in Thoroughbreds experimentally infected with *S equi* subsp *zooepidemicus*.[16] Concentrations of SAA peaked at Day 3 postinoculation and returned to baseline at Day 15 as compared with fibrinogen, which peaked at Days 4 to 5 and returned to baseline by

Day 22. Concentrations of SAA in horses were significantly lower in horses with equine influenza virus and equine herpes virus type 1 infection (median, 731 mg/L and 1173 mg/L, respectively; range, 0 to ≥3000 mg/L) than those positive for *S equi* subsp *equi* (median, 1953 mg/L; range, 0 to ≥3000 mg/L). Because of the significant overlap between horses with viral and bacterial respiratory disease, SAA concentrations alone cannot be used to distinguish between these two groups.[30]

When examining horses with equine asthma (separated into inflammatory airway disease or recurrent airway obstruction in the highlighted studies), multiple investigations have found increases in SAA concentration in affected horses compared with control subjects.[19,26] In contrast, other studies found no significant differences in SAA concentrations in horses with inflammatory airway disease or recurrent airway obstruction and control horses.[56,57] Therefore, the main utility of SAA measurement in equine asthma seems to be differentiating these cases from horses with infectious respiratory disease.

RHODOCOCCUS EQUI AND SERUM AMYLOID A

Some of the challenges with *Rhodococcus equi* include identifying subclinically affected foals before they develop clinical disease and determining which patients will benefit from antibiotic treatment, especially with issues of antimicrobial resistance. Several studies have investigated SAA as a possible predictor of *R equi* pneumonia in at-risk populations. The first study used a large, well-selected population of affected foals and age-matched control animals from endemic farms.[58] No predictive value for the incidence of pneumonia was found in SAA concentrations in 212 foals between 7 and 14 days old or 196 foals between 21 and 28 days of age nor in the onset of clinical signs of pneumonia. The authors concluded that "monitoring concentrations of SAA is not useful as a screening test for early detection of *R equi*."[58] A subsequent smaller study that assessed weekly screening with SAA to identify preclinical *R equi* infections in an endemic farm diagnosed with *R equi* pneumonia found similar results.[59] In the latter study, SAA concentrations were not associated with the development of sonographic evidence of lung abscessation and only two of six foals with pneumonia had high SAA concentrations.

Another more recent study investigated APPs in foals with bronchopneumonia caused by various pathogens, including *R equi*. In foals on a farm with endemic *R equi* infection, no correlation was seen between SAA concentrations and the radiographic score of foals who developed *R equi* pneumonia. The ability of SAA to predict development of *R equi* in foals using a cutoff point of greater than 53 μg/mL was found to be limited (sensitivity, 64%; specificity, 77%).[37] The limited utility of SAA in identifying preclinical cases of *R equi* is surprising because this organism generally yields robust increases in fibrinogen concentrations and leukocyte count, which are less sensitive than SAA for detection of other inflammatory diseases. No definitive answer for this discrepancy has been elucidated.

SURGERY AND SERUM AMYLOID A

Even minor surgical procedures cause inflammation and this is reflected in multiple reports that have demonstrated increases in SAA concentrations following elective procedures.[29,34,51,55] Recognizing that SAA can increase postsurgery is important, but serial measurement of SAA may be a useful early marker of horses with postoperative infections. In a study of horses after castration,[60] all horses had high SAA concentrations (around 400–600 mg/L) at Day 3 postoperatively, but those that developed infections still had SAA values in this range on the eighth day, whereas horses recovering

without complication had lower concentrations (around 200 mg/L). The infections were not reliably reflected by increases in rectal temperature, leukocyte count, or fibrinogen concentration, suggesting that SAA was a superior marker for infection. A subsequent study found that SAA concentrations increased in horses undergoing castration but those given perioperative penicillin and flunixin versus flunixin alone had lower SAA concentrations on Days 3 (515 mg/L vs 708 mg/L) and 8 (125 vs 545 mg/L) postcastration,[61] suggesting that mild subclinical infections after surgery result in appreciable differences in SAA concentration.

With other minor surgical procedures, peak SAA concentrations of 100 to 400 mg/L approximately 3 days after surgery is expected in cases uncomplicated by infection. Concentrations of SAA were also significantly lower in elective (defined as noninflamed) versus nonelective (preexisting inflammatory foci) cases.[34] Measurement of SAA was also able to delineate differing levels of surgical trauma based on invasiveness.[51] In several of these studies[34,51] the SAA response was found to be a more sensitive indicator of inflammation than various other APP or leukocyte responses, and concentrations dropped quicker than fibrinogen with resolution. This is particularly useful to the practitioner who must decide whether hematologic evidence of inflammation is simply a holdover from effects of surgery or indicative of postoperative infection that requires further diagnostic evaluation or treatment.

SERUM AMYLOID A IN REPRODUCTIVE HEALTH AND DISEASE

There are conflicting data on SAA concentrations in the periparturient period of healthy mares. One study found that SAA levels remained low in the 8-week prepartum period with slight increases in some mares in the last week before foaling.[20] Two other studies found no increases in SAA concentration before parturition.[62,63] Healthy mares do show increases in SAA concentration in the 24 to 36 hours following parturition, which return to baseline values 5 to 7 days postpartum[20,62]; in one study, the mean SAA concentration reached 62 mg/L (range, 0.7–305 mg/L) and 189 mg/L (range, 0–1615 mg/L) at 12 and 36 hours postpartum, respectively.[20]

Most reports have found no changes in serum SAA concentrations following breeding or infectious endometritis.[22,28,64,65] Only one study showed a significant increase in serum APP levels (SAA and fibrinogen) after experimental induction of endometritis.[66] Based on these results, measurement of SAA concentrations does not seem to be useful for endometritis assessment in horses.

Mares who experienced early embryonic death versus healthy control mares were more likely to have SAA concentrations greater than 30 mg/L.[18] Some mares in the early embryonic death group had increased SAA concentrations before ovulation (mean, 687 mg/L) that remained high until 10 days postovulation. The authors hypothesized that the mares with increased SAA concentrations before ovulation had undiagnosed endometritis before breeding.[18]

In mares with experimentally induced placentitis, SAA concentrations peaked between 274 and 4385 mg/L within 2 to 6 days after intracervical inoculation; mares generally aborted within 2 to 6 days of the initial rise in SAA greater than the reference interval.[20,63] Abortion was more likely in mares with high SAA concentrations compared with mares with SAA concentrations within the reference interval,[20] and values in the former group increased steadily until abortion, after which they rapidly decreased. In comparison, fibrinogen concentrations and white blood cell counts were not found to be useful markers of placentitis.[63] A prospective study examining fetal serum samples found that SAA concentrations were significantly increased in cases where a causative microorganism was identified and either fetal multiorgan

disease (10–40 mg/L) or placentitis (2.5 to >40 mg/L) was present, compared with cases of placentitis without an identifiable microorganism or in which no infectious or inflammatory cause was found (<2.5 mg/L).[67]

SERUM AMYLOID A AND DISEASES OF EQUINE JOINTS AND SYNOVIAL STRUCTURES

A comparatively large amount of literature is available regarding SAA concentrations in septic arthritis and tenosynovitis and, overall, SAA seems to be a sensitive marker of these diseases in adult horses. Healthy control horses have serum and synovial concentrations of SAA that are generally less than 1 mg/L. Repeated arthrocentesis (which increases the nucleated cell count and total protein) and intra-articular amikacin injection do not affect SAA concentrations and SAA measurement in these cases may be important because the effects of repeated sampling can confound assessment of treatment efficacy and resolution.[68,69] Additionally, more recent studies have found that repeated arthroscopy and repeated through-and-through joint lavage also do not affect synovial or serum SAA concentrations, whereas the nucleated cell count and total protein are increased following these procedures alone.[70,71] Although most of the SAA found within synovial fluid may be an ultrafiltrate from plasma, a joint-specific isoform of SAA is produced by synoviocytes.[72]

As in other diseases, bacterial infection of joints and other synovial structures seems to be the most potent stimulant of SAA production. Concentrations of SAA in plasma and synovial fluid are increased in horses with septic synovial disease (synovial fluid, mean of 39.2 mg/L and range of 0–368.9 mg/L; plasma, mean of 275.5 mg/L and range of 0–1421.8 mg/L) but not nonseptic (synovial fluid, mean of 0 mg/L and range of 0–29.7 mg/L; plasma, mean of 0.5 mg/L and range of 0–17 mg/L) or control groups.[73] A study examining SAA in experimentally induced inflammatory synovitis and septic arthritis found similar differences between groups in serum and synovial fluid SAA concentrations.[74] For the septic and aseptic groups, the mean peak synovial SAA concentrations were 135 mg/L (range, 60–555 mg/L) and 0 μg/L (range, 0–0), respectively, whereas the mean peak serum SAA concentrations were 663 mg/L (range, 217–1434 mg/L) and 0 mg/L (0–0), respectively. This study did find a delayed increase in synovial (no appreciable increase until 36 hours, at which time it peaked) compared with serum (began to increase at 24 hours, peaked at 36 hours) SAA concentrations after induction of septic synovitis.[74]

Horses with penetrating wounds to a synovial structure that presented within 24 hours after the initial injury had lower plasma SAA concentrations at admission (median, 23 mg/L) and a faster decrease following surgery, compared with horses requiring multiple surgeries, which had a median SAA concentration of 3378 mg/L at admission, with persistent increases 48 hours postoperatively (median, 2525 mg/L).[75]

LAMINITIS, OBESITY, AND SERUM AMYLOID A

Determining how SAA concentrations change in laminitis is complicated by the myriad of inflammatory and noninflammatory causes, and the variable chronicity and severity of the disorder. Additionally, conflicting data exist regarding the role of obesity and inflammation within horses. Concentrations of SAA were not increased in previously laminitic ponies that were in remission, but exercise caused slight increases in some ponies.[76,77] In obese equids, increases in SAA concentrations were correlated with higher body condition score and higher plasma insulin concentrations.[78] However, the SAA concentrations in all of the horses were within the reference interval

(3.8 mg/L was the highest result), so the diagnostic utility of using SAA to assess for inflammation in obese horses is uncertain.[78]

EXERCISE AND SERUM AMYLOID A

Several observational studies have evaluated the effects of long-distance rides in horses.[17,79,80] In endurance horses, SAA concentrations significantly increased (10-fold) from baseline after long-distance, but not after limited-distance, races.[80] Arabian horses that were just beginning endurance training had higher SAA concentrations versus baseline compared with experienced horses undergoing the same effort.[79] However, SAA concentrations increased similarly postrace in experienced and inexperienced horses.[17] Prerace SAA concentrations were significantly lower in Arabian endurance horses that finished the race versus those who could not complete the distance.[17]

A study of racing Standardbred trotters found that acute strenuous exercise did not cause significant increases in SAA concentration[81] and there was only a weak correlation between SAA concentration and cumulative training days in training thoroughbreds followed for several months of training.[82] Overall, SAA concentrations seem to increase to a greater degree with endurance exercise as compared with short-distance (including strenuous) work, with variations between types of exercise likely being subtle enough to make clinical utility of these findings minimal.

SERUM AMYLOID A AND PARASITES

In a study of horses experimentally infected with small and large strongyles, APPs were monitored over 161 to 164 days. Although haptoglobin and iron concentrations and albumin/globulin ratios were associated with strongyle burden, SAA concentrations were not and remained low throughout the study.[83] Additionally, no significant changes in SAA concentration were seen after anthelmintic treatment in two separate groups of experimentally infested and heavily parasitized horses.[84,85] This provides the practitioner with useful information because larval cyathostomiasis is a difficult diagnosis to make antemortem, and low SAA concentrations are uncommon with inflammatory colonopathies.

SERUM AMYLOID A AND VACCINATION

After vaccination with two different influenza and tetanus toxoid products, horses showed variable APRs with SAA concentrations increasing higher than 5 mg/L and peaking (~30–175 mg/L) at 48 hours after vaccination in 6/10 horses. Increased white blood cell counts, fibrinogen concentrations, and decreased serum iron concentrations were also noted. By 96 hours, SAA concentrations declined but had not quite reached baseline values.[86]

OTHER DISEASES AND SERUM AMYLOID A LEVELS

One prospective study evaluated horses with ocular disease (ulcerative keratitis) as compared with two control groups (positive control horses with systemic inflammation but no ocular disease and negative control horses with no evidence of ocular or systemic disease).[87] Compared with the negative control group, positive control horses, but not the ocular disease group, had significantly higher fibrinogen and SAA. The authors concluded that increases in APPs in patients with ocular disease should raise suspicion for systemic inflammation.

A retrospective study assessed the usefulness of SAA in the diagnosis of equine protozoal myeloencephalitis using stored serum or cerebrospinal fluid samples from 25 clinical cases.[88] Affected horses had low or undetectable SAA concentrations in both sample types, indicating that SAA measurement is unlikely to aid in a clinical diagnosis of this disease.

SUMMARY

SAA is a sensitive predictor of early inflammation and, because of its rapid onset and short half-life, tracks the course of disease closely. In most studies, it outperforms the other commonly used markers of inflammation, fibrinogen and white blood cell count, and also seems superior to the other acute phase markers, including haptoglobin, C-reactive protein, and serum/plasma iron. However, it is not useful to diagnose specific diseases and should not replace careful physical examination or diagnostic testing to identify of the cause of the inflammatory response.

Although SAA has many advantages, it is still not a diagnostic panacea. It seems to have limited validity in screening foals for *R equi* pneumonia, although it often increases to extremely high concentrations in pleuropneumonia in adult horses and is valuable in assessing response to treatment in such cases. SAA also does not reliably distinguish surgical from nonsurgical colic cases and, although it may possibly be oversensitive, serial testing of SAA is likely superior to that of fibrinogen in identifying postoperative infections. Any deviations from a steady fall after the first 2 to 3 days after surgery might prompt a search for infectious complications. Overall, practitioners should feel comfortable using SAA in lieu of fibrinogen (and certainly in preference to complete blood count (CBC), in the authors' opinion) for most cases where infectious or inflammatory disease is suspected, although measuring both initially may be useful for the practitioner with limited experience in interpreting the wide range of results with this marker.

DISCLOSURE

The authors have nothing to disclose.

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