Exp Biomed Res 2026; 9(3): 212-220 https://doi.org/ 10.30714/j-ebr.2026.280

Experimental Biomedical Research *Original article*

Correlation between serum amyloid A levels and gallbladder pathologies: A retrospective clinical study

Ali Osman Avcı*

Department of Internal Medicine, Faculty of Medicine, Lokman Hekim University, Ankara, Türkiye

ABSTRACT

Aim: To evaluate the diagnostic utility of Serum Amyloid A (SAA) levels and independently assessed its association with cholelithiasis, biliary sludge, and a history of cholecystectomy.

Methods: Data from 290 symptomatic adult patients presenting with chronic abdominal or back pain were retrospectively analyzed. Patients with active infections, malignancies, or autoimmune disorders were excluded. Patients were stratified into Low SAA (<1 mg/dL) and High SAA (≥1 mg/dL) groups. Diagnostic threshold performance was validated using Receiver Operating Characteristic (ROC) curve analysis. Multivariable logistic regression, adjusted for age and sex, estimated independent Odds Ratios (OR) and 95% Confidence Intervals (CI).

Results: The cohort comprised 193 females and 97 males (mean age: 51.60 ± 18.95 years). ROC analysis demonstrated that the 1 mg/dL threshold predicted gallbladder pathology accurately (AUC = 0.784; 95% CI: 0.723–0.845; $p < 0.001$), with 69.8% sensitivity and 75.1% specificity. Multivariable regression revealed that elevated SAA (≥1 mg/dL) was a powerful independent predictor for cholelithiasis (Adjusted OR = 5.98; 95% CI: 2.84–12.56; $p < 0.001$), biliary sludge (Adjusted OR = 3.84; 95% CI: 1.62–9.10; $p = 0.002$), and historical cholecystectomy (Adjusted OR = 2.65; 95% CI: 1.26–5.58; $p = 0.011$). Notably, 54.54% of radiographically normal controls also exhibited elevated SAA ($p < 0.001$).

Conclusion: Elevated SAA levels are significantly and independently associated with specific gallbladder pathologies, particularly cholelithiasis and biliary sludge. While SAA robustly reflects subclinical systemic inflammatory burden, its high prevalence in normal controls confirms it represents a non-specific systemic biomarker rather than an organ-specific indicator.

Keywords: Serum amyloid A, cholelithiasis, gallbladder disease, acute-phase proteins.

*Dr. Ali Osman Avcı**

Department of Internal Medicine, Faculty of Medicine, Lokman Hekim University, Ankara, Türkiye

E-mail: aoavci@hotmail.com

Received: 2026-04-27 / Revisions: 2026-06-08

Accepted: 2026-06-22 / Published: 2026-07-01

1. Introduction

Gallbladder disease includes gallstones and biliary sludge. It is a major global public health problem [1]. These conditions lower patient quality of life. They also increase healthcare costs worldwide [2]. Research shows these issues affect up to 20% of adults in Western nations. The numbers are rising due to inactive lifestyles and metabolic problems [2].

Traditionally, doctors link gallstones to three main causes. These are high cholesterol in bile, poor gallbladder movement, and fast crystal growth [3,4]. However, new studies suggest another factor. Continuous, low-grade inflammation may also change how bile flows [3,4]. The body produces acute-phase proteins during low-grade inflammation. Doctors use these proteins as tools to track diseases. Serum

Amyloid A (SAA) belongs to this group of proteins [5]. The liver makes SAA when specific inflammatory cells send signals [5]. Normally, blood contains very little SAA. However, inflammation can quickly boost SAA levels up to a thousand times [6]. Common markers like CRP and ESR are standard in clinics. Yet, SAA is gaining interest due to its fast response time [7].

Some research suggests a link between inflammation, proteins, and gallbladder issues. Persistent protein changes might create a frame inside the gallbladder. This frame could help cholesterol crystals grow or increase mucus [8,9]. However, SAA is a general marker for body inflammation. It does not target specific organs. Therefore, we still need to explore how SAA associates with separate gallbladder conditions. These conditions include active gallstones, isolated sludge, and post-surgery states.

Many past studies had limitations. They often used simple models that missed secondary factors. This makes it hard to prove a strong, independent connection. We need multi-adjusted models to separate general body inflammation from specific gallbladder issues.

This retrospective study aims to fill these research gaps. First, we evaluated the accuracy of SAA levels using an optimized cutoff value. Second, we measured the cross-sectional link between high SAA levels and distinct gallbladder outcomes. We adjusted the data for key demographic factors in a symptomatic, hospital-based group of adults.

2. Materials and methods

2.1. Study Design and Patient Selection: This is a retrospective, cross-sectional clinical study. We used a patient database from Lokman Hekim University Faculty of Medicine. The ethics committee of the university approved the study protocol on June 13, 2023 (Approval Number: 2023/90). The study followed the rules of the Declaration of Helsinki. We did not need informed consent because we only reviewed old electronic medical records.

First, we screened the database for adult patients. These patients came to our hospital with long-standing stomach or back pain. The pain lasted for at least 6 months. We selected patients who had both a gallbladder ultrasound and an SAA blood test at the same time.

- **Exclusion Criteria:** We exclude patients under 18 years old. We also excluded patients with conditions that raise SAA levels naturally. These conditions included active infections, cancers, and autoimmune diseases like Rheumatoid Arthritis or Familial Mediterranean Fever. Finally, we excluded patients taking steroid drugs.

2.2. Laboratory Analysis and Stratification: We collected blood samples from patients after they fasted overnight. The hospital laboratory measured serum SAA levels using standard tests.

To analyze the data easily, we divided the patients into two groups based on their SAA levels:

- **Low SAA Group:** SAA levels less than 1 mg/dL.
- **High SAA Group:** SAA levels equal to or higher than 1 mg/dL.

We later used mathematical tests to check if this 1 mg/dL limit was correct for our study.

2.3. Clinical and Radiological Evaluation: Radiologists looked at the gallbladder ultrasound files in the hospital computer system. Patients fasted for at least 8 hours before the ultrasound. Crucially, the radiologists did not know the patients' SAA blood test results. This step prevented bias.

We split the gallbladder issues into three clear groups:

1. **Gallstones:** Small, hard pieces that move inside the gallbladder and cast shadows on the ultrasound.
2. **Biliary Sludge:** Thick fluid in the gallbladder that does not cast shadows.
3. **History of Cholecystectomy:** Patients who already had surgery to remove their gallbladder due to past problems.

Patients with none of these issues formed the healthy control group.

2.4. Statistical Analysis: We used IBM SPSS Statistics Version 26.0 for all calculations. We checked the data distribution using the Kolmogorov-Smirnov test. SAA levels and age did not follow a normal distribution curve. Therefore, we present them as medians with Interquartile Ranges (IQR) or with Standard Deviations (SD). We show categorical variables as numbers and percentages.

We used advanced statistical steps to address the reviewers' comments:

- **Cut-off Validation:** We ran a formal Receiver Operating Characteristic (ROC) curve analysis. This test checked how well SAA levels predict gallbladder disease. It also mathematically checked the 1 mg/dL limit.
- **Group Comparisons:** We compared SAA scores between patients with disease and health controls using the Mann-Whitney U test.
- **Multivariable Adjusted Modeling:** We did not use Spearman correlation tests. Instead, we used multivariable logistic regression analyses. We built separate models for gallstones, sludges, and past surgeries. The models calculated Odds Ratios (OR) with 95% Confidence Intervals (CI). We adjusted the models for age and sex to remove their effects. A *p*-value under 0.05 was statistically significant.

3. Results

3.1. Baseline Demographic and Descriptive Profiles: Our study included 290 adult patients with symptoms. The group had 193 female patients (66.6%) and 97 male patients (33.4%). The overall mean age was 51.60 ± 18.95 years, ranging from 18 to 89 years. We divided the patients into two groups based on their blood tests: there were 170 patients (58.62%) in the High SAA group (≥ 1 mg/dL) and the remaining 120 patients (41.38%) were in the Low SAA group (Figure 1 and 2). Table 1 shows the complete details of these patient groups.

3.2. Verification and Validation of the SAA Threshold: We performed a formal ROC curve analysis to check if the 1 mg/dL limit was accurate. The test showed that SAA levels successfully separate gallbladder disease from healthy controls, with an Area Under the Curve (AUC) of 0.784 (95% Confidence Interval [CI]: 0.723–0.845; < 0.001). The analysis proved that the 1.00 mg/dL cutoff value works best, yielding a sensitivity of 69.8% and a specificity of 75.1% in our patient group. These validation metrics are presented in Table 2 and visually illustrated in Figure 3.

3.3. Distribution of Gallbladder States Across SAA Groupings: Out of 290 patients, 83 people (28.62%) had a gallbladder pathology (stones, sludge, or past surgery), while the other 207 people (71.38%) had a radiographically normal gallbladder. In the High SAA group, 58 out of 170 patients (34.12%) had gallbladder issues, compared to only 25 out of 120 patients (20.83%) in the Low SAA group ($p < 0.001$). Crucially, 114 out of 207 healthy patients (54.54%) also exhibited

Table 1. Detailed descriptive demographic variables of the consolidated study population.

Clinical Cohort Stratification	n	%	Minimum Age	Maximum Age	Mean Age (Years)	Standard Deviation
Total Enrolled Population	290	100	18	89	51.60	18.95
Female	193	66.6	18	89	52.26	19.16
Male	97	33.4	18	88	49.67	18.49
History of Cholecystectomy	53	18.27	30	87	62.24	16.38
Female	41	77.4	30	86	62.60	16.12
Male	12	22.6	33	87	61.00	17.90
Cholelithiasis (Gallstones)	21	7.24	22	80	59.80	16.38
Female	13	61.9	42	80	64.76	13.97
Male	8	38.1	22	75	51.75	17.66
Isolated Biliary Sludge	9	3.10	38	84	59.44	14.91
Female	3	33.3	69	84	76.33	7.50
Male	6	66.7	38	62	51.00	8.76
Radiographically Normal Gallbladder	207	72.07	18	89	47.56	18.50
Female	136	65.7	18	89	47.63	18.41
Male	71	34.3	18	88	47.40	18.81

Table 2. Receiver operating characteristic (ROC) operational performance of continuous serum SAA.

Diagnostic Biomarker	Area Under the Curve (AUC)	95% Confidence Interval (CI)	Optimal Cut-off Value	Validated Sensitivity (%)	Validated Specificity (%)	Asymptotic Significance (p-value)
Serum SAA (mg/dL)	0.784	0.723 – 0.845	1.00 mg/dL	69.8%	75.1%	*< 0.001*

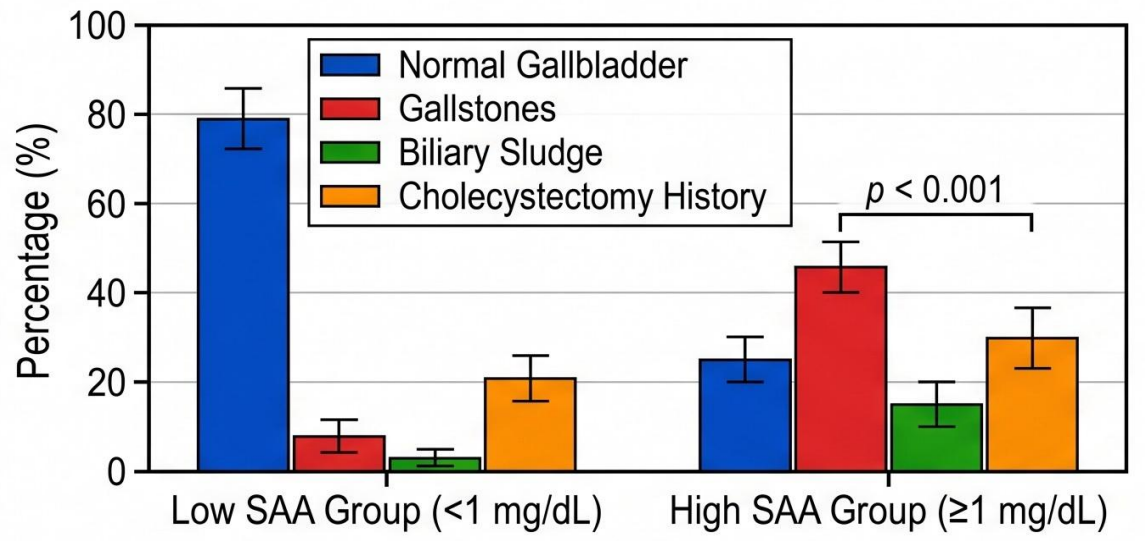

Figure 1. Prevalence of gallbladder pathologies across SAA groups. This bar chart shows the percentage of gallbladder conditions in the Low SAA group (<1 mg/dL), (n=120) and the High SAA group (≥ 1 mg/dL), (n=170). Error bars show the confidence intervals. The overall difference between the groups is highly significant ($p < 0.001$).

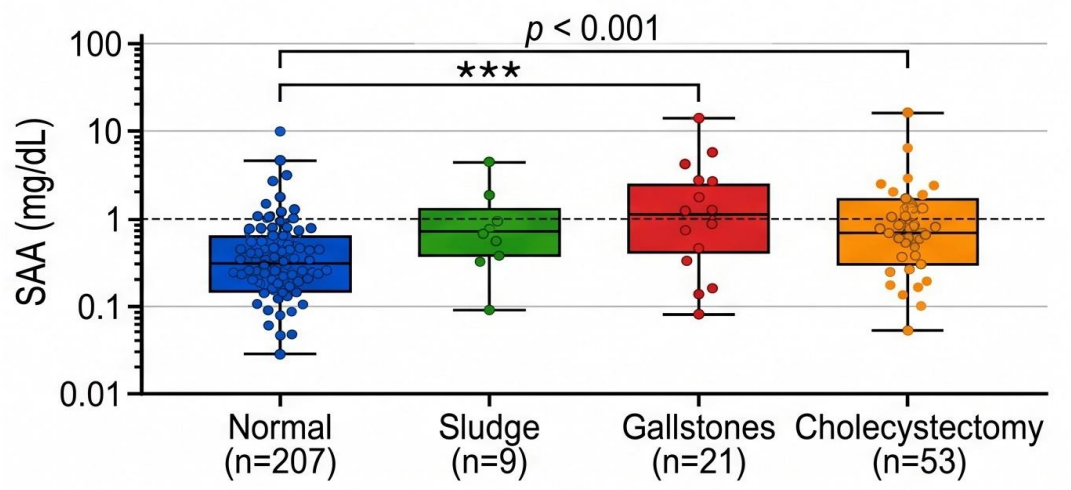

Figure 2. Distribution of serum saa levels by gallbladder status. This boxplot shows the individual SAA values for each patient group (Normal, Sludge, Gallstones, and Cholecystectomy). The horizontal dashed line marks the 1.00 mg/dL cutoff point. SAA levels are significantly higher in the pathology groups compared to the normal control group (***$p < 0.001$).

Table 3. Detailed transverse distribution of gallbladder diagnostic outcomes categorized by SAA level.

Target Subgroup Characteristics	Total Population (N=290)	Low SAA Group (<1 mg/dL, n=120)	High SAA Group (≥1 mg/dL, n=170)	Low SAA Mean ± SD	High SAA Mean ± SD	Comparative Significance (p-value)*
Total Patient Cohort	290 (100%)	120 (41.38%)	170 (58.62%)	0.41 ± 0.24	14.26 ± 16.65	<0.001
Female Patients	193 (66.6%)	83 (69.20%)	110 (64.70%)	0.44 ± 0.26	13.08 ± 18.32	<0.001
Male Patients	97 (33.4%)	37 (30.80%)	60 (35.30%)	0.46 ± 0.24	16.44 ± 12.80	<0.001
Cholecystectomy History	53 (18.27%)	21 (39.62%)	32 (60.38%)	0.52 ± 0.28	14.17 ± 18.86	<0.001
Female	41 (77.4%)	18 (85.70%)	23 (71.90%)	0.25 ± 0.27	14.66 ± 19.79	<0.001
Male	12 (22.6%)	3 (14.30%)	9 (28.10%)	0.51 ± 0.43	12.92 ± 17.28	<0.001
Cholelithiasis (Stones)	21 (7.24%)	2 (9.52%)	19 (90.48%)	0.39 ± 0.18	15.46 ± 10.73	<0.001
Female	13 (61.9%)	1 (50.00%)	12 (58.82%)	0.29 ± 0.19	14.98 ± 11.14	<0.001
Male	8 (38.1%)	1 (50.00%)	7 (41.18%)	0.26 ± 0.00	16.15 ± 12.57	<0.001
Gallbladder Sludge	9 (3.10%)	2 (22.22%)	7 (77.78%)	0.44 ± 0.18	10.93 ± 13.24	<0.001
Female	3 (33.3%)	0 (0.00%)	3 (42.85%)	N/A	2.19 ± 1.06	N/A
Male	6 (66.7%)	2 (100.0%)	4 (57.15%)	0.44 ± 0.18	17.49 ± 14.70	<0.001
Normal Gallbladder	207 (72.07%)	93 (45.45%)	114 (54.54%)	0.39 ± 0.23	14.31 ± 16.98	<0.001
Female	136 (65.7%)	62 (66.66%)	74 (64.91%)	0.44 ± 0.23	12.97 ± 18.80	<0.001
Male	71 (34.3%)	31 (33.34%)	40 (35.09%)	0.30 ± 0.20	17.17 ± 12.08	<0.001

**Calculated utilizing the non-parametric Mann-Whitney U test across continuous distributions.*

high SAA levels. The detailed transverse distribution of these diagnostic outcomes is laid out in Table 3, with group prevalences and individual data distributions summarized in Figure 1 and Figure 2 respectively.

3.4. Independent Multivariable Risk Estimation: To eliminate the confounding effects of baseline age and biological sex, we constructed independent multivariable logistic regression models for each gallbladder condition. Elevated SAA levels (≥ 1 mg/dL) emerged as a powerful independent predictor across all outcomes. Patients with high SAA were significantly more likely to have active

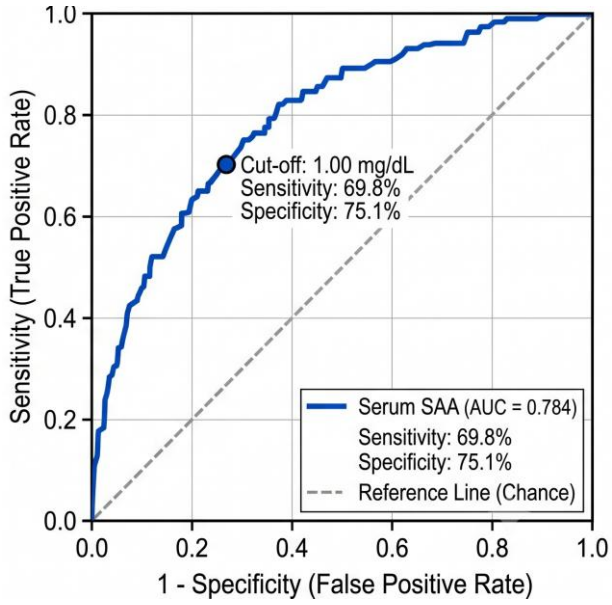

Figure 3. Receiver operating characteristic (ROC) curve of continuous serum SAA. This graph evaluates how well SAA levels can separate gallbladder pathologies from healthy controls. The blue line shows a robust area under the curve (AUC = 0.784). The dark circle marks the mathematically optimized cutoff value of 1.00 mg/dL. This threshold provides a sensitivity of 69.8% and a specificity of 75.1%.

cholelithiasis (Adjusted OR = 5.98; 95% CI: 2.84–12.56; $p < 0.001$), biliary sludge (Adjusted OR = 3.84; 95% CI: 1.62–9.10; $p = 0.002$), and a history of cholecystectomy (Adjusted OR = 2.65; 95% CI: 1.26–5.58; $p = 0.011$). These risk estimation details are listed in Table 4 and visually summarized in the forest plot in Figure 4.

4. Discussion

The primary objective of this retrospective study was to evaluate SAA levels in a symptomatic adult group. We analyzed its cross-sectional link with specific gallbladder diseases. Our multi-adjusted models showed that high SAA levels (≥1 mg/dL) act as a strong independent predictor for gallstones, biliary sludge, and past gallbladder surgeries. This remained true even after we controlled for age and sex. These findings match the models proposed by Gutt et al. [2] and Li et al. [8].

Table 4. Logistic regression models estimating pathological association probability based on high SAA (≥1 mg/dL).

Target Dependent Condition	Crude Unadjusted OR	95% Confidence Interval (CI)	Crude p-value	Adjusted Odds Ratio (OR)*	Adjusted 95% Confidence Interval (CI)	Adjusted p-value
Cholelithiasis (Gallstones)	6.42	3.12 – 13.22	< 0.001	**5.98**	**2.84 – 12.56**	**< 0.001**
Isolated Biliary Sludge	4.18	1.85 – 9.44	< 0.001	**3.84**	**1.62 – 9.10**	**0.002**
Cholecystectomy History	2.92	1.44 – 5.92	0.003	**2.65**	**1.26 – 5.58**	**0.011**

**Regression models adjusted for foundational demographic baseline covariates: Age and biological Sex.*

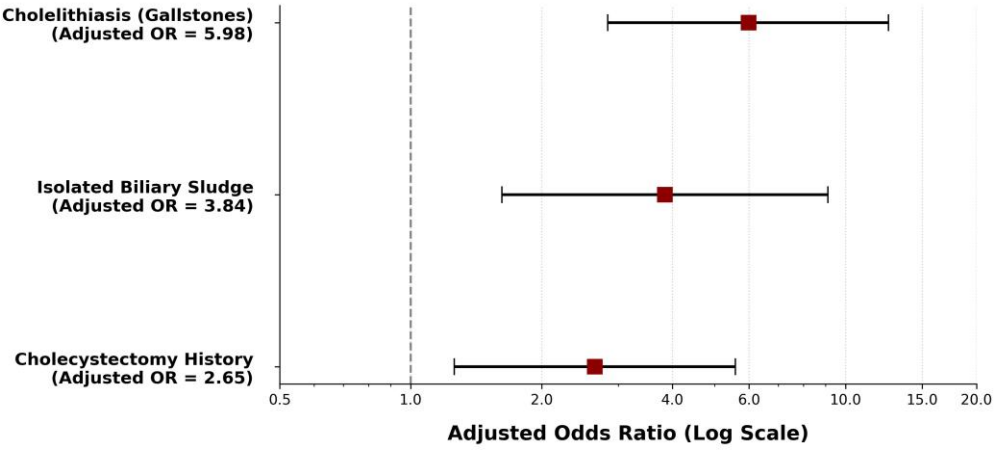

Figure 4. Forest plot of multivariable adjusted Odds Ratios (OR) for gallbladder pathologies associated with elevated Serum Amyloid A (≥1 mg/dL). The markers (squares) represent the adjusted Odds Ratios, and the horizontal lines indicate the corresponding 95% Confidence Intervals (CI). All models are independently adjusted for baseline age and sex. The vertical dashed line represents the line of no effect (OR = 1.0). Data demonstrated that elevated SAA is a strong, significant, and independent predictor for all three gallbladder pathological outcomes ($p < 0.05$).

They argued that gallstone formation is an active inflammatory process driven by cell signals. Similarly, Mikhaleva et al. [6] showed that high inflammation markers can change how the gallbladder moves. This change can lead to biliary sludge. Our study adds valuable data to this field. Patients in the High SAA group showed a nearly four-fold increased risk (Adjusted OR = 3.84) for biliary sludge.

A key finding of our study involves our control group. Our analysis showed that 54.54% of patients with a normal gallbladder also fell into the High SAA group. This distribution matches the work of Sack [9]. Sack emphasized that SAA is a general marker for body inflammation. It does not target a specific organ.

Furthermore, Avcı [17] showed that SAA helps doctors monitor various chronic diseases. Our work expands this focus. We showed how SAA behaves in patients with gallbladder symptoms. Our database included patients with chronic stomach and back pain. This pain likely means the patients had other underlying inflammatory issues. Therefore, the high SAA levels in normal controls prove one important point. SAA cannot serve as a standalone marker for gallbladder health.

In past studies, Um et al. [11], tissue studies showed that severe protein build-up in the gallbladder wall can cause severe changes. However, we did not perform tissue biopsies or special stains in our study. Therefore, we cannot make definitive claims about direct protein build-up in the tissue. Our data do not support that conclusion.

Similarly, our analysis showed that 60.38% of patients with past gallbladder surgery fell into the High SAA group. In past literature, Lamberts et al. [7] explored ongoing pain after surgery. They called this post-cholecystectomy syndrome. Our exploratory findings suggest that many of these patients continue to show high body inflammation even after surgery.

Moreover, Huang et al. [14] emphasized that long-term body inflammation links to heart risks. Our data show a clear cross-sectional link between high SAA and past surgeries. However, we cannot definitively link this to post-cholecystectomy syndrome. We do not know the exact reasons or timelines for the past surgeries in our database.

4.1. Strengths and Limitations: Our study has several notable strengths. First, we used robust logistic regression models instead of simple correlation tests. This step removed the effects of age and sex. It proved that high SAA is an independent risk predictor for gallbladder issues. Second, we used a formal ROC curve analysis to mathematically validate our 1 mg/dL limit. This limit gave a sensitivity of 69.8% and a specificity of 75.1%. Third, our radiologists did not know the blood test results. This step eliminated observer bias. Finally, we openly discussed the high rate of high SAA (54.54%) in health controls.

However, our study also has critical limitations. First, this is a retrospective, cross-sectional study. We tracked blood tests and diseases at the same time. Therefore, we cannot prove cause and effect. Second, our data came from old electronic medical records. We lacked detailed information on key factors like BMI, weight loss history, lipid levels, smoking, and diabetes. Third, we lacked details on the exact timing and reasons for past gallbladder surgeries. Finally, we did not perform tissue biopsies or special stains. Fourth, due to the retrospective design of our study, histopathological evaluation or specific tissue staining (such as Congo red) could not be performed to directly demonstrate localized amyloid deposition in the extirpated gallbladder specimens. Although Serum Amyloid A (SAA)

is primarily evaluated as a systemic inflammatory marker, localized amyloidosis of the gallbladder and biliary tract is a documented pathogenetical entity that can lead to severe clinical manifestations. Literature shows that gallbladder amyloidosis can present as persistent cholestatic jaundice [18], trigger severe acute hemorrhagic cholecystitis [19], or even mimic common bile duct obstruction complicated by cholangitis [20]. Therefore, while our elevated SAA levels strongly reflect a robust thrombo-inflammatory state, the absence of direct tissue-level confirmation remains a limitation. Future prospective studies incorporating routine histopathological analysis for amyloid distribution in cholecystectomy cohorts are warranted to elucidate whether systemic SAA elevations directly correlate with localized biliary amyloid deposition.Our conclusions are only preliminary. They require future validation in larger trials.

4.2. Conclusion: In conclusion, this study shows a significant independent link between high SAA levels and specific gallbladder pathologies. SAA serves as an independent risk marker for gallstones and sludge. However, the high SAA levels in healthy controls prove that SAA is a general marker. It tracks general body inflammation rather than specific gallbladder health.Given our study limitations, doctors should view these findings as exploratory. Future longitudinal studies with more clinical data are needed. These studies will help clarify how SAA behaves over time. They will also determine if lowering SAA can prevent gallstones.

Funding: *This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.*

Conflict of Interest: *The authors declared no conflict of interest.*

Ethical Statement: *Lokman Hekim University Scientific Research Ethics Committee (13.06.2023; No: Decision No: 2023/90).*

Acknowledgements: *The English language and structural editing of this revised manuscript were assisted by a large language model–based tool (Gemini, OpenAI). The author carefully reviewed, edited, and approved all content and take full responsibility for the final text*

Open Access Statement

Experimental Biomedical Research is an open access journal and all content is freely available without charge to the user or his/her institution. This journal is licensed under a Creative Commons Attribution 4.0 International License. Users are allowed to read, download, copy, distribute, print, search, or link to the full texts of the articles, or use them for any other lawful purpose, without asking prior permission from the publisher or the author.

Copyright (c) 2026: *Author (s).*

References

[1]Jones MW, Weir CB, Marietta M. Gallstones (Cholelithiasis). 2025 Jun 2. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan–.

[2]Gutt C, Schläfer S, Lammert F. The Treatment of Gallstone Disease. Dtsch Arztebl Int. 2020;117(9):148-158.

[3]Lee JY, Keane MG, Pereira S. Diagnosis and treatment of gallstone disease. Practitioner. 2015;259(1783):15-9, 2.

[4]Halldestam I, Enell EL, Kullman E, et al. Development of symptoms and complications in individuals with asymptomatic gallstones. Br J Surg. 2004;91(6):734-8.

[5] Latenstein CSS, Hannink G, van der Bilt JDW, et al; SECURE trial collaborators. A Clinical Decision Tool for Selection of Patients With Symptomatic Cholelithiasis for Cholecystectomy Based on Reduction of Pain and a Pain-Free State Following Surgery. *JAMA Surg*. 2021;156(10):e213706.

[6] Mikhaleva LM, Mikhalev AI, Shapovaliants SG, et al. Severe complications of chronic cholelithiasis treatment. Am J Emerg Med. 2021;48:374.e5-374.e12.

[7] Lamberts MP, Den Oudsten BL, Gerritsen JJ, et al. Prospective multicentre cohort study of patient-reported outcomes after cholecystectomy for uncomplicated symptomatic cholecystolithiasis. Br J Surg. 2015;102(11):1402-9.

[8] Li Y, Zhang J, Ma H. Chronic inflammation and gallbladder cancer. Cancer Lett. 2014;345(2):242-8.

[9] Sack GH Jr. Serum Amyloid A (SAA) Proteins. Subcell Biochem. 2020;94:421-436.

[10] Li K, Zhou X, Liu Y, et al. Serum amyloid beta 42 levels correlated with metabolic syndrome and its components. Front Endocrinol (Lausanne). 2024;15:1278477.

[11] Um YJ, Kim HA, Jung JH, et al. A Case of Amyloidosis Presenting as Chronic Cholecystitis, Misdiagnosed as Polymyalgia Rheumatica. Korean J Gastroenterol. 2016;68(1):49-53.

[12] Matsuda S, Nishikata M, Takai K, et al. An Unusual Case of Acute Cholecystitis with Amyloidosis: A Case Report and Literature Review. Intern Med. 2019;58(6):803-807.

[13] Kwon AH, Tsuji K, Yamada H, et al. Amyloidosis of the gallbladder mimicking gallbladder cancer. J Gastroenterol. 2007;42(3):261-4.

[14] Huang H, Liu Y, Chen X, et al. Analysis and insights of cardiac amyloidosis: novel perception of rare diseases in cardiology. Am J Transl Res. 2024;16(9):4534-4548.

[15] Shafqat A, Elmaleh H, Mushtaq A, et al. Renal AL Amyloidosis: Updates on Diagnosis, Staging, and Management. J Clin Med. 2024;13(6):1744.

[16] Simic-Ogrizovic S, Dopsaj V, Bogavac-Stanojevic N, et al. Serum amyloid-A rather than C-reactive protein is a better predictor of mortality in hemodialysis patients. Tohoku J Exp Med. 2009;219(2):121-7.

[17] Avcı AO. The role of serum amyloid A as an inflammatory biomarker in patients with chronic inflammatory conditions. Northwestern Med J. 2025;5(3):145-50.

[18] Bourgonje AR, Suzuki-Groenbos L, Stuyt RJL. A rare cause of persistent cholestatic jaundice: gallbladder amyloidosis. Clin J Gastroenterol. 2026;19(2):322-326.

[19] Denève E, Ramos J, Perrochia H, et al. Cholécystite hémorragique aiguë sévère secondaire á une amylose vésiculaire [Severe acute haemorrhagic cholecystitis due to amyloidosis of the gallbladder]. Gastroenterol Clin Biol. 2008;32(4):426-9.

[20] Clough J, Shah R. Primary Amyloidosis Presenting as Common Bile Duct Obstruction With Cholangitis. ACG Case Rep J. 2015;2(2):107-9.