SYSTEMATIC REVIEW

LACTATE DEHYDROGENASE AS A POTENTIAL PROGNOSTIC BIOMARKER IN SUBARACHNOID HAEMORRHAGE: A SYSTEMATIC REVIEW

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ABSTRACT

Background: Subarachnoid haemorrhage (SAH) lacks specific prognostic blood markers, but lactate dehydrogenase (LDH), linked to cellular damage, shows potential for predicting adverse outcomes in SAH patients. Elevated LDH levels may reflect anaerobic metabolism and tissue injury following SAH, providing insight into disease severity and complications. This review explores the relationship between lactate dehydrogenase levels and outcomes in SAH patients.

Method: A systematic review was conducted using PubMed, Europe PMC, ScienceDirect, and Google Scholar, searching for terms like "Lactate Dehydrogenase," "LDH," "Subarachnoid Haemorrhage," and "Outcome" up to March 3, 2025. Studies comparing LDH levels with Modified Rankin Score (mRS) and secondary outcomes such as Hunt-Hess grade, Fisher grade, complications, and mortality were included.

Result: Seven studies involving 5,985 participants met inclusion criteria. Higher LDH levels correlated with worse mRS scores, increased delayed cerebral ischemia (DCI), postoperative pneumonia, severe Hunt-Hess and Fisher grades, and higher mortality. Using the ROBINS-I tool, four studies showed low risk of bias, and three had moderate risk.

Discussion: Elevated LDH levels predict adverse outcomes in SAH, highlighting its prognostic value. As a marker of cellular damage and anaerobic metabolism, LDH reflects tissue injury and hypoxia post-SAH, explaining its link to complications like delayed cerebral ischemia (DCI) and pneumonia. This physiological basis supports its role in risk stratification, aiding early identification of high-risk patients for targeted interventions and improved outcomes.

Conclusion: Early measurement of LDH levels after SAH onset may help predict patient outcomes and complications, aiding clinical decision-making and improving patient management strategies.

Keywords: Lactate Dehydrogenase, Subarachnoid Haemorrhage, Prognosis, Outcome

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Introduction

Subarachnoid haemorrhage (SAH) is a life-threatening condition characterized by the buildup of blood between the arachnoid and pia mater.[1] Non-traumatic SAH, primarily caused by a ruptured intracranial aneurysm, remains the most common type, accounting for 85% of cases. The global incidence of SAH is 9 per 100,000 people per year, with 10% of patients dying before reaching the hospital.[2] The mortality rate within the first 24 hours can be as high as 25%, and 50% of patients die within the first 30 days.[2] Additionally, the disability rate for those who survive is 66%.[3] At present, the prediction of outcomes for patients with subarachnoid haemorrhage is mainly based on the patient's clinical grade at admission, as assessed by the World Federation of Neurological Surgeons (WFNS) score, with higher grades indicating a worse prognosis.[4] Other factors, such as age and pupillary light reflex, are also considered predictors of functional outcomes.[5] However, all of these scores are entirely dependent on clinical assessments and often disregard other factors that could affect patient outcomes.[4-5]. Thus, specific biomarkers are required promptly to predict future outcomes, enabling the optimization of treatment.

Lactate dehydrogenase is an enzyme present in nearly all body tissues. It plays a crucial role in the anaerobic metabolic pathway by catalyzing the conversion of pyruvate to lactate under anaerobic conditions. While lactate dehydrogenase levels have been used to predict outcomes in patients with heart disorders, cancer, trauma, central nervous system infections, and other conditions, no studies have yet explored the relationship between lactate dehydrogenase levels and the outcomes of patients with subarachnoid haemorrhage.[6-8]

Materials and Methods

The systematic review and meta-analysis were performed and documented in accordance to the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta Analyses) guidelines. Additionally, the review protocol was listed in the PROSPERO International Prospective Register of Systematic Reviews, assigned registration number CRD42024576454

The data were obtained by searching in the following databases: PubMed, EuropePMC, ScienceDirect, and Google Scholar. The search strategy involved using the keywords " Lactate Dehydrogenase", "LDH", "Subarachnoid Haemorrhage", "Prognosis", "Outcome", combine using Boolen operators such as "AND" and"OR". The keyword search terms are listed in Table 1. The search and processing was conducted on March 3, 2025.

The systematic review and meta-analysis included studies published between 2014 and 2024 that investigated the association between lactate dehydrogenase (LDH) levels and outcomes or complications in patients with subarachnoid haemorrhage. Eligible studies were required to report LDH ranges, with higher LDH levels classified as the intervention group and lower levels as

the comparison group. Primary outcomes focused on functional measures, such as the modified Rankin Scale (mRS) score, while secondary outcomes included Hunt-Hess grade, Fisher Grade, Complication, and Mortality rates.

We excluded studies that were non-English, conducted on animals or cadavers, available only as abstracts or conference papers, or not accessible in full text. This was done to ensure high-quality studies and achieve the most reliable results regarding the relationship between LDH levels and predicted outcomes in patients with subarachnoid haemorrhage.

Database searches and data extraction were conducted by seven authors (YYEA, AEP, JW, ME, MA, RF, MGS, HWN). Any disagreements were resolved through consultation with a neurology expert (YS) until a consensus was reached. The data were systematically processed using Google Sheets and Microsoft Excel 2019. Each study was carefully divided into several components, including the Study ID, study design, country of origin, sample size, average age, inclusion and exclusion criteria, type of subarachnoid haemorrhage (SAH), timing of LDH sampling, measured outcome, LDH values, and study conclusions (major findings).

Results

Literature Search

The flow of included studies through selection process depicted in PRISMA diagram shown in Figure 1. Initially, 531 records were identified through database searching consisting of PubMed (10), Europe PMC (200), ScienceDirect (300), and Google Scholar (21). After removing 58 duplicates, 473 records undergone title and abstract screening, resulting in exclusion of 455 articles. The full text of 18 articles were assessed and 11 articles were excluded due to duplication (10 articles) and inaccessible article (1 study). Finally, a total of 7 studies are included in this review. [5,9-14]

Study Characteristics

Characteristics of included studies can be shown in Table 2. This systematic review included seven studies conducted across China, Japan, Belgium, and other regions, with sample sizes ranging from 19 to 3,524 patients. Most studies utilized retrospective cohort designs, while one employed a prospective cohort methodology. All studies measured serum LDH levels at admission. Key inclusion criteria varied but generally required SAH diagnosis confirmed by imaging and appropriate aneurysm treatment. Exclusion criteria included comorbid neurological or systemic conditions, incomplete data, or delayed admission.

Serum LDH and Prognostic Outcomes in Aneurismal-SAH

Table 3 provides an integrated description of the findings from studies assessing the prognostic role of serum lactate dehydrogenase (LDH) levels in aneurysmal subarachnoid haemorrhage (aSAH) patients. The analysis encompasses various outcomes, including complications, mortality, and functional recovery.

LDH Levels and Postoperative Pneumonia (POP)

One study conducted by Ding et al (2019) reported that patients with POP had significantly higher LDH levels (261.26 ± 126.51 U/L) compared to those without POP (189.00
± 69.20 U/L). A threshold LDH level of 203.5 U/L yielded a sensitivity of 63.6% and specificity of 71.3% for predicting POP, suggesting LDH's utility as a predictive marker for postoperative complications.

LDH and Delayed Cerebral Ischemia (DCI)

Study conducted by Anan et al (2020) on aSAH patients who developed DCI demonstrated slightly elevated LDH levels (222.1 ± 56.4 U/L) compared to non-DCI patients (214.7 ± 47.4 U/L). Additionally, LDH concentrations in carotid cisternal cerebrospinal fluid (CSF) were identified as potential predictive biomarkers for DCI, reflecting early brain injury (EBI).

LDH and Neurological Outcomes

Multiple studies explored the relationship between LDH levels and functional outcomes measured by the modified Rankin Scale (mRS) and Glasgow Outcome Scale (GOS):

- Study conducted by Zheng et al. (2021) and Zheng et al (2022). Poor mRS outcomes (scores 3–6) were associated with higher LDH levels compared to good outcomes (scores 0–2). For example, LDH levels for mRS 3–6 ranged from 205.918 ± 59.203 U/L to 234.188 ± 108.336 U/L
- Worse Hunt-Hess and Fisher grades, which indicate greater disease severity, were associated with progressively higher LDH levels. For instance, Hunt-Hess Grade V and Fisher Grade IV showed LDH levels of 252.851 ± 93.302 U/L and 376.806 ± 89.308 U/L, respectively as shown in studies conducted by Zheng et al (2021) and Zan et al (2022) respectively.
- Study conducted by Cavali et al (2023) shown that the Glasgow Outcome Scale (GOS) analysis showed a significant difference between favorable (176.05 ± 8.72 U/L) and unfavorable outcomes (215.29 ± 14.29 U/L), reinforcing LDH's role as a biomarker for poor recovery.

Risk of Bias

The risk of bias of included studies were conducted using Cochrane Risk of Bias in Non-randomized Studies of Interventions (ROBINS-I) Tool. Most domains indicated a low risk of bias, resulting in 4 studies being classified as "low risk of bias" and 3 studies as "moderate risk of bias" in the overall final results (Figure 2).

Discussion

Our findings show that the LDH value rises alongside the mRS score, Hunt-Hess grade, and Fisher grade, and is linked to postoperative pneumonia, delayed cerebral ischemia, and mortality rates. Zheng et al. (2021) observed higher mRS scores in individuals with elevated LDH levels (mRS 5-6: 234,188 ± 108,336 U/L vs mRS 0: 179,247 ± 46,761 U/L). Similarly, Zheng et al. (2022) reported a comparable trend (mRS 3-6: 227 ± 83,125 U/L vs mRS

0-2: 180,378 ± 50,695 U/L). Subarachnoid clots in the sulci and fissures cause spreading depolarizations and acute cerebral infarction in the nearby cortex after a cerebral aneurysm rupture.[15] An increase in ischemic lesions has been associated with higher mRS scores, which contributes to the clinical condition of patients with aSAH.[16] Zheng et al. (2021) reported that higher LDH levels were seen in patients with higher Hunt-Hess (HH) grades (HH Grade 1: 163,880 ± 35,571 U/L vs HH Grade V: 252,851 ± 93,302 U/L). This aligns with the findings of Multi et al. (2024), which involved 6,130 patients, showing that the Hunt-Hess grading system can serve as a predictor for both patient outcomes and mortality in subarachnoid haemorrhage (SAH). The study indicated that higher HH grades are linked to worse outcomes and increased mortality.[15,17] Neuronal apoptosis and necrosis in SAH result in the release of LDH into the bloodstream, leading to elevated LDH levels. These results suggest that serum LDH levels can serve as an indicator of the severity of brain tissue injury. [15,17]

Higher LDH levels were also observed in the group with a higher Fisher Grade. Zen et al. (2022) noted a significant difference in LDH values between Fisher Grade 4 and Fisher Grade 1 (376,806 ± 89,308 U/L vs. 37,989 ± 14,115 U/L, respectively). Similarly, Zheng et al. (2021) reported similar findings (Fisher Grade 4: 210,811 ± 68,962 U/L vs. Fisher Grade 1: 169,492 ± 41,621 U/L). In SAH, the disruption of the blood-brain barrier leads to the breakdown of red blood cells in the cerebrospinal fluid.[18,19] The LDH released from these lysed red blood cells is absorbed into the bloodstream, which raises LDH levels. Therefore, a higher Fisher Grade is linked to higher LDH values. [18,19]

The Glasgow Outcome Scale (GOS), which categorizes recovery from death to good recovery, is a key measure of long-term neurological outcomes in subarachnoid haemorrhage (SAH) patients. Cavali et al. (2023) emphasize the role of LDH as a reliable biomarker in predicting unfavorable outcomes, particularly those with GOS scores of 1–3 (215.29 ± 14.29 U/L) versus more favorable outcomes (176.05 ± 8.72 U/L for GOS 4–5). Elevated LDH levels in patients with unfavorable GOS outcomes likely reflect severe neuronal injury and metabolic disturbances. This is further corroborated by the association between higher LDH levels and worse Hunt-Hess and Fisher grades, both of which are established indicators of disease severity. Neuronal apoptosis and necrosis, hallmarks of SAH pathology, contribute to the release of LDH into the bloodstream, further linking LDH to poorer neurological recovery.[5]

In terms of mortality, the association with LDH levels is particularly striking. Zan et al. (2022) found that patients with elevated Fisher grades (Grade IV: 376.806 ± 89.308 U/L) had significantly higher 90-day mortality rates, indicating that LDH is not only reflective of immediate injury but also of progressive secondary brain damage, such as delayed cerebral ischemia (DCI) and systemic complications. This aligns with findings by Ren et al. (2023), where higher LDH levels

were associated with mortality within seven days post-discharge. The modest difference in LDH levels between mortality and non-mortality groups in Ren's study (239.8 ± 70.5 U/L vs. 236.3 ± 12.4 U/L) suggests that LDH may be more indicative of cumulative injury over time rather than acute events. [13,14]

Interestingly, LDH's role in predicting mortality is supported by its ability to reflect systemic stress responses, including inflammation, oxidative stress, and cellular hypoxia. Elevated LDH levels in such contexts provide a snapshot of metabolic derangements and widespread tissue injury, underscoring its value in mortality risk stratification.[13,20-22]

Postoperative pneumonia (POP), a significant complication following aneurysmal SAH, is another domain where LDH demonstrates prognostic value. Ding et al. (2019) observed a clear differentiation in LDH levels between patients who developed POP (261.26 ± 126.51 U/L) and those who did not (189.00 ± 69.20 U/L), with a threshold of 203.5 U/L offering predictive utility. The higher LDH levels in POP cases likely reflect the inflammatory cascade and immune dysregulation triggered by SAH, which predispose patients to infections. Given the high morbidity associated with POP, identifying patients at risk using biomarkers like LDH could facilitate early intervention strategies, such as prophylactic antibiotics or enhanced pulmonary care.[9]

Inflammatory markers are commonly used in clinical practice. Common examples include lactate dehydrogenase (LDH), procalcitonin, interleukins, among others. These markers are known to increase in a variety of conditions such as infection, tumour, and even in cases of subarachnoid haemorrhage. To date, no studies have directly compared the effectiveness of these markers in predicting prognosis, as each plays a significant role in disease progression. However, LDH continues to be widely utilized as an inflammatory marker due to its reliability in indicating tissue damage, its broad accessibility across various laboratory settings, and its comparatively lower cost relative to other inflammatory biomarkers.[23,24]

Incorporating LDH as part of routine admission assessments for aneurysmal SAH patients could enable more tailored management strategies. For instance, patients with significantly elevated LDH levels could be earmarked for intensive monitoring, early prophylactic measures against complications, and more aggressive therapeutic interventions. Furthermore, longitudinal monitoring of LDH trends during hospitalization might provide additional insights into disease progression and the effectiveness of interventions.

This study has several limitations. First, there are still few studies that examine the relationship between LDH levels and outcomes in subarachnoid haemorrhage (SAH). Additionally, limited research reports on subgroup outcomes, and more studies are needed to compare factors such as the mRS score, HH Grade, Fisher Grade, complications, and mortality in SAH patients to strengthen the findings. The cutoff value for LDH to determine

prognosis cannot be established due to varying outcomes across studies. Another issue is the unclear timing of LDH measurements, particularly regarding how long after the event they were taken. Future research should aim to clarify the timing of LDH measurements from the onset of SAH to provide more reliable evidence.

Conclusion

The relationship between LDH levels and outcomes in SAH patients highlights its potential as a prognostic biomarker in aneurysmal SAH. LDH not only reflects the severity of initial injury but also serves as a harbinger of secondary complications and outcomes. Its routine application could refine prognostication and guide individualized treatment strategies, ultimately improving patient outcomes.

Conflict of Interest

The authors declared no conflict of interest.

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Table 1. Keywords search term

Databases	Search terms
PubMed	("LDH"[All Fields] OR ("l lactate dehydrogenase"[MeSH Terms] OR ("l lactate"[All Fields] AND "dehydrogenase"[All Fields]) OR "l lactate dehydrogenase"[All Fields] OR ("lactate"[All Fields] AND "dehydrogenase"[All Fields]) OR "lactate dehydrogenase"[All Fields])) AND ("prognosis"[MeSH Terms] OR "prognosis"[All Fields] OR "prognoses"[All Fields] OR ("outcome"[All Fields] OR "outcomes"[All Fields])) AND ("subarachnoid haemorrhage"[All Fields] OR "subarachnoid haemorrhage"[MeSH Terms] OR ("subarachnoid"[All Fields] AND "haemorrhage"[All Fields]) OR "subarachnoid haemorrhage"[All Fields])
Europe PMC	(Lactate Dehydrogenase OR LDH) AND (prognosis OR outcomes) AND Subarachnoid haemorrhage
ScienceDirect	(Lactate Dehydrogenase OR LDH) AND (prognosis OR outcomes) AND Subarachnoid haemorrhage
Google Scholar	(Lactate Dehydrogenase OR LDH) AND (prognosis OR outcomes) AND Subarachnoid haemorrhage

Table 2. Characteristic of the study

Study ID	Study Design	Country	Sample Size	Median/Mean Age	Inclusion Criteria	Exclusion Criteria
Ding, et al (2019)	Prospective Cohort	China	647	54.82 ± 11.30	**(1)** subarachnoid haemorrhage was caused by intracranial aneurysm, as confirmed by computed tomography angiography or digital subtraction angiography; (**2)** patient was admitted to hospital within 7 days of symptom onset; (**3)** admission serum LDH level was obtained; (**4)** treatment of aneurysm (clipping or interventional) was performed.	**(1)** age <18 years old; **(2)** MV use or diagnosis of pneumonia before surgical treatment; **(3)** patient death within first 48 hours of admission; **(4)** previous use of antiplatelet or anticoagulant medication, steroids, or immunosuppressants; **(5)** history of other neurologic diseases, such as intracranial tumor, stroke, or severe head trauma; and; **(6)** other systemic diseases, such as autoimmune disease, uremia, cirrhosis, cancer, and chronic lung and heart diseases.

Anan, et al (2020)	Retrospective Cohort	Japan	19	NA	**(1)** patients within 14 days of acute SAH	**(1)** cases with suspected meningitis (elevated white blood cells in CSF); **(2)** posterior aneurysm location; **(3)** patients treated with endovascular
Zheng, et al (2021)	Retrospective Cohort	China	202	NA	**(1)** diagnosis of SAH confirmed by CT; **(2)** presence of intracranial aneurysms confirmed using CT angiography (CTA) or digital subtraction angiography (DSA); **(3)** all aneurysms treated using microsurgical clipping; **(4)** CTA and/or DSA performed postoperatively; **(5)** patients were admitted 24 h after the onset of SAH.	**(1)** aSAH detected > 24 h after occurrence; **(2)** other cerebrovascular diseases (such as cerebral arteriovenous malformations, intracranial arteriovenous fistula, or moyamoya syndrome/disease) or intracranial tumors; **(3)** history of myocardial infarction, hepatitis, malignant tumor, pulmonary infarction, leukemia, hemolytic anemia, kidney disease, or progressive muscular atrophy
Zheng, et al (2022)	Retrospective Cohort	China	856	54.5 (10–86)	**(1)** aneurysmal subarachnoid haemorrhage (aSAH) was diagnosed by computed tomography (CT) and computerized tomography angiography (CTA) or digital subtraction angiography (DSA); **(2)** the patients were admitted 24 h after the occurrence of SAH; (3) cerebral aneurysms were treated by microsurgical clipping.	**(1)** aSAH detected > 24 h after the onset; **(2)** the presence of intracranial tumors in patients with other cerebrovascular diseases (such as intracranial arteriovenous malformations, arteriovenous fistula, and Moyamoya syndrome/disease); **(3)** patients with myocardial infarction, pulmonary infarction, hepatitis, kidney disease or progressive muscular atrophy, malignant tumor, leukemia, hemolytic anemia, etc.
Zan, et al (2022)	Retrospective Cohort	China	3524	51.77 (11.68) low ldh vs 56.76 (11.99) high ldh	(1) subarachnoid haemorrhage diagnosed via head computed tomography, magnetic resonance imaging, or angiography, and an aneurysm confirmed with cerebral angiography, magnetic resonance angiography, or computed tomography	(1) aneurysms caused by trauma or arteriovenous malformations; (2) fusiform aneurysms, and nondefinitive aneurysms; aneurysms were treated before ictus or; (3) lack of admission LDH, (4) wrong or non-existent registration in sichuan province

					angiography.	
Cavali, et al (2023)	Retrospective Cohort	Belgium	547	54.044 (±12.9)	1) age > 18 years; 2) diagnosis of ruptured aneurysm as the primary cause of SAH on computed tomography (CT) with angiographic confirmation (either computed tomography angiography or cerebral angiography)	1) pregnancy; 2) patients without 3 months follow up assessment reported in the medical records.
Ren, et al (2023)	Retrospective Cohort	China	190	62 [53–68]	(1) aSAH confirmed on brain computed tomography (CT) and CT angiography or digital subtraction angiography; (2) age ≥18 years	(1) incomplete or unavailable records; (2) refusal to consent; or (3) late admission (>7 days after the ictus of aSAH)

Table 3. Outcome measurement and LDH Values

Study, ID	Type of SAH	LDH Taken	Measured Outcome	LDH Values	Major Findings
Ding, et al (2019)	Aneurysmal Subarachnoid Haemorrhage	Admission	Postoperative Pneumonia (POP)	• **w/o POP**: 189.00 ± 69.20 U/L • **POP**: 261.26 ± 126.51 U/L	LDH might be a helpful predictor of POP occurrence in patients with aSAH. LDH level had a sensitivity of 63.6% and a specificity of 71.3% for predicting POP based on best threshold of 203.5 U/L associated with POP in patients with aSAH, even after adjusting for possible confounding factors.

Anan, et al (2020)	Aneurysmal Subarachnoid Haemorrhage	14 days post operation	Delayed Cerebral Ischemia (DCI)	• **Non-DCI**: 214.7 ± 47.4 U/L • **DCI**: 222.1 ± 56.4 U/L	Venous LDH was found to be higher in the DCI group. Lactate and LDH concentrations in carotid cisternal CSF may vividly reflect EBI and thus may provide predictive biomarkers for DCI following aSAH
Zheng, et al (2021)	Aneurysmal Subarachnoid Haemorrhage	Admission	Clinical Outcome based on mRS 0-2: Good Outcome 3-6: Poor Outcome	• **Good Outcome**: 205.356 ± 76.785 U/L • **Poor Outcome**: 227.119 ± 86.469 U/L	Serum levels of LDH correlated with Hunt–Hess grade, Fisher grade, and neurological functional outcome, and predicted the outcome of aSAH.
			Functional Outcome based on mRS 0: no symptoms 1-2: slight disability 3-4: moderate to serious disability 5-6: Severe Disability to death	• **mRS 0:** 179.247 ± 46.761 U/L • **mRS 1-2:** 193.977 ± 69.399 U/L • **mRS 3-4:** 205.918 ± 59.203 U/L • **mRS 5-6**: 234.188 ± 108.336 U/L	Serum levels of LDH correlated with Hunt–Hess grade, Fisher grade, and neurological functional outcome, and predicted the outcome of aSAH. Serum LDH was higher in patients, with higher mRS Score, howerver phosphate level might play a role. The LDH to phosphate ratio was a potential biomarker and could predict the unfavorable outcome of microsurgical clipping for rIA in 3 month.

			Hunt-Hess Grade	• **HH Grade 1:** 163.880 ± 35.571 U/L • **HH Grade II:** 174.981 ± 49.616 U/L • **HH Grade III:**188.306 ± 50.702 U/L • **HH Grade IV**:225.609 ± 69.509 U/L • **HH Grade V:** 252.851 ± 93.302 U/L	
			Fisher Grade	• **Fisher Grade 1:** 169.492 ± 41.621 U/L • **Fisher Grade 2:** 177.097 ± 42.621 U/L • **Fisher Grade 3:** 198.709 ± 72.553 U/L • **Fisher Grade 4:** 210.811 ± 68.962 U/L	
Zheng, et al (2022)	Aneurysmal Subarachnoid Haemorrhage	Admission	mRS Score 0-2 : Good Outcome 3-6 : Poor Outcome	• **Good Outcome:** 180.378 ± 50.695 U/L • **Poor Outcome:** 227 ± 83.125 U/L	Serum LDH was seen higher in Poor outcome group patients. LDH may be related to neuronal damage, cerebral hypoxia, and early brain injury after aneurysm ruptures.

Zan, et al (2022)	Aneurysmal Subarachnoid Haemorrhage	Admission	Mortality at 90 days	• **Fisher Grade 1:** 37.989 ± 14.115 U/L • **Fisher Grade 2:** 135.508± 18.017 U/L • **Fisher Grade 3:** 103.865 ± 23.384 U/L • **Fisher Grade 4:** 376.806 ± 89.308 U/L	In patients with aSAH, both baseline and longitudinal high-serum LDH levels were associated with all-cause mortality. Mortality was seen higher in patients with higher LDH at admission.
Cavali, et al (2023)	Aneurysmal Subarachnoid Haemorrhage	Admission	**GOS Score** 1: dead 2: persistent vegetative state 3: severe disability 4: moderate disability 5: good recovery 1-3: Unfavorable Outcome 4-5: Favorable Outcome	• **Favorable Outcome:** 176.05 ±8.72 U/L • **Unfavorable Outcome:** 215.29 ± 14.29 U/L	Lactate dehydrogenase is an easily available biomarkers for short term neurological outcome in acute SAH in and hospital mortality

			In Hospital Mortality	• **Non-mortality group:** 180.1 ± 9.7 U/L • **Mortality group:** 221.58 ± 15.45 U/L	
Ren, et al (2023)	Aneurysmal Subarachnoid Haemorrhage	Admission	Mortality 7 days after discharge	• **Non-mortality group:** 236.3 ± 12.4 U/L • **Mortality Group:** 239.8 ± 70.5 U/L	Laboratory screening may provide a useful tool for the management of aSAH patients requiring MV in stratifying risk levels for mortality and for better clinical decision-making

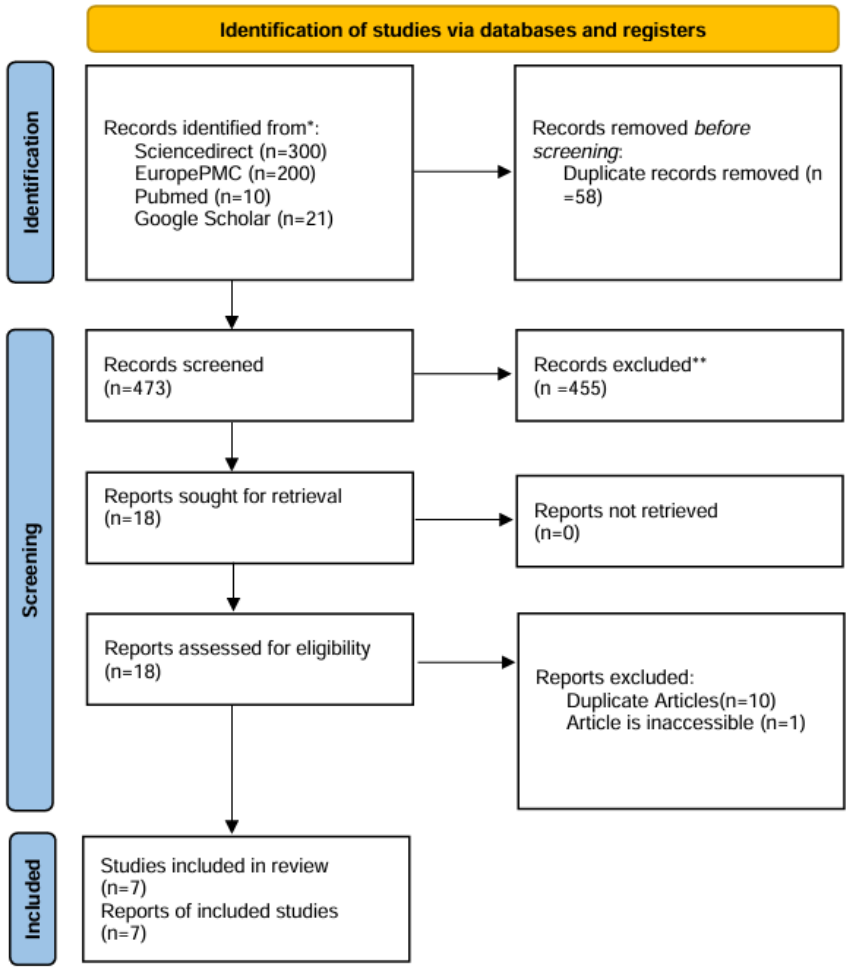

Figure 1. PRISMA Guidelines

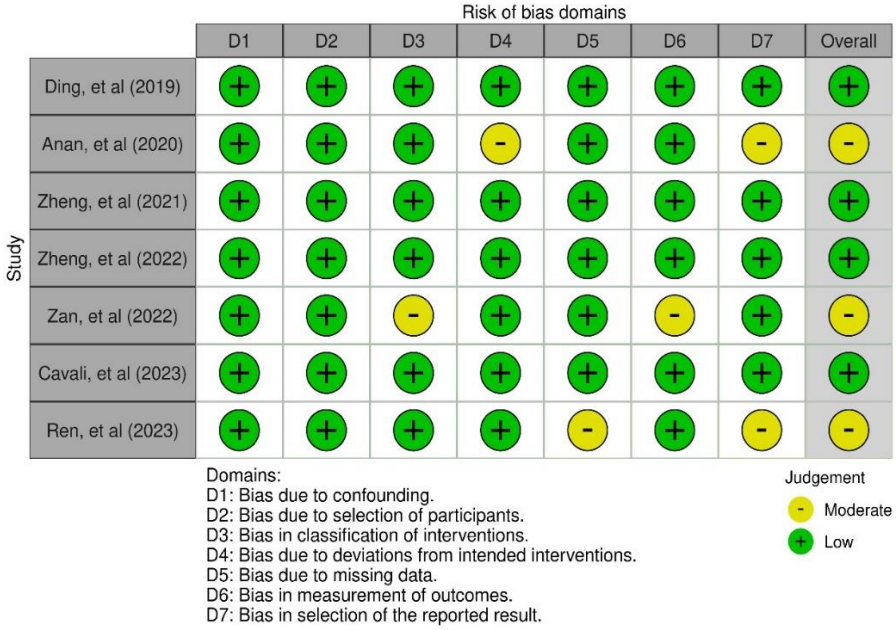

Figure 2. Risk of Bias of each study