

Haematoma attenuation on non-contrast CT and blood-count inflammatory ratios in spontaneous intracerebral haemorrhage: A systematic review of prognostic studies

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Abstract

Background: Early severity in spontaneous intracerebral haemorrhage (ICH) is shaped by two processes that standard scores capture poorly: instability of the clot, visible as low attenuation of the haematoma on non-contrast CT, and the systemic inflammatory response, reflected in routine blood-count ratios. We reviewed how haematoma attenuation and the neutrophil-to-lymphocyte ratio (NLR), lymphocyte-to-monocyte ratio (LMR) and mean platelet volume (MPV) relate to clinical severity in adults with spontaneous ICH.

Methods: PubMed/MEDLINE, Scopus, Web of Science, Cochrane CENTRAL, Google Scholar and Consensus were searched to 31 August 2026 for cohort studies with at least 50 participants. Risk of bias was appraised with QUIPS, synthesis was narrative following SWiM, and certainty was rated with GRADE for prognostic factors.

Results: Twenty studies (10,002 participants) were included. Low haematoma attenuation was independently associated with haematoma expansion, both on visual assessment (adjusted odds ratios 2.71 to 4.37) and on measurement (odds ratio 0.64 per 1 HU increase; moderate certainty). Higher NLR was associated with poor functional outcome and death (low certainty), with thresholds of 5.63 to 6.49 that were all derived from the study data. Low LMR was associated with early neurological deterioration, possibly in a non-linear way, and the only MPV study found no association with prognosis.

Conclusion: Haematoma attenuation relates mainly to expansion, and inflammatory ratios mainly to deterioration and outcome. Whether combining them adds value rests on one unreplicated model, and evidence on MPV is lacking.

Introduction

Spontaneous intracerebral haemorrhage (ICH) makes up only 10% to 20% of strokes, yet it causes a share of death and disability far larger than that. Historical 30-day mortality is 35% to 50%, and only about one survivor in five is independent at six months. In Asian populations, Indonesia included, ICH forms a larger proportion of strokes than in European ones, largely because hypertension is so often uncontrolled. Current guidance centres early care on blood pressure control, reversal of coagulopathy, management in a stroke or neurocritical care unit, and selection for surgery [1]. All of this depends on judging severity quickly and well. Scores such as the ICH score rely mostly on variables fixed at admission and say little about the two processes that drive the course of the disease over the first 24 to 72 hours.

The first of these is instability of the clot, which ends in haematoma expansion and can be estimated from the density of the haematoma on non-contrast CT (NCCT). Blood attenuation depends mainly on haemoglobin concentration and on how far the clot has retracted, so areas of low Hounsfield unit (HU) value inside or at the edge of a haematoma point to fresh, unretracted blood mixed with serum. These markers range from visual signs, such as intrahaematoma hypodensity and the black hole, blend and

swirl signs, to measured values such as minimum, mean and relative attenuation. In this review we refer to all of them as haematoma attenuation. Pooled data have linked them with expansion and poor outcome [2].

The second process is inflammation. Within hours of bleeding, haemoglobin breakdown products, thrombin and matrix components activate microglia, open the blood-brain barrier and draw leucocytes into the perihematoma tissue. The response shows up in peripheral blood. Stress-driven neutrophilia and lymphopenia raise the neutrophil-to-lymphocyte ratio (NLR), relative loss of lymphocytes lowers the lymphocyte-to-monocyte ratio (LMR), and platelet activation increases the mean platelet volume (MPV). All three come from the full blood count taken at almost every stroke admission, so they cost nothing extra and are available within minutes. In settings with limited resources that makes them far more practical than interleukin-6 or matrix metalloproteinase-9.

The imaging and blood-marker literatures have grown up side by side but rarely meet. Imaging studies predict expansion without inflammatory variables, and blood-marker studies look at outcome and death without considering haematoma density. A meta-analysis of 26 studies found NLR associated with poor outcome, death and neurological deterioration but not with expansion [3], while pooled imaging data showed a strong link between density markers and expansion [2]. The open question is therefore not whether each marker relates to outcome, but whether the two carry different information. We set out to review the association of haematoma attenuation and blood-count inflammatory ratios with clinical severity in adults with spontaneous ICH.

Methods

Protocol

The review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement [4]. A written protocol setting out the question, eligibility criteria, search, selection, extraction form, risk-of-bias tools and synthesis plan was prepared before the search began and kept at the authors' institution. It was not registered prospectively. Ethical approval was not needed because only published aggregate data were used.

Eligibility criteria

We included studies of adults aged 18 years or older with CT-confirmed spontaneous ICH treated in the acute phase that measured at least one of haematoma attenuation on baseline NCCT, LMR, NLR or MPV, and that reported at least one dimension of clinical severity with an explicit quantitative definition. Randomised trials and prospective or retrospective cohorts with multivariable analysis were eligible if they had at least 50 participants and full text in English or Indonesian. Clinical severity was treated as five dimensions: baseline severity, haematoma expansion, early neurological deterioration, secondary complications and final outcome. We excluded case reports and case series without comparison, narrative reviews, editorials, letters and protocols, conference abstracts without a full paper, secondary

ICH, primary subarachnoid haemorrhage, ischaemic stroke, paediatric and mixed stroke populations without separate ICH results, preclinical work, and reports whose population overlapped fully with an included study.

Search strategy

The search combined Medical Subject Headings with free-text terms in four blocks: population, imaging exposure, blood-marker exposure and severity outcome. The full strategy for each database, with the number of records retrieved, is given in Supplementary Table S1. The last search was run on 31 August 2026. A language filter was applied automatically only in PubMed/MEDLINE; elsewhere language was checked by hand. Reference lists of included articles and of earlier systematic reviews were also searched by hand.

Study selection and data extraction

After removal of duplicates, records were screened by title and abstract. This stage was done by a single reviewer rather than two. To limit error, the eligibility criteria were made operational before screening and the stage was repeated after an interval to check consistency, but an inter-rater agreement statistic could not be calculated. All reports read in full were reviewed again by the supervising author, and disagreements were settled by discussion. Data were extracted onto a piloted form: study identity, design and data source, sample size and demographics, timing of blood sampling and imaging, definition of each exposure, proposed thresholds and how they were derived, outcome definitions and frequency, crude and adjusted estimates with 95% confidence intervals (CIs), adjustment variables, and discrimination statistics.

Risk of bias and certainty of evidence

Because every included study asked a prognostic question, risk of bias was appraised with the Quality in Prognosis Studies (QUIPS) tool across its six domains, each rated low, moderate or high [5]. Methodological quality was also scored on the Newcastle-Ottawa Scale for readers who use it [6]. Certainty was rated for each exposure and outcome with the GRADE approach for prognostic factors. Following that guidance, evidence from cohort studies started at high certainty and was rated down for risk of bias, inconsistency, indirectness, imprecision and suspected publication bias [7]. The reason for each judgement is given in Table 4.

Synthesis

We judged before the search whether pooling would be possible and decided against it. Exposures were defined in many different ways (visual signs, minimum, mean or relative HU, and blood ratios taken at different times), regions of interest varied, severity was measured on different dimensions, and some publications from the same groups shared patients. The synthesis was therefore narrative and followed the Synthesis Without Meta-analysis (SWiM) guideline [8]. Studies were grouped by exposure and by severity dimension; adjusted odds ratios and discrimination statistics were the main metrics; and

direction of effect, precision and dose-response were used to judge strength of evidence. Adjusted estimates are shown in a forest plot without a summary estimate. Most thresholds were taken from receiver operating characteristic analysis in the same data, so they are reported as published and treated as exploratory [9].

Results

Study selection and characteristics

The database search found 1,062 records (PubMed/MEDLINE 486, Scopus 213, Web of Science 187, Cochrane CENTRAL 34, Google Scholar and Consensus 142), and hand-searching added 8. After 318 duplicates were removed, 752 records were screened and 641 excluded. Of 111 reports sought, 6 could not be obtained; of the 105 read in full, 85 were excluded, most often because neither attenuation nor an inflammatory ratio was measured ($n = 24$), the paper was a review, editorial or letter ($n = 14$), or it was a case report or series ($n = 12$). Twenty studies were included (Fig. 1, Table 1).

The 20 studies, published between 2016 and 2026, enrolled 10,002 participants. Seven addressed haematoma attenuation, ten inflammatory ratios, one MPV, and two combined imaging and blood markers. Most were retrospective cohorts; four used prospective registries or included prospective validation. Samples ranged from 83 to 2,465. The studies came from China, the Republic of Korea, the United States, Romania and Portugal, and one pooled cohort drew on eight university hospitals in five countries. Two imaging reports share patients from the same Boston cohort, so the participant total slightly overstates the number of distinct patients.

Risk of bias

Prognostic factor measurement and outcome measurement were mostly at low risk of bias (Table 2). Study participation was usually at moderate risk because of retrospective designs. Confounding was at moderate or high risk in several blood-marker studies that did not adjust for infection, glucose or corticosteroid use. Statistical analysis was at moderate risk where thresholds were derived and tested in the same data.

Haematoma attenuation

On visual assessment, intrahaematoma hypodensity was independently associated with expansion in 1,029 patients (adjusted odds ratio [OR] 3.42, 95% CI 2.21 to 5.31; kappa 0.87), with a similar result in a second cohort (OR 4.37, 95% CI 2.05 to 9.62) [10]. In a pooled cohort of 2,465 patients, hypodensity was present in 44.0% and was associated with expansion (OR 3.11, 95% CI 2.55 to 3.80); adding it raised the area under the curve (AUC) of a four-variable clinical model from 0.67 to 0.71 [11]. These two reports are not fully independent because the Boston cohort contributes to both.

Measured attenuation gave consistent results across metrics (Fig. 2). A minimum attenuation of 31 HU or less was associated with both expansion and poor 90-day outcome, with specificity of 92.0% and

sensitivity of 64.0% [12]. Each 1 HU increase in mean attenuation lowered the odds of expansion by 36% (adjusted OR 0.64, 95% CI 0.51 to 0.80) and raised the C statistic from 0.66 to 0.84 [13]. Measured density was associated with expansion in both small and large haematomas, including those in which visual signs were uncommon [14]. Attenuation of the non-hypodense region below 64 HU [15] and relative attenuation of 33.35 HU or less [16] were also associated with expansion, but discrimination was modest (AUC 0.589 and 0.665).

Inflammatory ratios and MPV

Two studies related admission NLR to expansion, both positively and with similar effect sizes (OR 1.22, 95% CI 1.09 to 1.38, threshold 6.49; and OR 1.19, 95% CI 1.12 to 1.27, threshold 5.63, AUC 0.84) [17, 18]. This differs from a previous meta-analysis that found no association (OR 1.04, 95% CI 0.99 to 1.08) [3]. Findings on final outcome were more consistent. Admission NLR was associated with 90-day functional outcome in patients treated surgically [19]. In one cohort admission NLR was not associated with in-hospital death after adjustment, whereas NLR at 72 hours was (OR 1.2, 95% CI 1.09 to 1.40; AUC 0.819; threshold 6.3); that analysis was limited to the 112 of 201 patients who survived 72 hours and is open to survivor bias [20]. The systemic inflammation response index was associated with both 3-month outcome and 1-month death, but NLR only with outcome [21]. Among 869 critically ill patients, NLR discriminated intensive care mortality best of five indices [22]. NLR was not associated with perihaematoma oedema after adjustment, although it remained associated with 90-day outcome [23].

Four studies examined LMR. Admission LMR was associated with neurological deterioration in the first week and with 90-day death, at thresholds of 2.21 and 2.19, and discriminated early deterioration better than death [24]. In 640 registry patients, a quartile analysis showed a J-shaped relation: the second quartile, not the highest, carried the lowest risk of poor outcome and death, and the lowest quartile the highest risk of death and hospital-acquired infection [25]. In critically ill patients LMR performed less well than NLR [22]. In basal ganglia haemorrhage, none of NLR, LMR or the platelet-to-lymphocyte ratio was associated with oedema growth, but baseline NLR discriminated 90-day outcome (AUC 0.854) [26]. A composite score built from six inflammatory markers was associated with expansion, deterioration at 48 hours, 30-day death and 3-month outcome, and performed better than most single markers [27]. Only one study qualified for MPV. MPV above 13 fL was associated with a 5.21-fold higher risk of haemorrhagic stroke occurring, but neither MPV nor platelet count was associated with prognosis [28].

Combined imaging and inflammatory models

A nomogram combining Glasgow Coma Scale score, NLR, the blend and swirl signs and hypodensity reached an AUC of 0.908 (95% CI 0.880 to 0.936) in a development cohort of 506 patients and 0.844 (95% CI 0.760 to 0.908) on external validation in 103 [29]. Hypodensity contributed an adjusted OR of 2.71 (95% CI 1.41 to 5.22). The second combined study found no relation with oedema dynamics, though baseline NLR discriminated 90-day outcome better than haematoma volume (AUC 0.854 vs 0.788) [26]. Proposed thresholds differed by marker, timing of measurement and outcome (Table 3), and specificity was generally higher than sensitivity.

Certainty was moderate for low haematoma attenuation and expansion. It was low for attenuation and functional outcome, for NLR and functional outcome or death, for NLR and expansion, for LMR, and for the composite and combined models, and very low for MPV (Table 4). Risk of bias, mostly from retrospective designs and incomplete adjustment, and inconsistency in sampling time and outcome definition were the main reasons for rating down.

Discussion

Imaging and blood markers in ICH have mostly been studied in separate literatures. Taken together, the studies in this review suggest that the two groups of markers relate to different dimensions of severity. Haematoma attenuation was associated mainly with structural progression, and inflammatory ratios mainly with neurological deterioration, functional outcome and death. Part of the apparent conflict in the literature may come from not separating these dimensions, so that a marker that looks strong in one study looks weak in another that measured something else.

Haematoma attenuation shows the state of the clot at the moment of the scan. It reflects how far the clot has retracted and whether fresh blood is present, both of which bear directly on whether bleeding is still going on. Blood ratios reflect a process that differs in both time and place. Admission NLR probably mirrors the early neuroendocrine stress response to the size of the injury, while NLR on day three reflects the secondary inflammatory cascade that has developed since. The stronger association of 72-hour NLR than admission NLR with death [20] fits this view, although survivor bias limits what that comparison can show. In practical terms, attenuation relates to whether a haematoma is likely to grow in the next few hours, and NLR to how severe the systemic response is and how likely complications are.

Links between the two pathways are plausible but remain hypotheses. Haemoglobin concentration directly affects HU values, and mean HU appears to mediate part of the relation between haemoglobin and expansion [30]. The tissue at the haematoma margin that looks dense or hypodense early is also where leucocytes and oedema accumulate later. Thrombin, finally, takes part both in clot retraction and in microglial activation. None of the included studies measured both groups of markers in a way that would test these links.

Inconsistent timing of blood sampling is one of the largest sources of variation in the blood-marker studies. Serial studies show that NLR rises and then falls, peaking at about 48 hours [31, 32], so values from different time points are not interchangeable. A threshold of 5.63 for samples from the emergency department [18] cannot be applied to samples on day three, for which 6.3 was proposed [20], and neither compares with the 12.35 to 14.46 reported after surgery [31]. This may explain why a pooled analysis found no association between NLR and expansion [3] while studies using early samples did. Studies of NLR or LMR in ICH should report sampling time from symptom onset, not from arrival. All thresholds in this review were also derived from the data in which they were tested, and none was validated externally, so they are likely to look better than they will in new patients [9, 18].

The J-shaped LMR pattern deserves attention because most biomarker work assumes a straight-line relation [25]. Very low LMR may reflect stroke-induced immunodepression and greater risk of infection, and very high LMR a relative lack of monocytes that slows clot clearance. If this is confirmed, a single cut-off such as 2.21 [24] oversimplifies the relation, and quartiles or splines would be more suitable.

Only one study qualified for MPV, even though MPV is part of the review question. This reflects the literature rather than the search. MPV rises with storage time in EDTA, varies between analysers and lacks international standardisation. Most MPV research has addressed ischaemic stroke and aneurysmal subarachnoid haemorrhage; MPV did not change in primary ICH while it rose in cortical infarction [33], and it was associated with poor outcome in subarachnoid haemorrhage [34], a population we excluded. In ICH the expected direction is also unclear, since more active platelets should in theory help haemostasis. Absence of evidence here is not evidence of absence.

The combined nomogram reached the highest discrimination in this review [29], but it comes from a single two-centre Chinese population, external validation included only 103 patients, and the fall from 0.908 to 0.844 is the usual sign of optimism. Because three imaging signs entered the model together, the added value of NLR cannot be separated out. Certainty is low, and the result should be treated as a hypothesis to test rather than as an established finding.

The clinical message is cautious. Both NCCT and the full blood count are already part of routine care and need no contrast, special reagents or software. On current evidence, haematoma hypodensity together with a raised NLR might reasonably prompt closer observation and an earlier repeat scan, but no study has shown that acting on these markers' changes outcome, so they are prognostic rather than predictive of treatment benefit. NLR and LMR are also affected by infection, corticosteroids, malignancy and autoimmune disease, and they predict stroke-associated pneumonia [35], so a high value may signal infection as much as injury severity. Normal values do not exclude risk.

Limitations

Some limitations belong to the primary studies. Most were retrospective and single-centre, fifteen of twenty came from China or the Republic of Korea, and none came from Southeast Asia, where anaemia and endemic infection may shift baseline blood ratios. Adjustment for infection, glucose and corticosteroids was inconsistent, thresholds were derived in the same data, and few studies reported CIs for sensitivity and specificity. These problems lower certainty mainly through risk of bias and inconsistency, and they probably make the reported associations look stronger than they are.

Other limitations concern our own methods. Title and abstract screening were done by one reviewer, which raises the chance that eligible studies were missed. The protocol was not registered. Google Scholar and Consensus searches are harder to reproduce than database searches. We did not pool the data, so no summary estimate or measure of heterogeneity is given, and with few studies per exposure, small-study effects and selective publication could not be tested; if present, they would also push the reported associations upwards. Evidence was unbalanced across domains, with ten studies on NLR and

one on MPV, and no study measured all the markers in the same patients, so our view that the markers are complementary is inferred rather than shown. These limitations affect mainly how complete and precise the evidence is.

Future work should measure haematoma attenuation and all inflammatory ratios together in one prospective cohort, with blood sampling at fixed times from onset. MPV needs dedicated study with standardised handling, thresholds need validation in Southeast Asian patients, and any combined model should be simple enough to use within the first hour and should be validated externally.

Conclusion

In spontaneous ICH, low haematoma attenuation on NCCT is associated mainly with haematoma expansion, with moderate certainty, while higher NLR and lower LMR are associated mainly with neurological deterioration, poor functional outcome and death, with low certainty. The two groups of markers appear to reflect different processes and may be complementary, but the only model combining them has not been replicated. Evidence on MPV in spontaneous ICH remains very limited. Prospective studies that measure both groups of markers in the same patients, with standardised sampling times and externally validated thresholds, are needed.

Declarations

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Competing interests the authors have no relevant financial or non-financial interests to disclose.

Ethics approval Not applicable. This is a systematic review of published studies and involved no new data collection from human participants.

Consent to participate Not applicable.

Consent for publication Not applicable.

Data availability All data analysed in this review are taken from the published studies cited. The extraction form and extracted data are available from the corresponding author on reasonable request.

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Tables

Tables are available in the Supplementary Files section.

Figures

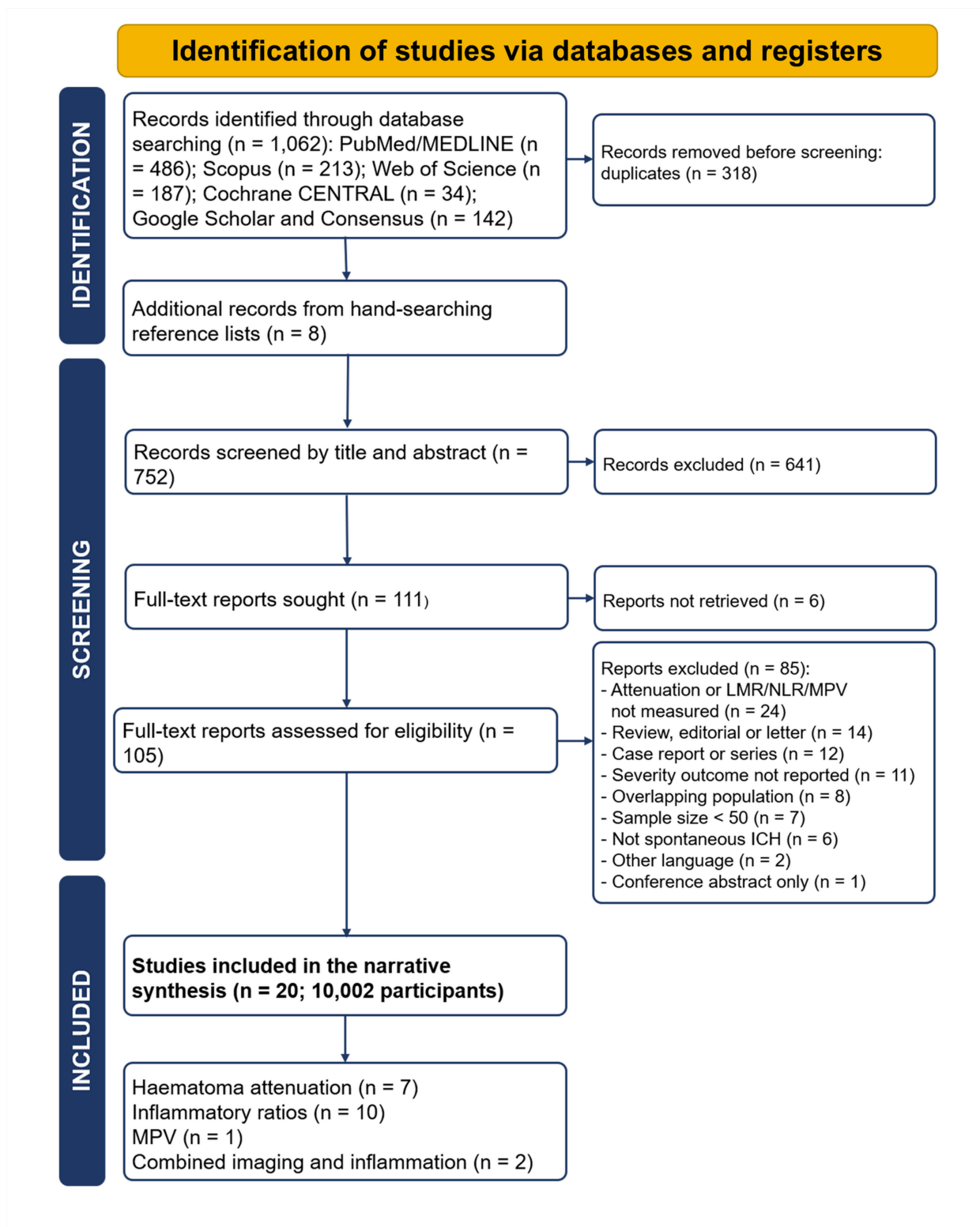
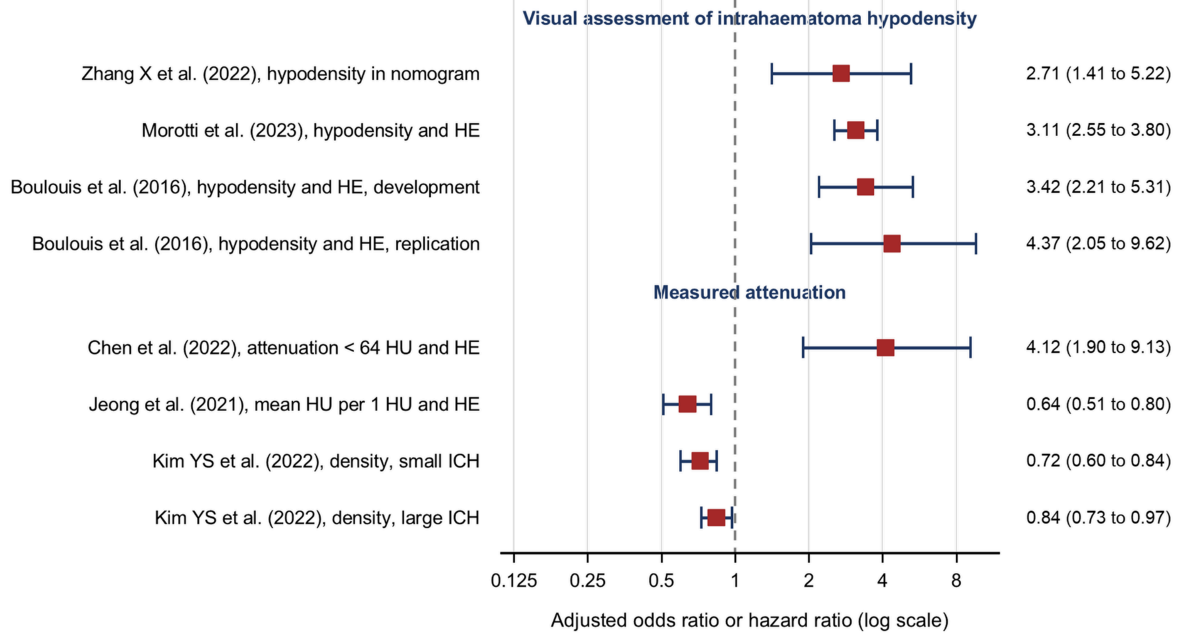


Figure 1

PRISMA 2020 flow diagram of study selection

A. Haematoma attenuation on non-contrast CT



B. Inflammatory ratios (NLR, PLR, LMR, SIRI)

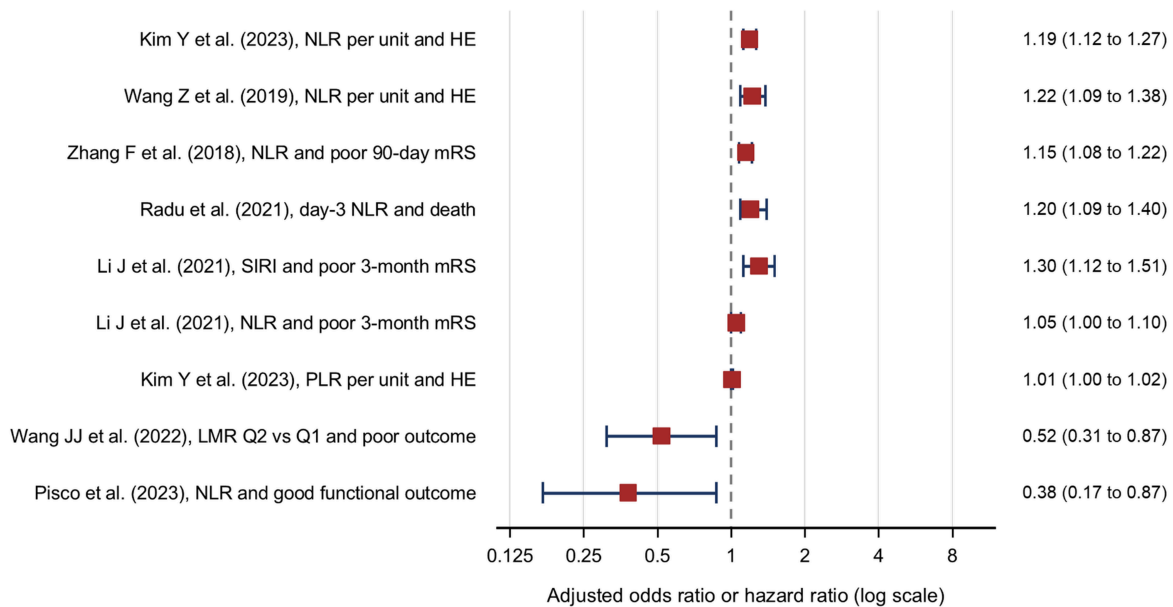


Figure 2

Adjusted estimates from the included studies, by exposure domain. The plot is descriptive and no pooled estimate is shown because exposure and outcome definitions differed. In panel A, estimates below 1 for measured attenuation mean that higher attenuation is associated with lower odds of expansion. In panel B, the estimate of Wang JJ et al. (2022) means that a slightly raised LMR is associated with better outcome, and the estimate of Pisco et al. (2023) is expressed against good functional outcome, so a value below 1 means that higher NLR is associated with worse outcome. HE, haematoma expansion; HU,

Hounsfield unit; ICH, intracerebral haemorrhage; LMR, lymphocyte-to-monocyte ratio; mRS, modified Rankin Scale; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; SIRI, systemic inflammation response index

Supplementary Files

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