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Timing Is Everything: A Systematic Review of Optimal Repeat Computed Tomography Protocols in Traumatic Brain Injury

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Abstract

Traumatic brain injury (TBI) remains a global health challenge, with computed tomography serving as the primary diagnostic tool for initial evaluation. However, significant variability exists in repeat computed tomography (CT) scanning protocols, ranging from routine scheduled imaging to selective approaches based on clinical deterioration. This systematic review synthesized evidence from 1247 initially identified records, ultimately including 26 studies that met inclusion criteria, to determine optimal timing strategies for repeat CT scanning in patients with TBI. The analysis revealed dramatic heterogeneity in hemorrhagic progression rates (0.4–65%) and intervention requirements across studies, largely explained by differences in TBI severity. Patients with mild TBI (Glasgow Coma Scale [GCS] 13–15) demonstrated consistently lower progression rates (0.4–42%), intervention rates (0.13–0.9%), and mortality (0.13–1.2%) compared with moderate–severe TBI cohorts, which exhibited progression rates of 42.3–61%, intervention rates of 8.9–24%, and mortality of 13–18%. Critical temporal patterns emerged, with Fletcher-Sandersjöö demonstrating that 94% of hematomas ceased progressing within 24 h postinjury, establishing a crucial surveillance window. Multiple predictors of progression were identified, including concomitant intracranial lesions (subarachnoid hemorrhage odds ratio [OR] 3.28, subdural hemorrhage OR 4.35), advanced age, and antiplatelet therapy. Notably, patients undergoing initial CT scanning within 2–3 h postinjury showed higher rates of subsequent progression, suggesting that early scans warrant scheduled follow-up regardless of clinical status. These findings support severity-stratified approaches to repeat imaging, with routine protocols potentially justified in moderate–severe TBI, while selective strategies may be appropriate for patients with stable mild TBI. The evidence emphasizes balancing diagnostic yield against radiation exposure concerns, advocating for personalized protocols based on individual risk factors rather than universal approaches.

Keywords: computed tomography; hemorrhage progression; repeat imaging; risk stratification; timing protocols; traumatic brain injury

Introduction

Traumatic brain injury (TBI) remains a leading cause of mortality and morbidity worldwide, with an incidence reported at approximately 350 per 100,000 population.[1] Computed tomography (CT) has established itself as the primary diagnostic modality for the initial evaluation of patients with suspected TBI, enabling rapid detection of intracranial pathologies that may require urgent neurosurgical intervention. While the role of initial CT scanning in TBI management is well established, there is ongoing debate regarding the optimal approach to repeat imaging, particularly in patients with mild to moderate TBI who present with stable neurological status.[2,3]

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Current clinical practice demonstrates significant variability in the utilization of repeat CT scans. Some institutions implement standardized protocols requiring routine follow-up imaging at predetermined intervals (typically 6–24 h after the initial scan), regardless of clinical status. Other centers adopt a more selective approach, reserving repeat imaging for patients who demonstrate neurological deterioration.[4,5] This variability reflects the lack of robust evidence-based guidelines regarding the timing and necessity of repeat CT scanning in TBI management.

Several studies have attempted to evaluate the clinical utility of routine versus selective repeat CT scanning. Brown et al. reported that while repeat CT scans prompted by neurological deterioration led to interventions in approximately 38% of cases, routine scans resulted in management changes in only 1–2% of patients.[6] Similarly, a more recent study by Beedkar et al. found that routine repeat CT scans led to surgical intervention in just 3.5% of cases, raising questions about resource utilization and cost-effectiveness.[1] Moreover, the cumulative radiation exposure associated with multiple CT scans presents additional concerns regarding potential long-term cancer risks, particularly in younger populations.[7]

Recent guidelines, including those from the Scandinavian Neurotrauma Committee, have recommended repeat imaging within 6–8 h for specific high-risk patients, such as those with epidural hematomas managed nonoperatively.[2] However, substantial heterogeneity persists in both recommendations and clinical practice. The optimal timing for repeat imaging remains unclear, with studies reporting intervals ranging from 4 to 48 h.[8]

Several factors may influence the decision-making process regarding repeat CT scanning, including initial CT findings, Glasgow Coma Scale (GCS) score, anticoagulation status, age, and mechanism of injury. Identification of specific risk factors that predict deterioration could enable more targeted use of repeat imaging, potentially improving resource allocation without compromising patient outcomes.[8–10]

This systematic review aims to synthesize the current evidence regarding the optimal timing of repeat CT scanning in patients with TBI, to inform evidence-based protocols that balance diagnostic yield with resource utilization and patient safety considerations.

Material and Methods

Search strategy and study selection

A comprehensive literature search was conducted following Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines in PubMed/MEDLINE, Cochrane Library, EMBASE, and clinical trials databases from inception to March 2025. The search strategy included combinations of MeSH terms and keywords related to primary terms: "repeat CT scan," "traumatic brain injury," "head trauma," and "timing." Secondary search terms included "follow-up imaging," "surveillance CT," "serial CT," and "neurological deterioration." We also manually searched reference lists of included studies and relevant reviews to identify additional eligible studies.

Studies were eligible for inclusion if they met the following criteria: (1) systematic reviews and meta-analyses, randomized controlled trials, prospective cohort studies, retrospective analyses, or clinical practice guidelines; (2) patients with TBI requiring repeat CT imaging; (3) reporting of timing protocols for repeat CT scans; and (4) reporting of at least one clinical outcome measure including progression rates, intervention rates, mortality data, or complication rates. We excluded studies that: (1) focused exclusively on nontraumatic brain pathology; (2) included only pediatric populations without adult comparison; (3) were case reports, editorials, or technical notes without outcome data; or (4) had insufficient data on repeat CT timing protocols.

Two independent reviewers screened titles and abstracts for potential eligibility, followed by full-text review of potentially relevant articles. Disagreements were resolved by consensus or consultation with a third reviewer.

Data extraction and quality assessment

Data extraction was performed by two independent reviewers using a standardized form. Extracted information included study characteristics (study design, sample size, population demographics, follow-up duration), clinical parameters (initial GCS scores, injury types, timing of repeat scans, clinical outcomes), and statistical measures (progression rates, intervention rates, mortality data, complication rates).

Quality assessment was performed using the Newcastle–Ottawa Scale for observational studies and the AMSTAR-2 tool for systematic reviews.

Results

The initial search yielded 1247 records, of which 472 remained after removal of duplicates. After screening titles and abstracts, 158 articles were retrieved for full-text review. Ultimately, 26 studies met the inclusion criteria and were included in the systematic review (Fig. 1). Most were observational studies (21), three were narrative reviews, and two were systematic reviews and meta-analyses. The overall quality was high, with 16 high-quality and 5 moderate-quality observational studies. Of the two systematic reviews, one was of low quality and one moderate.

Study characteristics

The analyzed literature comprised 12 studies focusing on timing for serial head CT scans in adult populations with TBI (Table 1). Sample sizes ranged from 46 to 1594

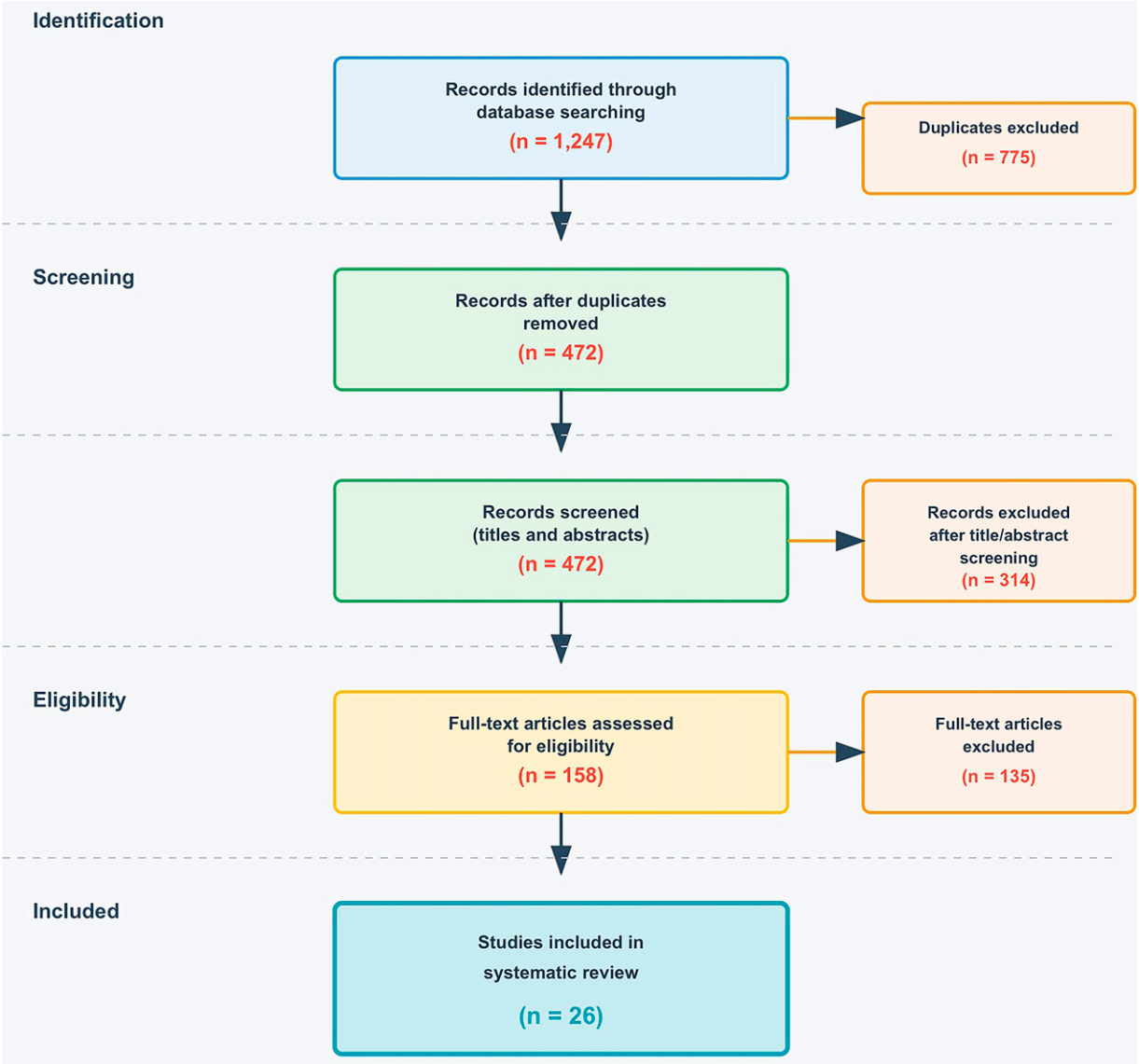

FIG. 1. PRISMA flow diagram. PRISMA, Preferred Reporting Items for Systematic reviews and Meta-Analyses.

patients. Patient demographics varied significantly across studies; mean/median age ranged from 33 to 82 years, with male predominance (60–87%) in most cohorts.[11,15] Several studies specifically examined patients with traumatic intracranial hemorrhage (tICH), while others focused on distinct populations, such as patients on anticoagulation therapy.[11,16] Injury severity also varied substantially, with some studies exclusively enrolling patients with mild TBI (GCS 14–15), while others included moderate-to-severe TBI (GCS 3–13) or the full spectrum of injury severity.

Timing protocols

Initial CT scans were typically performed within hours of injury, with time intervals ranging from 30 min to 6+ h post-trauma. Multiple studies demonstrated that the timing of imaging significantly influenced detection of hemorrhage progression. Oertel et al. reported that 48.6% of patients who underwent initial CT scanning within 2 h of injury showed progressive hemorrhagic injury on subsequent imaging, compared with lower rates when initial scanning occurred later.[22] Repeat CT scan protocols varied considerably across studies: Some mandated follow-up at fixed intervals of 6 h,[12] 24 h,[21] or both 24 and 72 h,[15] while others employed variable timing based on clinical judgment. Fletcher-Sandersjöö et al. provided compelling evidence that 33% of hematomas had stopped progressing within 3 h, 66% within 8 h, and 94% within 24 h of injury, suggesting the critical

Table 1. Summary of Studies Examining Delayed Traumatic Intracranial Hemorrhage and Progression

Study	*Study characteristics*	*Clinical parameters*	*Statistical measures*	*Quality assessment*
Verschoof et al. (2018)[11]	**Design:** Retrospective multicenter **Sample:** 905 patients with mTBI on anticoagulation **Demographics:** Median age 82 years, 47% male **Follow-up:** 3 months	**Initial GCS:** 13–15 **Injury types:** All types of traumatic ICH **Timing of repeat CT:** Within 24 h of first scan **Outcomes:** Death, neurosurgical intervention	**Progression rate:** 0.4% (4/905) with delayed ICH symptoms **Intervention rate:** 0.9% needed neurosurgery **Mortality:** 0.44% (4/905) **Complications:** 9 patients deteriorated due to ICH	High
Moskopp et al. (2024)[12]	**Design:** Retrospective single-center **Sample:** 213 patients with TBI **Demographics:** Mean age 67.6 years, M:F ratio 1.8:1 **Follow-up:** Hospital course	**Initial GCS:** 56% with GCS 13–15, 23% with GCS 3–8 **Injury types:** Contusions, SDH, EDH, tSAH **Timing of repeat CT:** 6 h after admission **Outcomes:** Surgery, mortality, GOS	**Progression rate:** 7.3% (9/123) early deterioration **Intervention rate:** 3/114 (2.6%) required surgery after CT 6 h **Mortality:** Not specifically reported **Complications:** 8.5% late deterioration	High
Fletcher-Sandersjöö et al. (2023)[15]	**Design:** Single-center observational cohort **Sample:** 643 patients with moderate-to-severe TBI **Demographics:** Median age 47 years, 74% male **Follow-up:** 12 months	**Initial GCS:** 4–13 (median 7) **Injury types:** Contusions, SDH, EDH, tSAH **Timing of repeat CT:** 24 and 72 h after injury **Outcomes:** GOS at 12 months	**Progression rate:** 61% showed hematoma expansion **Intervention rate:** Not specified **Mortality:** 18% at 12 months **Complications:** Not specifically reported	High
Alahmadi et al. (2010)[13]	**Design:** Retrospective review **Sample:** 98 patients with brain contusions **Demographics:** Age and gender not specified **Follow-up:** 6 months	**Initial GCS:** Various (not specified) **Injury types:** Cerebral contusions **Timing of repeat CT:** Within 24 h **Outcomes:** Need for neurosurgery, GOS	**Progression rate:** 45% (44/98) significant progression **Intervention rate:** 4% (4/98) required delayed evacuation **Mortality:** Not specifically reported **Complications:** Not specifically reported	High
Juratli et al. (2014)[14]	**Design:** Prospective observational **Sample:** 153 patients with TBI **Demographics:** Mean age 42 years, M:F ratio 2.7:1 **Follow-up:** 1 year	**Initial GCS:** Not specified **Injury types:** Contusions, various ICH **Timing of repeat CT:** Within 24 h **Outcomes:** mRS at discharge and 1 year	**Progression rate:** 42% (64/153) showed progression **Intervention rate:** 3/153 (2%) required surgical intervention **Mortality:** 2.6% (4/153) **Complications:** Thromboembolic events in 13%	High
Chauny et al. (2016)[16]	**Design:** Systematic review and meta-analysis **Sample:** 1594 patients across 7 studies **Demographics:** Varied across studies **Follow-up:** Varied	**Initial GCS:** 14–15 **Injury types:** Various ICH in anticoagulated patients **Timing of repeat CT:** Within 24 h **Outcomes:** Death, neurosurgical intervention	**Progression rate:** 0.6% (95% CI: 0–1.2%) **Intervention rate:** 0.13% (95% CI: 0.02–0.45%) **Mortality:** 0.13% (2/1594) **Complications:** Not specifically reported	Moderate
Jeng et al. (2008)[17]	**Design:** Prospective observational **Sample:** 81 patients with TBI **Demographics:** Mean age 33 years, M:F ratio 6.36:1 **Follow-up:** 6 months	**Initial GCS:** Median 12 (range 4–15) **Injury types:** Contusions and hematomas **Timing of repeat CT:** Within 24 h, 4 days, 7 days **Outcomes:** Death, disability	**Progression rate:** 16.4% (43/262) expanding hematomas **Intervention rate:** 86% (37/43) expanding hematomas required surgery **Mortality:** 51.3% (19/37) in operated group **Complications:** Not specifically reported	High
White et al. (2009)[18]	**Design:** Retrospective cohort **Sample:** 46 patients with traumatic intracerebral contusion **Demographics:** Mean age 38 years, 82% male **Follow-up:** Discharge and hospital course	**Initial GCS:** Mean 9 (41% severe, 9% moderate, 50% mild TBI) **Injury types:** Intracerebral contusions/hematomas **Timing of repeat CT:** Within 24 h **Outcomes:** GCS changes, mortality	**Progression rate:** 65% showed progression **Intervention rate:** Not specified **Mortality:** 20% (7/35) with expansion vs. 4% (1/25) without **Complications:** Not specifically reported	High
Kreitzer et al. (2014)[19]	**Design:** Retrospective cohort **Sample:** 323 patients with mild TBI and ICH **Demographics:** Mean age 42 years, 73% male **Follow-up:** Hospital course	**Initial GCS:** 14–15 **Injury types:** Various ICH (SAH 47%, SDH 41%, contusions 24%) **Timing of repeat CT:** Median 6 h after first scan **Outcomes:** Death, neurosurgical intervention	**Progression rate:** 42% showed changes on repeat CT **Intervention rate:** 0.9% (3/323) required neurosurgery **Mortality:** 1.2% (4/323) **Complications:** 8.7% (28/323) had return ED visits within 1 week	High

(*continued*)

Table 1. (Continued)

Study	*Study characteristics*	*Clinical parameters*	*Statistical measures*	*Quality assessment*
Yadav et al. (2006)[20]	**Design:** Prospective observational **Sample:** 262 patients with contusions/hematomas **Demographics:** Mean age 35 years, M:F ratio 2.05:1 **Follow-up:** Not specified	**Initial GCS:** Various (stratified in analysis) **Injury types:** Intracerebral contusions/hematomas **Timing of repeat CT:** Within 24 h **Outcomes:** Surgery, mortality	**Progression rate:** 16.4% (43/262) expanding hematomas **Intervention rate:** 86% (37/43) of expanding hematomas required surgery **Mortality:** 51.3% (19/37) in operated group **Complications:** Not specifically reported	High
Narayan et al. (2008)[21]	**Design:** Prospective multicenter observational **Sample:** 56 patients with TBI and tICH **Demographics:** Median age 42.5 years, 75% male **Follow-up:** 15 days	**Initial GCS:** 4–14 (median 8) **Injury types:** Traumatic intracerebral hemorrhage **Timing of repeat CT:** 24 and 72 h after injury **Outcomes:** Functional outcomes, mortality	**Progression rate:** 51% showed hematoma expansion **Intervention rate:** 8.9% (5/56) required surgery **Mortality:** 13% (8/60) **Complications:** Thromboembolic events in 13%	High
Oertel et al. (2002)[22]	**Design:** Prospective cohort **Sample:** 142 patients with TBI **Demographics:** Mean age 34 years, M:F ratio 4.3:1 **Follow-up:** 6 months	**Initial GCS:** Median 8 (range 3–15) **Injury types:** Various ICH (EDH, SDH, IPCH, SAH) **Timing of repeat CT:** Mean 6.9 h after first scan **Outcomes:** Need for surgery, ICP course, 6-month GOS	**Progression rate:** 42.3% overall; 48.6% if first CT <2 h postinjury **Intervention rate:** 24% required surgery due to progression **Mortality:** Not specifically reported **Complications:** Progressive brain shift and swelling in 23% with PHI	Moderate

Bold, studies highlights; CI, confidence interval; CT, computed tomography; EDH, epidural hematoma; GCS, Glasgow Coma Scale; GOS, Glasgow Outcome Scale; ICH, intracranial hemorrhage; ICP, intracranial pressure; IPCH, intraparenchymal contusion/hematoma; mRS, modified Rankin Scale; mTBI, mild traumatic brain injury; PHI, progressive hemorrhagic injury; SAH, subarachnoid hemorrhage; SDH, subdural hematoma; TBI, traumatic brain injury; tICH, traumatic intracerebral hemorrhage; tSAH, traumatic subarachnoid hemorrhage.

window for hemorrhage evolution predominantly occurs early postinjury.[15]

Outcomes data

Rates of hemorrhage progression varied dramatically across studies, ranging from 0.4% to 65%.[11,18] This wide variation likely reflects differences in study populations, imaging protocols, and progression definitions. Neurosurgical intervention rates among patients with progressive hemorrhage ranged from 0.13% in anticoagulated patients with mild TBI[16] to 86% in patients with expanding hematomas.[20] Mortality rates also varied substantially from 1.2% in mild TBI[19] to over 50% in operated patients with expanding hematomas.[20] Several studies demonstrated that hemorrhage progression correlated with worse outcomes, including increased intracranial pressure,[22] longer intensive care unit stays,[12] higher mortality,[18] and poorer functional outcomes. Fletcher-Sandersjöö et al. identified a dose–response relationship between hematoma expansion and functional outcomes, with every 1 mL increase in hematoma volume associated with a 6% increased risk of worse Glasgow Outcome Scale (GOS) scores at 12 months.[15]

Factors predicting hemorrhagic progression

The main characteristics of the studies focusing on predictive factors for progression of hemorrhagic lesions in TBI are listed in Table 2.

The frequency of hemorrhagic progression of cerebral contusions varies considerably across studies, ranging from 1.5% to 65%.[33] This variance is partially explained by differences in study methodology, including varying definitions of progression, timing of follow-up imaging, and patient selection criteria. Most studies define progression as a relative increase in contusion volume (typically ≥30%), although some include absolute volume increases (≥10 mL) or radiological findings correlated with clinical deterioration.

Numerous factors have been identified as predictors of contusion progression (Table 3). Radiological factors appear to be most consistently associated with progression, particularly the presence of concomitant intracranial lesions such as subarachnoid hemorrhage (odds ratio [OR] 3.28), subdural hemorrhage (OR 4.35), and epidural hemorrhage (OR 1.47).[23] Initial contusion characteristics including location, multiplicity, and volume also play important roles, with frontal contusions, multiple contusions, and bilateral contusions conferring increased risk.[28] Interestingly, there are conflicting findings regarding initial contusion volume, with some researchers reporting larger volumes as predictive,[24] while others found smaller initial volumes (<5 mL) associated with greater progression.[35] Clinical factors identified across multiple studies include older age, lower initial GCS scores, coagulopathy, and antiplatelet therapy.[29,31] Notably, patients on antiplatelet medications appear to have

Table 2. Main Characteristics of Key Studies for Hemorrhagic Progression of Cerebral Contusions

Study	*Design*	*Sample size*	*Population*	*Definition of progression*	*Progression rate*	*Key findings*	*Quality assessment*
Peng et al. (2024)[23]	Systematic review and meta-analysis	8 studies, 2543 patients	Patients with TBI and contusions	Varied across studies	16.4–51.0%	SAH (OR 3.28), SDH (OR 4.35), EDH (OR 1.47), and contrast extravasation (OR 11.81) were significant predictors of contusion progression	Low
Iaccarino et al. (2014)[24]	Retrospective, multicenter	352 patients	Any severity TBI with contusions	≥30% increase in volume	42.3%	Independent predictors of unfavorable outcome: increased age, lower GCS, clinical deterioration, and onset/increase of midline shift. Clinical deterioration was associated with midline shift and basal cistern compression rather than hematoma growth	High
Jirlow et al. (2024)[25]	Narrative review	N/A	Patients with TBI and contusions	Varied across studies	40–50%	Emphasized the traumatic penumbra concept. Initial volume, site of injury, mechanism, history of hypertension, current smoking, coagulopathy, and presence of coexisting SAH/SDH predict progression	N/A
Kurland et al. (2012)[26]	Narrative review	N/A	Patients with TBI and contusions	Varied across studies	Up to 75%	Proposed novel mechanism for contusion progression involving microvascular failure; mechanosensitive transcription factors (Sp1 and NF-κB) cause upregulation of Sur1 channels, leading to endothelial cell death and capillary fragmentation	N/A
Shafiei et al. (2023)[27]	Retrospective cross-sectional	218 patients	Mild TBI with contusions	>30% increase in size	50.9%	Presence of SAH, SDH, and EDH were predictors of radiological progression. Patients with SDH and EDH were more likely to undergo surgery	High
Rehman et al. (2019)[28]	Prospective cohort	246 patients	Any severity TBI with contusions	>30% increase in initial volume	44.7%	Independent predictors: multiplicity, bilateral lesions, initial volume >20 mL, frontal location, concomitant EDH (OR 3.90), SDH (OR 2.91), and SAH (OR 2.27)	High
Uccella et al. (2018)[29]	Retrospective	1608 patients	Mild TBI (GCS 15)	Any hemorrhage on CT	8.8%	Patients on antiplatelet therapy (13.8%) were at significantly higher risk of intracranial hemorrhage than the general population (6.4%). Anticoagulated patients (8.1%) showed no increased risk	Moderate
Allison et al. (2017)[30]	Retrospective	286 patients	Moderate and severe TBI	≥30% and ≥10 mL increase	21%	Developed HPC Score (SAH = 2 points, SDH = 1 point, skull fracture = 1 point). Scores 0–2 had 4.0% risk of progression; scores 3–4 had 34.6% risk. Early red blood cell transfusion reduced risk	High
Seddighi et al. (2013)[31]	Prospective cross-sectional	203 patients	Mild TBI	GCS deterioration (decrease ≥2 points)	1.5%	Coagulopathy, anticoagulant drug use, GCS 13–14, increased age, midline shift, cerebral contusion, diffuse cerebral edema, and SDH predicted deterioration	Moderate
Raymond et al. (2023)[32]	Retrospective	2137 patients	Isolated head injury on ACAP therapy	Positive CT after initial negative CT	0.023% (dtICH) 8.2% (initial)	Dual therapy or Coumadin therapy made up the majority of traumatic intracranial hemorrhage. Male gender, cirrhosis, and chemotherapy were associated with delayed hemorrhage	Moderate
Adatia et al. (2021)[33]	Narrative review	N/A	Patients with TBI and contusions	Varied across studies	16–75%	Summarized clinical and radiological predictors: older age, male sex, lower GCS, hypertension, smoking, coagulopathy, initial contusion size, location, coexisting lesions, cisternal compression, and skull fracture	N/A
Narayan et al. (2008)[21]	Prospective	56 patients	TBI with GCS 4–14	Any increase in volume	51% at 24 h, 53% at 72 h	Initial contusion size correlated with progression. No association between change in lesion volume and functional outcomes at discharge	High
Oertel et al. (2002)[22]	Retrospective	Multiple lesion types	Patients with TBI and multiple lesion types	Varied across studies	51% for contusions	Contusions had the highest rate of progression compared with other traumatic lesions. Progressive hemorrhage was associated with worse outcome and mortality	Moderate
Chang et al. (2006)[34]	Retrospective	113 patients	Any severity TBI with contusions	Any increase in volume	35–38%	Presence of SAH, SDH, and larger initial contusion size predicted progression. Growth >5 cm^3 independently associated with need for surgical intervention	High
Cepeda et al. (2015)[35]	Retrospective	782 patients	Moderate and severe TBI	≥33% increase or new lesion	64%	Risk factors: initial volume <5 mL, cisternal compression, decompressive craniectomy, older age, falls, multiple contusions, and hypoxia	High
Beaumont and Gennarelli (2006)[36]	Retrospective	21 patients	Any severity TBI with contusions	>5% increase	47.6%	Ratio of edema to no edema was correlated with progression. Contusions with edema on initial scan were less likely to progress	Moderate

ACAP, anticoagulant and antiplatelet; CT, computed tomography; EDH, epidural hematoma; dtICH, delayed traumatic intracranial hemorrhage; GCS, Glasgow Coma Scale; HPC, hemorrhagic progression of contusion; INR, International Normalized Ratio; OR, odds ratio; NF-κB, nuclear factor kappa B; SAH, subarachnoid hemorrhage; SDH, subdural hematoma; Sp1, specificity protein 1; Sur1, sulfonylurea receptor 1; N/A, not applicable; TBI, traumatic brain injury.

significantly higher risk of progression compared with those on anticoagulants.[32] Comorbidities such as hypertension, cirrhosis, and ongoing chemotherapy treatment have also been implicated.[27] Advanced imaging findings, including contrast extravasation on CT angiography (OR 11.81) and the "multihematoma fuzzy sign," represent promising newer predictors.[23] Several studies have developed scoring systems incorporating these factors to predict progression risk, such as the Hemorrhagic Progression of Contusions (HPC) Score proposed by Allison et al., which assigns points for subarachnoid hemorrhage, subdural hematoma, and skull fracture, differentiating between low-risk (4.0% progression) and high-risk (34.6% progression) patients.[30]

Subgroup analysis

Impact of TBI severity on hemorrhage progression and outcomes. Subgroup analysis comparing outcomes based on TBI severity revealed significant differences in hemorrhage progression, intervention requirements, and mortality (see Table 4). Studies focused exclusively on mild TBI (GCS 13–15) demonstrated significantly lower rates of hemorrhage progression (0.4–42%), neurosurgical intervention (0.13–0.9%), and mortality (0.13–1.2%) compared with moderate–severe TBI cohorts (GCS 3–12), which exhibited progression rates of 42.3–61%, intervention rates of 8.9–24%, and mortality rates of 13–18%.[11,15,19] This severity-based stratification maintained consistency even when controlling for timing of initial imaging. Notably, studies examining mixed TBI severity populations showed the widest variation in outcomes,[18,20] highlighting the importance of severity-based patient stratification when formulating clinical recommendations. An interesting finding across severity subgroups was that earlier initial CT scanning (<2–3 h postinjury) identified significantly higher rates of progression on subsequent imaging, supporting the theory proposed by Oertel et al. that early imaging captures the hemorrhage in an actively evolving state.[22] These findings suggest that routine repeat imaging may be more valuable in moderate–severe TBI, while selective repeat imaging strategies based on clinical factors may be more appropriate for patients with mild TBI who have normal neurological examinations.

Age-related considerations. Advanced age consistently emerges as a significant independent predictor of HPC across multiple studies, though the specific mechanisms and thresholds require further examination (Table 5). Several studies have identified increased age as an independent risk factor for contusion progression. Iaccarino et al. identified older age as one of the four most reliable predictors of unfavorable outcome in multivariate analysis, alongside lower GCS score, clinical deterioration, and midline shift.[24] Similarly, Cepeda et al. developed a nomogram for predicting TBI progression that included older age among its significant independent variables.[35] In their study, Seddighi et al. found that patients who experienced clinical deterioration had a significantly higher mean age (40.6 years) compared with those who remained stable (33.8 years, $p = 0.01$).[31]

Anticoagulation status. The relationship between anticoagulation/antiplatelet therapy and contusion progression presents a more complex picture with some conflicting evidence, particularly between different types of agents (Table 5).

Regarding antiplatelet agents, Uccella et al. found that patients on antiplatelet therapy had a significantly higher risk of intracranial hemorrhage compared with nonanticoagulated patients. The probability of higher intracranial bleeding for antiplatelet drugs was estimated at 100%, with a relative risk of 158.8 – 296.2% (median 216.3%).[29] There appears to be a distinction between generations of antiplatelet drugs: patients on second-generation antiplatelet drugs showed a higher rate of hemorrhage (18%) compared with first-generation drugs (12.8%), though this did not reach statistical significance ($p < 0.1$).[29] Dual antiplatelet therapy carries particularly high risk: Patients on dual therapy showed higher rates of hemorrhage (25.8%) compared with single antiplatelet therapy (13.4%).[29]

Surprisingly, for anticoagulants, Uccella et al. found no significant difference in hemorrhage rates between anticoagulated patients and the general population ($p < 0.97$), with comparable median rates (9.2% vs. 8.9%).[29] No significant difference was observed between traditional and newer anticoagulants in terms of hemorrhage risk (9.3% vs. 9.2%, $p > 0.87$).[29] Raymond et al. reported that "dual therapy or Coumadin therapy made up the majority of tICH [traumatic intracranial hemorrhage]," suggesting particular concern with these regimens.[32] Seddighi et al. found that "presence of coagulopathy and anticoagulant drug use predicted further deterioration," with statistical significance (p values 0.04 and <0.001, respectively).[31] White et al. showed that coagulopathy with International Normalized Ratio >1.2 was associated with contusion progression.[18] In contrast, Kurland et al. noted that "the relationship between HPC and measurable coagulopathy, while clinically useful, is not one of simple cause and effect," pointing out that many patients with normal coagulation parameters still develop HPC.[26]

Discussion

This systematic review reveals considerable heterogeneity in both the methodology and findings regarding repeat CT scanning in TBI. The reported rates of hemorrhagic progression vary dramatically from 0.4% to 65% across studies,[11,18] reflecting differences in study populations,

Table 3. Factors Associated with Hemorrhagic Progression of Cerebral Contusions

Category	*Factors*	*Strength of association*	*Key studies*
Radiological factors	Subarachnoid hemorrhage	OR 2.27–6.33	Peng et al. (2024),[23] Allison et al. (2017),[30] Rehman et al. (2019)[28]
	Subdural hemorrhage	OR 2.91–4.35	Peng et al. (2024),[23] Allison et al. (2017),[30] Rehman et al. (2019)[28]
	Epidural hemorrhage	OR 1.47–3.90	Peng et al. (2024),[23] Rehman et al. (2019)[28]
	Contrast extravasation	OR 11.81	Peng et al. (2024)[23]
	Midline shift or its progression	Significant predictor	Iaccarino et al. (2014),[24] Seddighi et al. (2013)[31]
	Cisternal compression	Significant predictor	Iaccarino et al. (2014),[24] Cepeda et al. (2015)[35]
	Multiple contusions	~3× higher risk	Rehman et al. (2019)[28]
	Bilateral contusions	~3× higher risk	Rehman et al. (2019)[28]
	Frontal contusion location	1.5× higher risk	Rehman et al. (2019)[28]
	Multihematoma fuzzy sign	Significant predictor	Referenced in Adatia et al. (2021)[33]
Clinical factors	Lower initial GCS (13–14)	Significant predictor	White et al. (2009),[18] Seddighi et al. (2013)[31]
	Older age	Independent predictor	Iaccarino et al. (2014),[24] Cepeda et al. (2015)[35]
	Coagulopathy/INR >1.2	Significant predictor	White et al. (2009),[18] Seddighi et al. (2013)[31]
	Antiplatelet therapy	Higher risk than anticoagulants	Uccella et al. (2018),[29] Raymond et al. (2023)[32]
	History of hypertension	4.5× higher risk	Referenced in Adatia et al. (2021)[33]
	Current smoking	6× higher risk	Referenced in Adatia et al. (2021)[33]
	Fall as injury mechanism	Significant predictor	Cepeda et al. (2015)[35]
	Hypoxia	Significant predictor	Cepeda et al. (2015)[35]
	Cirrhosis	Significant predictor	Raymond et al. (2023)[32]
	Chemotherapy	Significant predictor	Raymond et al. (2023)[32]
Procedural factors	Decompressive craniectomy	3× higher risk	Cepeda et al. (2015)[35]
	Short interval from injury to initial CT	Significant predictor	Referenced in Adatia et al. (2021)[33]

CT, computed tomography; GCS, Glasgow Coma Scale; INR, International Normalized Ratio; OR, odds ratio.

imaging protocols, and definitions of progression. This variability complicates the development of standardized guidelines but also highlights the need for nuanced, patient-specific approaches to repeat imaging after TBI.

A key finding from this synthesis is the significant impact of TBI severity on hemorrhage progression and subsequent outcomes. Studies focused exclusively on mild TBI (GCS 13–15) consistently demonstrated lower rates of progression (0.4–42%), neurosurgical intervention (0.13–0.9%), and mortality (0.13–1.2%) compared with cohorts with moderate–severe TBI.[11,16,19] This contrasts with progression rates of 42.3–61%, intervention rates of 8.9–24%, and mortality rates of 13–18% in moderate–severe TBI.[15,21,22] Such substantial differences suggest that severity-specific approaches to repeat imaging may be justified.

The timing of initial CT scanning emerges as another critical factor influencing the detection of hemorrhage progression. Multiple studies demonstrated that initial scans performed within 2–3 h of injury identified significantly higher rates of progression on subsequent imaging.[22] This finding aligns with the concept of capturing hemorrhages in an actively evolving state, as proposed by Fletcher-Sandersjöö et al., who noted that 33% of hematomas had stopped progressing within 3 h, 66% within 8 h, and 94% within 24 h postinjury.[15] This temporal pattern suggests a critical window for hemorrhage evolution that is predominantly early postinjury.

Several predictors of contusion progression have been consistently identified across studies. Radiological factors appear most strongly associated with progression, particularly the presence of concomitant intracranial lesions such as subarachnoid hemorrhage (OR 2.27–6.33), subdural hemorrhage (OR 2.91–4.35), and epidural hemorrhage (OR 1.47–3.90).[23,28,30] Other significant predictors include contrast extravasation on CT angiography (OR 11.81), midline shift, cisternal compression, multiple contusions, and bilateral contusions.[23,24,28]

Table 4. Hemorrhage Progression by Traumatic Brain Injury Severity

TBI severity	*Studies*	*Progression rate*	*Intervention rate*	*Mortality rate*
Mild TBI (GCS 13–15)	Verschoof et al. (2018)[11] Kreitzer et al. (2014)[19] Chauny et al. (2016)[16]	0.4–42%	0.13–0.9%	0.13–1.2%
Moderate–severe TBI (GCS 3–12)	Fletcher-Sandersjöö et al. (2023)[15] Narayan et al. (2008)[21] Oertel et al. (2002)[22]	42.3–61%	8.9–24%	13–18%
Mixed severity	Yadav et al. (2006)[20] Juratli et al. (2014) White et al. (2009)[18] Alahmadi et al. (2010) Jeng et al. (2008)	16.4–65%	2–86%	2.6–51.3%

GCS, Glasgow Coma Scale; TBI, traumatic brain injury.

Table 5. Comparative Analysis of Age and Anticoagulation Status in Contusion Progression

Factor	*Subgroup*	*Risk of progression*	*Key studies*	*Clinical implications*
Age	Older age (generally >60–65)	Independently associated with progression	Iaccarino et al. (2014),[24] Cepeda et al. (2015),[35] Seddighi et al. (2013)[31]	• More vigilant monitoring • Consider lower threshold for admission • Consider earlier follow-up imaging
Antiplatelet therapy	First generation (ASA)	12.8% hemorrhage rate	Uccella et al. (2018)[29]	• Monitor for clinical deterioration • Consider follow-up imaging
	Second generation (e.g., clopidogrel)	18% hemorrhage rate	Uccella et al. (2018)[29]	• Higher risk warrants close observation • Consider reversal agents if deterioration
	Dual antiplatelet therapy	25.8% hemorrhage rate	Uccella et al. (2018)[29]	• Particularly high risk • Lower threshold for ICU admission • Consider early follow-up imaging
Anticoagulants	Traditional (e.g., warfarin)	9.0% hemorrhage rate No significant difference from general population	Uccella et al. (2018)[29]	• Check INR/coagulation parameters • Consider reversal if elevated
	Newer agents (e.g., DOACs)	8.3% hemorrhage rate No significant difference from general population	Uccella et al. (2018)[29]	• Limited options for reversal • Monitor clinically
Combined risk factors	Older age + antiplatelet therapy	Significantly elevated risk	Multiple studies	• Consider as high-risk group • Lower threshold for intervention
	Anticoagulation + cirrhosis	Significantly higher risk ($p = 0.047$)	Raymond et al. (2023)[32]	• Close monitoring • Consider early intervention
	Anticoagulation + chemotherapy	Significantly higher risk ($p = 0.011$)	Raymond et al. (2023)[32]	• Higher risk of delayed hemorrhage • Consider extended observation

ASA, acetylsalicylic acid; DOAC, direct oral anticoagulant; ICU, intensive care unit; INR, International Normalized Ratio.

Clinical factors also contribute significantly to progression risk. Advanced age consistently emerges as an independent predictor,[24,35] likely due to age-related cerebrovascular changes, including reduced vessel elasticity and increased fragility.[25] Anticoagulation and antiplatelet therapy present a more complex picture. Patients on antiplatelet therapy, particularly dual therapy or second-generation agents, demonstrated significantly higher risks of intracranial hemorrhage compared with nonanticoagulated patients.[29] Interestingly, some studies found no significant difference in hemorrhage rates between patients on anticoagulants and the general population,[29] though others identified coagulopathy as a significant risk factor.[18,31]

Clinical implications

The findings of this review have several important clinical implications for the management of patients with TBI. First, the evidence supports a stratified approach to repeat CT scanning based on TBI severity. For patients with moderate–severe TBI (GCS 3–12), the high rates of progression (42.3–61%) and subsequent intervention (8.9–24%) suggest that routine repeat imaging is warranted, regardless of clinical status. In contrast, for patients with mild TBI (GCS 13–15) who maintain normal neurological examinations, a more selective approach may be appropriate, as intervention rates are consistently below 1% in this population.[8,16,19]

Timing considerations should also inform clinical protocols. The evidence indicates that most hemorrhagic progression occurs within the first 24 h postinjury, with Fletcher-Sandersjöö et al. demonstrating that 94% of hematomas had ceased progressing by this time point.[15] This suggests that if routine repeat imaging is performed, it should be scheduled within this critical window. The finding that initial scans performed within 2–3 h postinjury identified higher rates of progression on follow-up imaging[22] further suggests that patients with very early initial scans might benefit from a scheduled repeat scan, even in the absence of clinical deterioration.

The identified risk factors for progression provide a framework for risk stratification in clinical decision-making. Patients with multiple risk factors, such as concomitant intracranial lesions, older age, and coagulopathy, may benefit from more vigilant monitoring and lower thresholds for repeat imaging. Several studies have attempted to formalize this approach through predictive scoring systems, such as the HPC Score developed by Allison et al., which demonstrated the ability to differentiate between low-risk (4.0% progression) and high-risk (34.6% progression) patients based on the presence of subarachnoid hemorrhage, subdural hematoma, and skull fracture.[30]

The differential impact of anticoagulation status on progression risk has important management implications. Patients on antiplatelet therapy, particularly those on dual therapy, warrant careful observation and consideration of repeat imaging. However, for patients on anticoagulants alone, routine repeat imaging may be less justified based solely on anticoagulation status. Raymond et al. highlighted the importance of considering combined risk factors, noting that patients who have received chemotherapy and are on anticoagulation or antiplatelet medication are at higher risk of hematoma progression and should be closely monitored with repeat CT scans.[32]

Finally, the potential risks associated with radiation exposure from repeat CT scans must be balanced against

the diagnostic benefits. Cao et al. identified an inordinately increased cancer risk associated with CT scans in adults, with cancer risks positively correlated with radiation dose and the number of CT sites scanned.[7] This highlights the importance of judicious use of repeat imaging, particularly in younger patients and those at low risk for progression.

Proposed algorithm

Based on the data that emerged from this systematic review, we propose an algorithm to optimize the timing of CT in patients with TBI (Fig. 2). This is supported by the following key evidence.

Temporal patterns of hemorrhagic progression demonstrate that 33% of hematomas cease progression within 3 h postinjury, 66% within 8 h, and 94% within 24 h, establishing critical windows for surveillance imaging.[15] Severity-stratified analysis reveals marked differences in progression rates between mild TBI (0.4–42%) and moderate–severe TBI (42.3–61%), with corresponding disparities in intervention requirements (0.13–0.9% vs. 8.9–24%, respectively), thus justifying differential imaging protocols. Notably, when the initial scanning is performed within 2–3 h postinjury, it identifies significantly higher rates of progression on subsequent imaging, suggesting that very early initial scans warrant scheduled follow-up regardless of clinical status.[22] Multivariate analyses consistently identify radiological predictors of progression, including concomitant subarachnoid hemorrhage (OR 3.28), subdural hemorrhage (OR 4.35), and epidural hemorrhage (OR 1.47).[23] Clinical risk factors with substantial evidential support include advanced age, antiplatelet therapy, particularly dual therapy, and coagulopathy.[29,31] The algorithm's risk-stratified approach reconciles the imperative for timely detection of clinically significant progression with concerns regarding radiation exposure, striking an evidence-based balance that optimizes resource utilization while minimizing unnecessary imaging in low-risk populations where repeat scanning yields minimal clinical benefit.

The proposed algorithm's primary function is not to identify patients who would benefit from a repeated CT scan, but rather to be integrated into validated protocols, such as the Brain Injuries Guidelines,[37] as a guide for determining the optimal timing interval when one or more follow-up CT scans are appropriate.

Research gaps and limitations

Despite the substantial body of literature on this topic, several important research gaps remain. First, there is a lack of standardization in the definition of "progression" across studies, with thresholds ranging from any increase in volume to specific percentage increases (typically ≥30%) or absolute volume changes (≥10 mL). This inconsistency complicates cross-study comparisons and meta-analyses.

Second, conflicting findings exist regarding certain risk factors. For example, some researchers identified larger initial contusion volumes as predictive of progression,[24] while others found smaller initial volumes (<5 mL) associated with greater progression.[35] Such discrepancies highlight the complex pathophysiology of contusion evolution and the need for more nuanced understanding of these mechanisms.

Third, although multiple predictive scoring systems have been proposed,[8,30] most lack external validation in diverse populations. The generalizability of these tools across different clinical settings and patient demographics remains unclear.

Fourth, the optimal timing for repeat imaging remains incompletely defined. While studies have provided valuable insights into the temporal pattern of hemorrhage evolution, the specific timing that maximizes diagnostic yield while minimizing unnecessary scans has not been definitively established.

Finally, most studies have focused on short-term outcomes, such as radiological progression, need for neurosurgical intervention, and in-hospital mortality. The relationship between hemorrhage progression, repeat imaging strategies, and long-term functional outcomes remains underexplored.

Due to the extreme heterogeneity of the data collected, a meta-analysis could not be performed.

Future directions

Future research should address these gaps through several approaches. Large, prospective multicenter studies with standardized definitions and protocols are needed to better characterize the natural history of traumatic intracranial lesions and establish evidence-based guidelines for repeat imaging. These studies should stratify patients by TBI severity, age, and other key risk factors to enable more personalized approaches to management.

The development and external validation of comprehensive risk prediction tools represent another important direction. Such tools should incorporate both radiological and clinical predictors and be validated across diverse populations to ensure generalizability. The BRAIN-CT Score proposed by Taddei et al. represents a promising step in this direction, although further validation is needed.[8]

Investigation of novel biomarkers as adjuncts to imaging could potentially enhance risk stratification and reduce reliance on repeat CT scans. Blood-based biomarkers such as S100B, glial fibrillary acidic protein, and ubiquitin C-terminal hydrolase-L1 (UCH-L1) have shown promise in predicting the absence of intracranial injury[9] and could be incorporated into clinical algorithms to identify patients who might safely avoid repeat imaging.

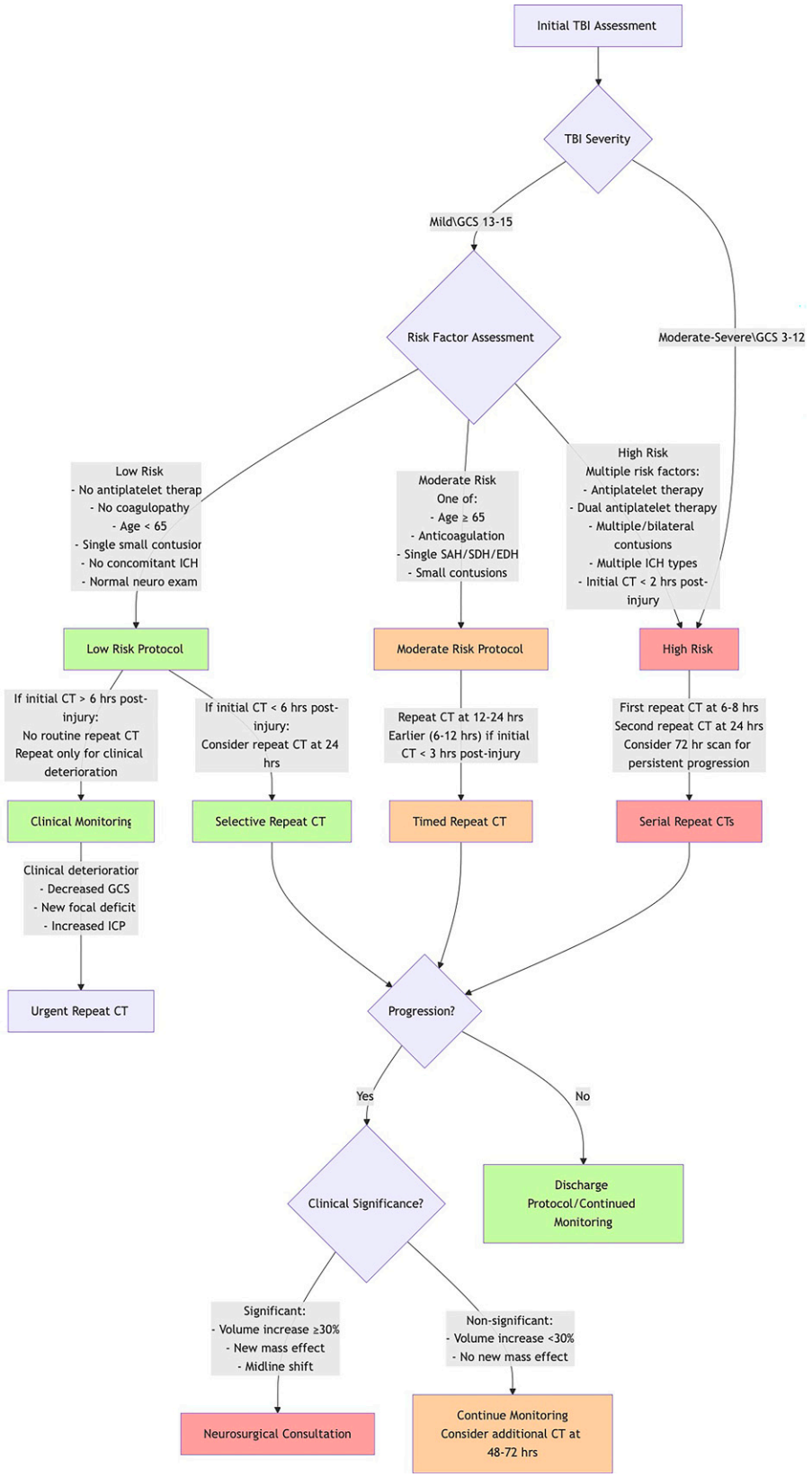

FIG. 2. Algorithm for optimal CT timing in TBI patients. CT, computed tomography; TBI, traumatic brain injury.

Conclusion

This systematic review identifies significant heterogeneity in approaches to repeat CT scanning after TBI and provides evidence supporting a stratified approach based on injury severity and patient-specific risk factors. While routine repeat imaging may be warranted in moderate–severe TBI, a more selective approach appears appropriate for mild TBI with normal neurological examinations. Future research should focus on standardizing definitions, validating risk prediction tools, and exploring novel biomarkers to enhance the precision and cost-effectiveness of post-TBI imaging protocols.

Transparency, Rigor, and Reproducibility Summary

This study was not formally registered because it represents a systematic review of existing literature rather than a primary data collection study. The analysis plan was not formally preregistered, but the team member with primary responsibility for the analysis (lead author) certifies that the analysis plan was prespecified according to PRISMA guidelines. A comprehensive search strategy was planned to identify all relevant studies from inception to March 2025, with predetermined inclusion and exclusion criteria established prior to study selection.

The initial search yielded 1247 records, of which 472 remained after duplicate removal. Following title and abstract screening, 158 articles underwent full-text review, with 26 studies ultimately meeting inclusion criteria and being analyzed. Two independent reviewers performed all screening and data extraction processes, with disagreements resolved by consensus or consultation with a third reviewer. Data collection was performed by investigators blinded to study hypotheses during the initial screening phase.

Data were acquired between January and March 2025 using systematic database searches of PubMed/MEDLINE, Cochrane Library, EMBASE, and clinical trials databases. Data extraction was performed using standardized forms, with quality assessment conducted using the Newcastle–Ottawa Scale for observational studies and the AMSTAR-2 tool for systematic reviews. All datasets were analyzed simultaneously following completion of data extraction. No analysis failures occurred during the study period.

All search strategies, databases, and analytical approaches used are widely available and reproducible using standard systematic review methodology. The inclusion criteria represent established standards in the field of TBI research, and outcome measures analyzed (hemorrhagic progression rates, intervention rates, mortality) are recognized clinical endpoints. Statistical analysis was performed by investigators with specific training in systematic review methodology and meta-analysis techniques.

Due to extreme heterogeneity in study populations, imaging protocols, and outcome definitions across included studies, correction for multiple comparisons was not applicable, as meta-analysis could not be performed. Both original study findings and synthesized interpretations have been reported transparently. No replication or external validation studies have been performed or are planned at this time, as this represents a comprehensive synthesis of available evidence.

Data from this systematic review are not available in a public archive because they consist entirely of previously published literature that is publicly accessible through the original publications. The complete search strategy, inclusion/exclusion criteria, and data extraction forms will be made available by emailing the corresponding author. There is no analytic code associated with this study beyond standard systematic review methodology as described in the article. The authors agree to provide the full content of the article on request by contacting the corresponding author.

Authors' Contributions

All authors contributed to the study conception and design. Material preparation, data collection, and analysis were performed by G.T., R.B., and S.C. The first draft of the article was written by G.T., and all authors commented on previous versions of the article. All authors read and approved the final article.

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