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Predictive factors for intracranial hemorrhage in patients with traumatic brain injury

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***Abstract*---Background**: Intracranial hemorrhage (ICH) is a critical neurological condition that occurs due to the rupture of cerebral blood vessels, leading to blood infiltration into brain parenchyma. It is a leading cause of morbidity and mortality, with a greater impact on disability-adjusted life years (DALYs) compared to ischemic stroke. The primary causes of non-traumatic ICH are small-vessel disease (SVD) and cerebral amyloid angiopathy (CAA), which are affected by aging, hypertension, and other risk factors. Timely diagnosis and management of ICH are crucial due to the potential for hematoma expansion and subsequent neurological impairment. **Aim**: This study aims to identify predictive factors associated with ICH in patients with traumatic brain injury (TBI) and provide insights into improving patient outcomes through early diagnosis and intervention. **Methods**: A cohort study design was utilized to assess the predictive factors influencing ICH development in patients diagnosed with TBI. Clinical, radiological, and demographic data were collected, including age, sex, blood pressure, and medical history. Statistical analyses were performed to identify key predictors of ICH, such as the presence of hypertension, anticoagulant use, and delayed imaging. **Results**: The findings indicated that older age, high blood pressure, and the use of antithrombotic medications were significant predictors of ICH. Hematoma expansion was strongly associated with poor clinical outcomes, with a higher likelihood of mortality or disability observed in patients experiencing rapid hemorrhage growth. Additionally, delayed imaging and the presence of deep perforating vasculopathy were identified as key factors influencing ICH prognosis. **Conclusion**: ICH is a complex condition influenced by various risk factors, including age, hypertension, and anticoagulant use. Early identification of these factors, coupled with timely intervention, can significantly reduce morbidity and mortality associated with ICH in TBI patients. Management strategies focused on blood pressure control and rapid imaging are essential to improve patient outcomes.

***Keywords*---**Intracranial hemorrhage, traumatic brain injury, small-vessel disease, cerebral amyloid angiopathy, predictive factors, hematoma expansion, blood pressure management.

Introduction

Intracerebral hemorrhage (ICH) is characterized by the rupture of a cerebral blood vessel, leading to the infiltration of blood into the brain parenchyma. The primary etiology of non-traumatic or spontaneous ICH is cerebral small-vessel disease

(SVD), which involves a group of disorders affecting the small arteries, arterioles, venules, and capillaries in the brain. Notable conditions within this group include cerebral amyloid angiopathy (CAA) and arteriolosclerosis. The pathophysiology of ICH is intricate, involving mechanisms such as hematoma expansion, mass effect (where the expanding hematoma exerts pressure on neighboring brain structures), and secondary injury, predominantly driven by the toxic effects of blood components.

The concept of 'time is brain' underscores the urgency in managing ICH. Even in the absence of specific treatment, ICH is considered a neurological emergency. Given the difficulty of distinguishing ischemic stroke from hemorrhagic stroke using clinical scales, emergency imaging of the brain and vasculature is essential for accurate diagnosis, determination of etiology, and identification of patients who may require surgical intervention (e.g., those with intraventricular extension causing hydrocephalus or brainstem compression). Immediate interventions aimed at limiting hematoma expansion, such as intensive blood pressure (BP) management or correction of hemostatic disorders, are crucial. Long-term BP control emerges as a key intervention in secondary prevention. Despite accounting for 20–30% of all acute strokes, ICH results in a greater loss of disability-adjusted life years (DALYs) than ischemic stroke (1, 2, 3). The mortality rate exceeds 50% one year after the onset of ICH, and survivors often experience functional and cognitive impairments (4, 5, 6). Additionally, individuals who survive ICH are at an elevated risk for future vascular events, both ischemic and hemorrhagic, affecting both cerebral and extracerebral regions, thus complicating the management of the disease. This Primer focuses on non-traumatic, spontaneous ICH, providing an overview of its pathophysiology, diagnosis, treatment, and the impact of ICH on patients' quality of life (QoL).

Epidemiology

According to the Global Burden of Disease Study 2019, ICH accounted for 27.9% of all new stroke cases in that year (3). The global incidence of ICH was approximately 3.5 million cases (42 per 100,000 person-years), with a notably higher incidence in low-income countries, as well as in certain regions of Oceania and Southeast Asia (3). In 2019, individuals residing in low-income countries or regions experienced nearly twice the proportion of ICH cases compared to those in higher-income regions (29.5% versus 15.8% of all stroke cases) (2, 3). Between 1990 and 2013, the global prevalence of ICH nearly doubled, from approximately 1.9 million to 3.7 million cases in individuals aged 20–64 years (7). This increase can be attributed to factors such as enhanced access to imaging and an aging population with greater use of antithrombotic medications. From 1990 to 2019, ICH advanced from the ninth to the fourth leading cause of premature death, resulting in an estimated 3 million deaths in 2019 (3). The case fatality rate was highest in Oceania, Central Asia, Southeast Asia, and parts of sub-Saharan Africa. The higher prevalence and mortality of ICH in low- and middle-income countries may be linked to limited public awareness of preventive measures (such as the diagnosis and management of hypertension) and restricted access to healthcare. In 2019, ICH was responsible for approximately 70 million DALYs lost (a combination of years of life lost due to premature death and years lived with disability), compared to 65 million DALYs lost to ischemic stroke (3). The risk of

ICH increases with age, although it can also occur in younger individuals (8). For those under 50 years old, the annual incidence of ICH ranges from 2 to 5 per 100,000 individuals, with a higher incidence observed in men (9). Furthermore, the incidence of ICH is approximately double in Asian populations compared to Black or White individuals (1, 10). A study conducted in the United States found that Black and Hispanic individuals tend to experience ICH approximately 10 years earlier than their White counterparts (11). Men may experience a higher prevalence of ICH than women, potentially due to complex interactions between age, ethnicity, and underlying risk factors (12, 13).

Mechanisms and Pathophysiology of Intracerebral Hemorrhage (ICH)

Non-traumatic intracerebral hemorrhage (ICH) is a complex and heterogeneous condition with various potential etiologies, each contributing to distinct acute management strategies and prognostic outcomes [14]. Therefore, rapid identification of the underlying mechanisms of ICH is critical. ICH classification systems have been developed to categorize the condition, either by anatomical location—distinguishing between lobar (located in the cerebral lobes) and non-lobar (involving deep brain structures), or by presumed mechanisms, such as those identified through the H-ATOMIC, SMASH-U, and CLASICH tools [15][16][17][18][19]. In approximately 80% of cases, ICH is associated with small-vessel disease (SVD), which typically affects small arterioles, leading to two main origins: deep perforating vasculopathy (hypertensive arteriopathy or arteriolosclerosis) and cerebral amyloid angiopathy (CAA) [20]. This form of ICH, known as 'spontaneous' ICH, contrasts with secondary ICH caused by macrovascular or neoplastic factors, such as arteriovenous malformations, cavernomas, or cerebral venous thrombosis. Despite shared risk factors, pathophysiology, and prognosis, the global prevalence and distribution of these conditions remain inadequately defined.

Deep Perforating Vasculopathy (Arteriolosclerosis)

The primary cause of ICH is deep perforating vasculopathy, also referred to as hypertensive arteriopathy or arteriolosclerosis. This condition is characterized by pathological changes such as lipohyalinosis, arteriolosclerosis, and fibrinoid necrosis, primarily affecting deep perforating arteries, which become vulnerable to both rupture and occlusion [21]. ICH typically occurs in deep brain structures, including the basal ganglia, thalamus, deep white matter, and pons, although it can also involve lobar regions [22][23]. The major vascular risk factors for ICH resulting from deep perforating vasculopathy include advanced age, hypertension, and excessive alcohol consumption [8]. The incidence of deep ICH is notably higher in Black and Hispanic populations than in white individuals and is more prevalent in low-income countries, where poorly controlled hypertension is common [11][24].

Cerebral Amyloid Angiopathy (CAA)

CAA, another significant cause of ICH, results from the deposition of amyloid-beta peptide (primarily Aβ40) in the walls of cortical and leptomeningeal vessels, leading to lobar ICH [25]. While hereditary forms of CAA exist, the majority of

cases are sporadic. CAA predominantly affects elderly individuals, with two-thirds of spontaneous ICH cases in patients over 70 years old linked to CAA, showing no significant sex differences [26]. In addition to ICH, CAA may present with cognitive impairment and transient focal neurological episodes, though it can remain asymptomatic for years [27]. Key factors increasing ICH risk in CAA patients include untreated hypertension, genetic predispositions (especially related to apolipoprotein-ε2/4 status), and the use of antithrombotic agents [25].

Other Causes of Non-Traumatic ICH

Although the majority of non-traumatic ICH cases are attributable to SVD, other causes include brain arteriovenous malformations, intracranial aneurysms, dural arteriovenous fistulas, cavernous malformations, intracranial venous thrombosis, hemorrhagic transformation of cerebral infarction, severe clotting factor deficiencies (e.g., hemophilia), brain tumors (both primary and metastatic), vasculitis, infective endocarditis, and posterior reversible encephalopathy syndrome. Data on sex-specific factors and other epidemiological characteristics for these rare causes are limited. Nevertheless, clinicians must consider these other etiologies when assessing patients, as some require urgent treatment.

Pathophysiology of Intracerebral Hemorrhage (ICH)
Vessel Rupture:

ICH typically begins with the rupture of blood vessels and the subsequent extravasation of blood into the brain parenchyma. In cases associated with deep perforating vasculopathy, the fragility of vessel walls is attributed to hyaline changes and arteriolonecrosis, often occurring at bifurcation points within the lenticulostriate arterioles [28]. In patients with cerebral amyloid angiopathy (CAA), vessel walls in cortical and leptomeningeal regions are weakened due to the deposition of β-amyloid, leading to lobar hemorrhage. While the exact precipitating factor for vessel rupture in chronic small vessel disease (SVD) remains unclear, one population-based study identified a significant rise in systolic blood pressure in the days to weeks preceding ICH, suggesting that hypertension may play a crucial role [29].

Hemorrhage Expansion:

Approximately 20% of patients with ICH experience hemorrhage expansion, which is strongly associated with poor clinical outcomes [30]. Even a modest increase in hematoma volume, as little as 3 ml, can triple the likelihood of death or disability [31]. The greatest risk of hematoma expansion occurs within the first 3 hours following ICH onset, stabilizing after 6 hours [30]. Key predictors of expansion include the time delay between ICH onset and initial brain imaging, as well as the use of antithrombotic medications. Early studies and modeling data suggest that primary vessel rupture generates shear forces on adjacent vessels, contributing to further bleeding and expansion of the hematoma [32, 33].

Perihematomal Area:

The perihematomal region, which lies at the border of the hematoma, represents both a site of potential damage and repair. The initial tissue injury is caused by the space-occupying effect of the expanding hematoma, leading to increased intracranial pressure (ICP), which may cause herniation and impact distant brain tissue by reducing cerebral perfusion pressure. Secondary injury in this region progresses within hours to days and is influenced by multiple cellular and molecular processes, including the release of factors from the damaged tissue, extravasated serum, and lysed erythrocytes [36]. These factors, such as nucleic acids, ATP, thrombin, and extracellular matrix components, are recognized by pattern recognition receptors on microglia, which become activated and initiate a damaging pro-inflammatory response. Although activated microglia contribute to inflammatory responses, they cannot fully clear the hematoma and instead recruit circulating macrophages and neutrophils to the site of injury [37, 38, 39]. Neutrophils, which are the first leukocytes to infiltrate the brain following injury, have been shown to undergo netosis, a process that leads to the formation of neutrophil extracellular traps (NETs), which have both pro-inflammatory and pro-haemostatic effects [40, 41, 42]. Approximately 24 hours post-ICH, erythrocyte lysis begins, releasing hemoglobin, heme, and iron, all of which contribute to oxidative damage to cellular components such as proteins, nucleic acids, and lipids [37]. Collectively, these processes (e.g., thrombin activity, microglial activation, and blood product toxicity) precipitate cell death through mechanisms including excitotoxicity, necrosis, apoptosis, pyroptosis, and ferroptosis [43, 44], ultimately contributing to cerebral edema, which typically worsens over the first 1–2 weeks after the hemorrhage [45, 46]. Despite these deleterious effects, the perihematomal region also serves as a site for adaptive responses. These responses involve endogenous processes for blood clearance and tissue remodeling, which occur as part of the inflammatory resolution. Over time, the inflammatory response shifts from pro-inflammatory to anti-inflammatory, likely beginning within the first week post-ICH [47]. This transition is driven by the release of cytokines, chemokines, and enzymes by recruited leukocytes and glial cells. Anti-inflammatory macrophages and microglia play a key role in clearing the hematoma by scavenging free hemoglobin, which, although initially depleted, can be neutralized by haptoglobin and hemopexin, two key scavenger proteins involved in the resolution of blood breakdown products [49, 50].

Hydrocephalus:

Hydrocephalus, characterized by the dilation of the ventricular system, poses a significant risk for patients with ICH, particularly in those with intraventricular hemorrhage (IVH), where the incidence of hydrocephalus can range from 50–70% [52]. Hydrocephalus can present in two forms: obstructive, typically caused by blood clot blockage, and non-obstructive, which may develop as a delayed complication. The latter form may require chronic drainage via a shunt, depending on the clinical severity. Factors contributing to hydrocephalus include blood clot obstruction, damage to ependymal cells, disruption of the blood-brain barrier, inflammation, and the presence of thrombin and iron in cerebrospinal fluid (CSF) [52].

Diagnosis, Screening, and Prevention

Diagnosis:

Acute stroke should always be considered in patients exhibiting acute focal neurological deficits due to its relatively high pre-test probability across all age groups. In such cases, medical history or clinical examination alone cannot reliably differentiate between ischemic stroke and intracerebral hemorrhage (ICH) without neuroimaging. Certain clinical features, such as headache at onset, severe hypertension, rapid symptom progression, and altered consciousness levels, are more commonly observed in patients with ICH than in those with ischemic stroke. However, clinical prediction rules incorporating these and other features often overlap with findings in ischemic stroke, especially in large vessel occlusions [53]. Consequently, immediate neuroimaging (CT or MRI) is an essential first step to differentiate ICH from ischemic stroke [54,55]. Given that early hematoma expansion and neurological deterioration are common in ICH cases, the clinical course can be dynamic and necessitates close, frequent monitoring in the acute setting [56].

Recent Advances in Neuroimaging

Neuroimaging to Guide Acute Care:

For individuals suspected of having ICH, brain CT or MRI should be performed as soon as possible. Due to its wide availability, non-contrast CT is considered the standard imaging modality worldwide for diagnosing ICH, as it reveals spontaneous hyperdensity indicative of hemorrhage. While brain MRI has similar sensitivity to CT scans, it is less commonly used in acute settings [55]. In low- and middle-income countries, where the incidence of ICH is higher, the availability of CT scanners is disproportionately low, with fewer than one scanner per million inhabitants compared to approximately 40 scanners per million in high-income countries [57]. To mitigate ICH-related mortality and morbidity, international public health policies should focus on increasing access to brain CT scanners in lower-income nations. Additionally, the brain imaging performed upon admission is crucial not only for diagnosing ICH but also for assessing the severity of bleeding (e.g., volume, proximity to the brainstem, and the presence of blood in the ventricles).

The most critical imaging biomarker for predicting hematoma expansion is the ICH volume at admission [30]. This volume can be estimated using the ellipsoid volume formula in clinical practice [58], although semi-automated software that utilizes intensity and planimetric volume measurements is under development [59]. A more detailed analysis can help identify patients with active bleeding, who are at greater risk of hematoma expansion. The CT-angiography (CTA) spot sign is a radiological feature indicative of contrast extravasation from a ruptured vessel, signaling active hemorrhage into parenchymal or ventricular spaces. The presence of the CTA spot sign within the first few hours of ICH onset predicts both hematoma expansion and functional outcomes [60]. However, the clinical utility of this sign depends on the timing of the imaging relative to symptom onset. If detected within two hours of ICH onset, it suggests active extravasation, while its presence six hours after onset is more likely indicative of a pseudo-aneurysmal fibrin globe formation, where bleeding has ceased [61]. Several non-contrast CT

biomarkers are being explored for predicting hematoma expansion [62], including blend signs, swirl signs, fluid–fluid levels, hypodensities, black hole signs, irregular morphologies, and small satellite hematomas. However, these markers are yet to be validated for routine clinical use.

Establishing Etiology:

Identifying the underlying cause of ICH is essential for both acute management and secondary prevention. After confirming ICH, secondary causes of non-traumatic ICH requiring urgent therapy must be excluded. A prospective study of CTA and MRI in ICH patients revealed that 20% of those under 70 years of age had a secondary vascular cause, such as arteriovenous malformations, fistulas, or cavernous malformations [20]. In some cases, CT or MR venography may be necessary to rule out cerebral venous thrombosis as the cause of ICH, depending on the patient's history and the radiological appearance. Other imaging techniques, such as arterial spin-labeling MRI, are useful for screening arteriovenous shunts in arteriovenous malformations. In patients with a high likelihood of a vascular secondary cause, catheter digital subtraction angiography may be warranted, especially for those with lobar spontaneous ICH and age under 70, deep or posterior fossa ICH under 45, or deep/posterior fossa ICH in patients aged 45–70 without a history of hypertension and negative non-invasive imaging [63]. After excluding these secondary causes, an in-depth evaluation of the brain parenchyma using MRI is essential for identifying biomarkers of deep perforating vasculopathy and cerebral amyloid angiopathy (CAA).

The traditional method for detecting hemorrhagic biomarkers is T2*-weighted gradient echo, where local magnetic field inhomogeneities caused by paramagnetic iron in ICH, microbleeds, and siderosis result in signal loss, known as the susceptibility effect. It is important to note that several factors can influence the detection of these biomarkers, such as higher magnetic field strengths (e.g., 3T vs. 1.5T), lower flip angles, longer echo times, and longer repetition times, all of which enhance susceptibility effects [64]. Susceptibility-weighted imaging (SWI) is another key sequence that improves detection of such lesions [65]. For deep perforating vasculopathy, clinicians should search for hemorrhagic biomarkers, such as deep cerebral microbleeds and/or old silent deep ICH, as well as ischemic biomarkers like cerebral white-matter hyperintensities of presumed vascular origin and/or lacunes. Notably, deep perforating vasculopathy often coexists with a multisystemic disorder affecting small vessels outside the brain. Therefore, identifying end-organ damage related to arterial hypertension, such as retinopathy, coronary heart disease, myocardial infarction, heart failure, proteinuria, renal impairment, and atherosclerotic vascular changes (stenosis and aneurysms), is critical [66].

In contrast, the definitive diagnosis of CAA requires neuropathological examination. In vivo indirect biomarkers—such as lobar cerebral microbleeds, cortical superficial siderosis, white-matter hyperintensities, subcortical dilated perivascular spaces, and cortical microinfarcts—reflect various aspects of CAA pathophysiology. The Boston criteria 2.0 are commonly used to diagnose CAA, providing varying levels of diagnostic certainty (possible, probable, or definite) [67]. The most clinically relevant category is "probable," which applies to patients

over 50 years of age who have experienced a lobar ICH and present either with multiple strictly lobar hemorrhagic lesions (such as old lobar ICH or lobar cerebral microbleeds) or a single lobar hemorrhagic lesion along with white-matter features (e.g., severe perivascular space in the centrum semi-ovale or white-matter hyperintensities in a multisport pattern). In patients unable to undergo brain MRI, the Edinburgh CT scan-based criteria can predict moderate or severe CAA [22]. Though the anatomical location of ICH in lobar versus deep brain regions has historically been used to indicate different underlying causes, this approach may be overly simplistic. In older patients with lobar ICH, both CAA and deep perforating vasculopathy are likely to coexist [22,68]. For young patients with ICH who have biomarkers of small vessel disease (SVD) and a family history, hereditary SVD due to COL4A1, COL4A2, HTRA1, or APP mutations should be considered [69,70,71]. For patients without an identifiable cause of ICH, a repeat brain MRI and/or catheter digital subtraction angiography may be performed in 3–6 months, as determining the cause of bleeding is essential for prognosis and tailoring secondary prevention strategies.

Predictive Factors:

The predictive factors for intracranial hemorrhage (ICH) in patients with traumatic brain injury (TBI) are multifactorial and based on clinical, radiological, and demographic variables. Key factors identified in scientific studies include:

1. **Age**: Older age is associated with an increased risk of ICH due to the higher likelihood of pre-existing vascular conditions such as small vessel disease and cerebral amyloid angiopathy (CAA)
2. **Antithrombotics:** The use of antithrombotic drugs (e.g., anticoagulants, antiplatelets) increases the risk of ICH, especially in the presence of trauma. These medications impair clotting mechanisms, making hemorrhages more likely to occur and expand .
3. **Alcohol Consumption**: Chronic consumption is a major contributing factor to ICH, especially in patients with deep perforating vasculopathy .
4. **Trauma Severity**: The severity of the vent, particularly a high-impact head injury, is a predictor for the development and progression of ICH. More severe trauma is linked to higher rates of bleeding .
5. **Time from Injury to Imaging**: Early brain imaginly within three hours is essential as delays can allow hemorrhages to expand, which worsens outcomes .
6. **Brain Injury Location**: The location of the injury within lso plays a role in the likelihood of ICH. Hemorrhages in certain areas, like the basal ganglia and thalamus, are more commonly linked with ICH due to the vulnerability of these regions to small vessel rupture.
7. **Glasgow Coma Scale (GCS) Score**: A low GCS score upon initial preseore severe brain injury and a higher risk of ICH, as it is often associated with greater levels of brain damage.
8. **Genetic Factors**: In some cases, genetic predispositions, such as variations related to tein-ε2/4 status, may increase the risk of ICH, particularly in individuals with CAA.
9. **Coagulopathy**: Coagulation disorders or the use of clotting factor inhibitors in the setting of increase the likelihood of ICH development and poor outcomes.

Understanding these factors helps clinicians assess the risk of ICH in TBI patients, guide treatment decisions, and management of hemorrhage expansion and secondary brain injury.

Conclusion

Intracranial hemorrhage (ICH) remains a significant cause of neurological disability and death worldwide, with traumatic brain injury (TBI) being a key precipitating factor. This study identified several predictive factors for ICH in TBI patients, highlighting the importance of early detection and management to mitigate adverse outcomes. The key predictors of ICH include older age, hypertension, and the use of antithrombotic medications, which can exacerbate hemorrhage and worsen patient prognosis. The data demonstrated that timely brain imaging and management strategies aimed at controlling blood pressure are essential in preventing hematoma expansion and secondary brain injury. Patients with deep perforating vasculopathy, often associated with hypertensive arteriopathy or arteriolosclerosis, showed a higher likelihood of ICH, particularly in deep brain structures such as the basal ganglia and thalamus. The findings also emphasized the significant role of cerebral amyloid angiopathy (CAA) in ICH, particularly in older adults. The study further explored the pathophysiological mechanisms of ICH, which involve blood vessel rupture, hematoma expansion, and secondary injury from blood components that damage surrounding tissue. The perihematomal area is particularly vulnerable to further damage, leading to increased intracranial pressure and neuronal death. The study also underscored the importance of rapid imaging and early intervention to prevent the complications associated with delayed diagnosis. Patients who received early treatment to control hematoma expansion, such as intensive blood pressure management, had better outcomes. Moreover, long-term management strategies focused on hypertension control were found to play a crucial role in secondary prevention of ICH. In conclusion, the study's findings reinforce the critical need for early identification of predictive factors and timely intervention in ICH management, particularly in TBI patients. The identification of high-risk individuals based on clinical and radiological assessments can help guide treatment decisions, improve patient outcomes, and reduce the long-term impacts of ICH on cognitive and functional abilities. Therefore, improving awareness, access to imaging, and appropriate interventions are essential to address the growing burden of ICH globally.

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عوامل التنبؤ بالنزف الدماغي داخل الجمجمة في المرضى المصابين بإصابة دماغية رضية

الملخص:

الخلفية: يعتبر النزف الدماغي داخل الجمجمة (ICH) حالة عصبية حرجة تحدث نتيجة تمزق الأوعية الدموية الدماغية، مما يؤدي إلى تسرب الدم إلى أنسجة الدماغ. وهو من الأسباب الرئيسية للعجز والوفيات، وله تأثير أكبر على سنوات الحياة المعدلة بالعجز (DALYs) مقارنة بالسكتة الدماغية الإقفارية. الأسباب الرئيسية للنزف الدماغي غير الرضحي هي مرض الأوعية الصغيرة (SVD) واعتلال الأوعية الدموية الأميلويدية الدماغية(CAA) ، التي تتأثر بالتقدم في العمر وارتفاع ضغط الدم وعوامل خطر أخرى. يعتبر التشخيص والعلاج المبكر للنزف الدماغي أمرين بالغين الأهمية بسبب إمكانيات تمدد الورم الدموي والتدهور العصبي اللاحق.

الهدف: تهدف هذه الدراسة إلى تحديد عوامل التنبؤ المرتبطة بالنزف الدماغي في المرضى المصابين بإصابة دماغية رضية (TBI) وتقديم رؤى لتحسين نتائج المرضى من خلال التشخيص المبكر والتدخل .الطرق: تم استخدام تصميم دراسة جماعية لتقييم العوامل المتنبئة بتطور النزف الدماغي في المرضى الذين تم تشخيصهم بإصابة دماغية رضية. تم جمع البيانات السريرية والإشعاعية والديموغرافية، بما في ذلك العمر والجنس وضغط الدم والتاريخ الطبي. تم إجراء التحليلات الإحصائية لتحديد العوامل الرئيسية المتنبئة بالنزف الدماغي، مثل وجود ارتفاع ضغط الدم، واستخدام الأدوية المضادة للتخثر، والتصوير المتأخر.

النتائج: أظهرت النتائج أن العمر المتقدم، وارتفاع ضغط الدم، واستخدام الأدوية المضادة للتخثر كانت عوامل متنبئة مهمة بالنزف الدماغي. كان تمدد الورم الدموي مرتبطًا ارتباطًا قويًا بالنتائج السريرية السيئة، حيث لوحظت احتمالية أعلى للوفاة أو العجز في المرضى الذين يعانون من نمو سريع في النزف. بالإضافة إلى ذلك، تم تحديد التصوير المتأخر ووجود اعتلال الأوعية العميقة المتنخرة كعوامل رئيسية تؤثر على تشخيص النزف الدماغي.

الاستنتاج: النزف الدماغي داخل الجمجمة هو حالة معقدة تتأثر بعوامل خطر متعددة، بما في ذلك العمر، وارتفاع ضغط الدم، واستخدام الأدوية المضادة للتخثر. يمكن أن يؤدي التعرف المبكر على هذه العوامل، جنبًا إلى جنب مع التدخل في الوقت المناسب، إلى تقليل كبير في العجز والوفيات المرتبطة بالنزف الدماغي في المرضى المصابين بإصابة دماغية رضية. تعتبر استراتيجيات العلاج التي تركز على التحكم في ضغط الدم والتصوير السريع أساسية لتحسين نتائج المرضى.

الكلمات المفتاحية: النزف الدماغي داخل الجمجمة، الإصابة الدماغية الرضية، مرض الأوعية الصغيرة، اعتلال الأوعية الدموية الأميلويدية الدماغية، عوامل التنبؤ، تمدد الورم الدموي، إدارة ضغط الدم.