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Risk factors for infarct growth and haemorrhagic or oedematous complications after endovascular treatment — a literature review

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ABSTRACT

Introduction. Acute ischaemic stroke (AIS) is caused by significant disturbances in the cerebral bloodflow (CBF) that lead to brain ischaemia and eventually result in irreversible brain tissue damage. The main goal of its treatment is to restore bloodflow to the areas at risk of necrosis. Intravenous thrombolysis (IVT) and mechanical thrombectomy (MT) are the mainstay of current therapy, with the latter being widely employed in selected patients with radiologically proven large vessel occlusion (LVO). Despite convincing evidence of its efficacy, up to half of patients undergoing endovascular treatment (EVT) still do not achieve a beneficial functional outcome; this is mainly due to unfavourable brain tissue sequelae. Therefore, factors associated with known adverse brain changes, such as larger infarct size or haemorrhagic and oedematous complications, should be adequately addressed.

Objectives. To review the available literature describing AIS brain tissue outcome assessed by computed tomography (CT) and/ /or magnetic resonance imaging (MRI) in patients undergoing MT treatment. Additionally, to evaluate the association of post- -MT tissue changes with short- and long-term prognosis.

Material and methods. We searched the PubMed, Scopus, EMBASE, and Google Scholar databases according to established criteria.

Results. We found a total of 264 articles addressing the most common types of AIS tissue sequelae after EVT (i.e. MT with or without IVT as bridging therapy) by brain CT and MRI. These were: follow-up infarct volume (FIV), cerebral oedema (COD) and haemorrhagic transformation (HT). As the next step, 37 articles evaluating factors associated with defined outcomes were selected. Several non-modifiable factors such as age, comorbidities, pretreatment neurological deficit, and collateral circulation status were found to affect stroke tissue sequelae, to varying degrees. Additionally, some factors including time to treatment initiation, selection of treatment device, and periprocedural systemic blood pressure, the modification of which can potentially reduce the occurrence of an unfavourable tissue outcome, were identified. Some recently revealed biochemical and serological parameters may play a similar role.

Conclusions. The identification of factors that affect post-MT ischaemic area evolution may result in studies assessing the effects of their modification, and potentially improve clinical outcomes. Modifiable parameters, including periprocedural systemic blood pressure and some biochemical factors, may be of particular importance.

Key words: ischaemic stroke, endovascular treatment, follow-up infarct volume, haemorrhagic transformation, brain oedema

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Introduction

Stroke is the second most common cause of death worldwide after ischaemic heart disease [1]. It disables several million people every year, resulting in a significant global economic burden. Therefore, advances in therapy and the identification of factors that could improve stroke survivors' prognoses are important objectives of healthcare organisations [2].

Acute ischaemic stroke (AIS) is associated with a significant cerebral bloodflow (CBF) impairment that leads to brain ischaemia [3, 4]. The area of tissue damage with irreversible bioenergetic failure and necrosis is called the ischaemic core. The functionally impaired surrounding zone, called the penumbra, can potentially be saved by restoring local perfusion. Without prompt reperfusion, this salvageable area will eventually turn into a necrotic ischaemic core [5]. Therefore, a swift restoration of bloodflow to the penumbra zone is the main goal of AIS therapy [6]. The two currently recommended reperfusion treatment methods are intravenous thrombolysis (IVT) and mechanical thrombectomy (MT). The latter has been shown to have higher recanalisation rates and provide better functional outcomes than IVT alone in the setting of radiologically confirmed large vessel occlusion (LVO) [7, 8]. It is thus considered as the mainstay of therapy in this selected group of patients [9]. IVT is recommended as the bridging therapy for MT treatment [10].

Despite the convincing evidence of the efficacy of endovascular treatment (EVT) in the management of LVO-caused AIS, up to half of patients treated with this method do not achieve a favourable functional outcome [11]. Tissue changes that can be easily assessed by noninvasive imaging methods soon after MT treatment may potentially serve as a rapidly available and convenient surrogate marker for short- and long-term functional outcomes. These markers may include infarct size and haemorrhagic or oedematous changes.

Therefore, the aim of our review was to analyse the available literature that describes the influence of selected factors on the tissue sequelae of AIS evaluated by neuroimaging studies in patients treated with MT. At the same time, we evaluated the role of these changes in the patient's short- and long-term prognoses.

Material and methods

An electronic literature search and review was performed in April 2022 to evaluate the most common AIS tissue sequelae of post-MT treatment noninvasive assessment with widely available imaging studies: computed tomography (CT) or magnetic resonance imaging (MRI). They were: final infarct volume (FIV), cerebral oedema (COD), and haemorrhagic transformation (HT). The scientific databases PubMed, Embase, and Scopus were searched using the following searching terms: 'acute ischaemic stroke'; 'infarct volume'; 'haemorrhagic transformation'; 'brain oedema'; 'thrombectomy'; 'endovascular therapy'; , and 'predictors'. Bolean operators 'AND' were used to combine search terms in different lists. In addition, all selected articles were scanned for references to other potentially relevant papers.

Results

A total of 264 articles that met the criteria were identified. As the next step, 37 articles presenting the association of modifiable and non-modifiable parameters with the radiologically evaluated tissue sequelae of AIS were selected and analysed in detail.

In all of the reviewed articles, post-treatment follow-up imaging (CT or MRI) was performed 24–36 hours after the initiation of therapy, or earlier if there was significant neurological deterioration. It is worth noting that we found no paper summarising all these correlations together in the available literature. The review results are set out in Tables 1–3.

Additionally, the relationship between identified AIS post-MT tissue sequelae and patient functional outcomes was reviewed. This is set out in Table 4.

Discussion

Factors associated with acute ischaemic stroke tissue sequelae after EVT

Follow-up Infarct Volume

Recanalisation of LVO with brain tissue reperfusion is thought to be the strongest predictor of FIV. In *post-hoc* analysis of the MR CLEAN trial, patients who underwent EVT had smaller infarct size on follow-up imaging compared to the controls [12]. Post-MT FIV has been shown to be closely related to the degree of recanalisation [13]. The median FIV was found to be smaller, with at least a 2b score on the Treatment in Cerebral Ischaemia (TICI) scale (successful reperfusion) compared to only partial or no reperfusion (TICI 0-2a) [14]. A larger reduction of FIV has been observed in patients with greater neurological deficit [> 14 points assessed by National Institutes of Health Stroke Scale (NIHSS)]. Therefore, NIHSS score on admission, as a surrogate marker of tissue-at-risk size, may also be associated with FIV [15].

Time to reperfusion is another pivotal parameter affecting the final size of the ischaemic area. Rapid recanalisation helps to reduce infarct growth and limits eventual volume of the necrotic zone. This association may be even more pronounced in subjects with moderate-to-poor collateral circulation that accelerates the infarct growth [16]. Similarly, good collateral flow has been shown to be associated with smaller initial infarct core and lower FIV [13, 17]. Both high hypoperfusion index ratio (HIR) and lower relative cerebral blood volume (rCBV) have been shown to be independent predictors of increased FIV in the subgroup of endovascularly treated patients [16]. Therefore, assessing the quality of collateral circulation may

Table 1. Factors affecting follow-up infarct volume in patients treated with mechanical thrombectomy

Parameter	Publication	Factor	Correlations
FIV	Demeestere et al. 2021 [23]	Gender	Lower infarct progression and FIV in women (median 26 mL *vs.* 50 mL for men, p < 0.001)
	Arenillas et al. 2018 [16]	Collateral circulation	Higher FIV values with high HIR and lower rCBV
	Al-Dasuqi 2020 [13]		Collateral status is predictor of FIV (p < 0.001)
	Compagne et al. 2019 [12]	Treatment used	Lower values in patients treated with MT
	Boers et al. 2019 [14]	Degree of recanalisation achieved	Lower values are associated with successful recanalisation (28 mL ≥ 2b *vs.* 86 mL < 2b, p < 0.001)
	Al-Dasuqi et al. 2020 [13]		Patients with successful reperfusion had smaller FIV (p < 0.001)
	Simonsen et al. 2018 [18]	Type of anaesthesia	Lower values with GA. No significant differences of infarct progression between GA and CS subgroups
	Pikija et al. 2018 [20]	Haemodynamic parameters	Lower values correlate with higher SBP deviation rates during MT (to > 120% of pretreatment values)
	Petersen et al. 2019 [21]		Higher values correlate with higher ΔMAP (difference between MAP on admission and lowest MAP during EVT until recanalisation). Each 10 mmHg decrease in MAP before reperfusion increased risk of worse outcomes by 22%
	Maglinger et al. 2021 [22]	Biomarkers	Intracranial VCAM1 correlates with infarct volume

CS — conscious sedation; EVT — endovascular therapy; FIV — follow-up infarct volume; GA — general anaesthesia; HIR — hypoperfusion index ratio; MAP — mean blood pressure; MT — mechanical thrombectomy; rCBV — relative cerebral blood volume; SBP — systolic blood pressure; VCAM 1 — vascular cell adhesion molecule 1

Table 2. Factors affecting haemorrhagic transformation in patients treated with mechanical thrombectomy

Parameter	Publication	Factor	Correlations
Hemorrhagic transformation	Broussalis et al. 2013 [32]	Type of MT device used	Greater risk with older Merci-type systems
	Feng et al. 2020 [33]	Collateral status	Higher risk associated with lower collateral status
	Cao et al. 2020 [34]		Lower risk associated with good collateral status (OR 0.76, 95% CI 0.73–0.80)
	Enomoto et al.2020 [30]	Comorbidities	Higher risk in patients with diabetes (30.1% *vs.* 17.2%; p = 0.006) and with anticoagulant therapy (30.1% *vs.* 19.3%; p = 0.026)
	Feng et al. 2020 [33]		Higher risk in patients with atrial fibrillation (61.8% *vs.* 37.5%, p = 0.025) and coronary artery diseases (61.8% *vs.* 25.0%, p = 0.001)
	Cao et al. 2020 [34]		Atrial fibrillation (OR 2.35, 95% CI 1.96–2.82), and higher serum glucose levels (OR 1.70, 95% CI 1.57–1.85) are independent risk factors of haemorrhagic transformation
	Enomoto et al.2020 [30]	Time to recanalisation	Higher risk in patients with longer onset to reperfusion time [294.5 (195–461) min *vs.* 241 (185–354) min; p = 0.017]
	Honig et al. 2022 [29]	Time to admission	Longer time from symptom onset to admission (OR 1.002 per minute 95% CI 1.001–1.003) independently associated with parenchymal haematoma
	Lee et al. 2019 [27]	Thrombectomy procedure duration	Longer procedure duration (OR = 1.046, 95% CI 1.016 to 1.077, p = 0.003) independently associated with higher chance of parenchymal haematoma
	Rizvi et al. 2019 [28]	Efficiency of recanalisation	Higher risk with lower TICI score
	Lee et al. 2019 [27]		Successful reperfusion (OR = 0.246, 95% CI 0.093 to 0.651, p = 0.005) independently associated with lower chance of parenchymal haematoma
	Feng et al. 2020 [33]	Biochemical parameters	Higher risk with higher PLR
	Lee et al. 2019 [27]		Hyperlipidemia (OR = 0.221, 95% CI 0.064 to 0.767, p = 0.017) independently associated with lower chance of parenchymal haematoma
	Honig et al. 2022 [29]		Hyperlipidemia (OR 3.12; 95% CI 1.12–8.7) independently associated with risk of parenchymal haematoma (PH 2)
	Jian et al. 2020 [46]		Elevated admission bilirubin is independent predictor of haemorrhagic transformation
	Lin et al. 2021 [47]		Lower fibrinogen levels (OR, 0.41; 95% CI, 0.23–0.72; p = 0.002) and platelet counts (OR, 0.58; 95% CI, 0.33–0.99; p = 0.048) independently associated with higher risk of haemorrhagic transformation
	Kim et al. 2020 [48]		Higher risk correlates with higher values highsensitivity CRP (≥ 3 mg/L)
	Diestro et al. 2021 [50]		Higher neutrophil counts, low WBC counts, low lymphocyte counts, and low PLR associated with haemorrhagic transformation
	Qiu et al. 2022 [49]		Lower baseline serum magnesium levels (< 0.80 mmol/L) on admission associated with increased risk of HT

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Table 2. cont. Factors affecting haemorrhagic transformation in patients treated with mechanical thrombectomy

Parameter	Publication	Factor	Correlations
	Pikija et al. 2018 [20]	Haemodynamic parameters	Lower risk with higher mean SBP/MAP during MT
	Feng et al. 2020 [33]		Higher risk correlates with higher systolic blood pressure on admission (155.8 *vs.* 137.8 mmHg, p = 0.001)
	Mistry et al. 2017 [41]		Higher values of SBP independently correlated with higher rate of haemorrhagic complications
sICH	Jiang et al. 2015 [52]	Gender	Higher incidence in women (OR 8.50, 95% CI 1.71–42.17)
	Zang et al. 2021 [24]	Comorbidities	Higher risk in patients with diabetes and hyperglycaemia on admission
	Huang et al. 2021 [44]		Increased rate of sICH in patients with AF after MT compared to those without AF
	Lasek-Bal et al. 2022 [45]		AF did not increase risk of sICH
	Cabrera-Maqueda et al. 2021 [37]	Bridging therapy	MCA had higher risk of sICH after bridging therapy than without (16.4% *vs.* 8.6%, p = 0.038)
	Chen et al. 2021 [38]		No significant difference in clinical effect between direct EVT and bridging therapy
	Qian et al. 2020 [35]	Neurological deficit	More frequent in patients with greater neurological deficit (NIHSS)
	Tian et al. 2021 [36]		Higher risk in patients with greater neurological deficit (NIHSS) — adjusted OR, 1.06 (95% CI, 1.10–1.12)
	Aly et al. 2021 [51]	Biochemical parameters	Lower risk with lower NRL values
	Lasek-Bal et al. 2021 [64]		Elevated CRP and leukocytosis on first day of stroke increases risk of sICH
	Tian et al. 2021 [36]		Higher risk with higher glucose levels on arrival at hospital — adjusted OR, 1.14 (95% CI, 1.00–1.29)
	Li et al. 2020 [43]		Postoperative glucose values independently associated with sICH
	Anadani et al. 2019 [40]	Haemodynamic parameters	Higher risk with higher mean and maximum SBP

AF — atrial fibrillation; CRP — C-reactive protein; EVT — endovascular therapy; HT — haemorrhagic transformation; MAP — mean blood pressure; MCA — middle cerebral artery; MT — mechanical thrombectomy; NIHSS — National Institutes of Health Stroke Scale; NRL — neutrophil-to-lymphocyte ratio; PLR — platelet-to-lymphocyte ratio; SBP — systolic blood pressure; sICH — symptomatic intracranial haemorrhage; TICI — Treatment in Cerebral Ischaemia; WBC — white blood cell

Table 3. Factors affecting cerebral oedema in patients treated with mechanical thrombectomy

Parameter	Publication	Factor	Correlations
Cerebral oedema	Du et al. 2020 [56]	Comorbidities	Higher risk in patients with hypertension, atrial fibrillation and diabetes
Malignant cerebral oedema	Huang et al. 2021 [44]	Haemodynamic parameters	Higher risk with higher mean SBP and increased SBP variability during first 24 hours after EVT (especially > 165 mmHg)
	Kauw et al. 2022 [55]	Cerebrospinal fluid volume	Lower CSF/ICV ratio associated with occurrence of malignant cerebral oedema (OR per percentage point, 1.2; 95% CI 1.1–1.3, p < 0.001).
	Tracol et al. 2020 [53]	Time to thrombectomy treatment	Time to MT (p = 0.018) independent predictor of malignant cerebral oedema
	Huang et al. 2019 [54]	Collateral status	Worse collateral score significantly associated with risk of malignant brain oedema (grade 1 *vs.* grade 0: OR = 0.727; 95% CI 0.192 to 2.753; p = 0.638; grade 2 *vs.* grade 0: OR = 0.130; 95% CI 0.021 to 0.819; p = 0.030)
	Huang et al. 2019 [54]	Occlusion localisation	ICA occlusion is associated with risk of malignant brain oedema (OR = 3.746; 95% CI 1.169 to 12.006; p = 0.026)
	Huang et al. 2021 [44]	Efficiency of recanalisation	Lower risk in patients with successful recanalisation (14% *vs.* 34.3%)

CSF/ICV — intracranial cerebrospinal fluid/intracranial volume; EVT — endovascular therapy; ICA — internal carotid artery; MT — mechanical thrombectomy; SBP — systolic blood pressure

Table 4. Acute ischaemic stroke sequelae affecting functional outcome

Parameter	Publication	Correlations
FIV	Compagne et al. 2019 [12], analysis of randomised clinical trials	Smaller FIV associated with better outcome (acOR per 10 mL 0.60, 95% CI 0.52–0.68)
	Boers et al. 2018 [58], analysis of randomised clinical trials	Large FIV associated with worse functional outcome [OR = 0.88 (95% CI 0.87 to 0.89) per 10 mL]
		FIV of ≥ 133 mL specific for unfavourable outcome
	Al-Aljani et al. 2016 [57], analysis of randomised clinical trials	FIV independently associated with 90-day modified Rankin Scale
Haemorrhagic transformation	Kaesmacher J et al. 2017 [59], retrospective analysis	Haemorrhagic infarction and parenchymal haematoma independently associated with lower rates of good neurological outcome (aOR 0.086, 95% CI 0.008–0.902, p = 0.041 and aOR 0.282, 95% CI 0.131–0.606, p = 0.001)
	Li et al. 2020, [43] retrospective analysis	Parenchymal haematoma associated with mortality at 90 days
	Shen et al. 2022, [60] retrospective analysis	Mortality higher for patients with sICH (54.8 *vs.* 25.4%, p < 0.001)
		NIHSS score within 24 h after EVT higher in patients with sICH than in patients without sICH [16 (12, 25) *vs.* 13 (8, 18), p < 0.001]
		Patients with sICH showed worse functional outcomes (93.5% of cases)
Cerebral oedema	Davoli et al. 2018 [63], observational study	Malignant cerebral oedema may reduce beneficial effects of EVT

EVT — endovascular therapy; FIV — follow-up infarct volume; NIHSS — National Institutes of Health Stroke Scale; sICH — symptomatic intracranial haemorrhage

be helpful in estimating the tissue-at-risk size and functional treatment effect.

The type of anaesthesia may be another modifiable factor associated with post-MT follow-up infarct size. In one study, FIV was smaller in a group of patients undergoing EVT under general anaesthesia (GA) compared to conscious sedation (CS), but without statistically significant differences of the infarct progression between both groups [18].

The role of periprocedural haemodynamic parameters on treatment outcomes of MT is now widely discussed. Dynamic autoregulation of the cerebrovascular circulation maintains a relatively constant CBF over a wide range of systemic blood pressure (BP). In AIS, the focal cerebral autoregulation within the ischaemic area may be impaired, exposing the penumbra zone to the deleterious fluctuations of systemic BP. Its excessive variability may, therefore, lead to faster infarct core progression and larger FIV [19]. It has been shown that higher intraprocedural SBP deviation rate (a value assessed before the start of the procedure and the induction of anaesthesia) was associated with lower FIV and better functional outcome [20]. Petersen et al. highlighted that up to 87% of patients during EVT may experience a reduction in MAP, potentially affecting clinical outcomes. One study found that ΔMAP (defined as the difference between MAP on admission and the lowest MAP during EVT until recanalisation) was associated with increased infarct growth and larger final infarct volume [21].

One study has shown increased levels of systemic and intracranial vascular cell adhesion molecule 1 ($VCAM_1$) to be associated with larger infarct volume and worse prognosis in MT-treated patients [22]. Although the nature of this association is not fully understood, it points to some promising new biomarkers of ischaemic brain tissue damage and clinical outcome.

Demographic factors affecting FIV, such as age and sex, have been addressed by several studies. In some analyses, women were found to have better collateral circulation, which probably accounted for smaller ischaemic lesions on baseline perfusion imaging. Females also had a slower progression of ischaemic core and lower FIV [23]. According to the same study, the smaller infarct volume in women correlated with better mRS scores and a lower likelihood of severe disability or death. This suggests that women may have better clinical outcomes following EVT. Despite the fact that older patients have a worse prognosis than younger ones, age does not appear to be related to infarct growth or larger FIV after EVT [24, 25]. This could modify the association between FIV and functional outcome, indicating a lesser FIV threshold for poor prognosis among older people [26].

Factors affecting the follow-up infarct volume in patients treated with endovascular treatment are set out in Table 1.

Haemorrhagic transformation

Similarly to final infarction size, haemorrhagic transformation (HT) of AIS has been found to be associated with the degree and timing of reperfusion in MT-treated patients. Successful recanalisation has been shown to be an independent protective factor for parenchymal haematoma (PH). Even within the cohort of successful reperfusion, intracerebral haemorrhage (ICH) rates were found to be significantly lower in cases of TICI-3 compared to TICI-2b [27, 28]. Longer time from symptom onset to hospital admission, longer time from symptom onset to reperfusion time, and longer EVT procedure duration are all associated with higher HT incidence [27, 29, 30]. Such findings point to the protective effect of promptly introduced MT on haemorrhagic stroke complications, although

metanalysis of five randomised clinical trials (RCTs) assessing MT versus controls did not show statistically significant differences in the incidence of PH or symptomatic intracranial haemorrhage (sICH) between both groups [31]. These effects may be also influenced by the type of MT device used, with older Merci-type systems posing a greater risk of HT [32].

The association of timely reperfusion with a lower risk of HT suggests that other factors influencing the pace of progression of penumbra to infarct core could play a similar role. This was confirmed by two studies finding poor collateral status to be an independent risk factor of post-MT HT [33, 34]. Similarly, haemorrhagic complications of EVT might be affected by the size of tissue at risk, as neurological deficit on admission measured by NIHSS score has been found to increase the risk of sICH after EVT [35, 36].

Therefore, it appears that the size of hypoperfused brain tissue, the volume of ischaemic core at the time of reperfusion, and the degree of recanalisation may be the primary factors affecting the risk of post-MT HT, with timing of successful recanalisation, baseline NIHSS score, and collateral status being their surrogate markers in the assessment of brain tissue dynamic changes [27, 28].

There is contradictory data regarding an association between IVT bridging therapy and post-MT haemorrhagic complications. Some authors have found the risk of sICH to be higher with bridging IVT administration, but others did not show such a relationship [37, 38]. In one study, bridging IVT significantly increased the risk of sICH in patients with less than 6 points in the Alberta Stroke Programme Early CT Scores (ASPECTS) scale on pretreatment imaging [39].

EVT is associated with significant variability of systemic BP. Systemic BP changes may affect local haemodynamic parameters within brain vasculature and increase the incidence of haemorrhagic treatment complications. It has been shown that higher values of pretreatment SBP and mean SBP/MAP during intervention are associated with a lower likelihood of HT [20, 33]. On the other hand, patients with higher mean and maximum post-treatment SBP have a higher risk of ICH and sICH [40, 41]. The protective effect of higher intraprocedural systemic BP on haemorrhagic complications may be explained by avoiding the deleterious consequences of systemic hypotension, which is thought to accelerate penumbra to infarct core progression by compromising collateral flow. On the other hand, hypertension after MT treatment has been found to elevate the HT risk [42]. Bloodflow restoration, in terms of previous ischaemic endothelial injury and increased tissue permeability, may explain this observation.

There is some data showing that several comorbidities and biochemical parameters affect the incidence of HT after MT treatment. A prior-to-stroke diagnosis of diabetes and higher admission blood glucose levels significantly elevate the risk of both HT and sICH [24, 34, 36]. Post-procedural hyperglycaemia was also found to be an independent risk factor of a latter haemorrhagic post-treatment complication [43]. Several studies have shown that the incidence of HT may also be increased in patients with a diagnosis of atrial fibrillation (AF) [33, 34], whereas data on sICH frequency in this group of patients is contradictory. Some authors have found AF to be associated with sICH occurrence [44], while others have not [45]. Coronary artery disease is another comorbidity that may affect post-MT HT occurrence [33].

Other biochemical factors found to elevate the risk of post-MT HT are: high levels of bilirubin and C-reactive protein (CRP), and low fibrinogen and magnesium (< 0.80 mmol/L). Higher levels of CRP also affected the sICH incidence [46–49]. An association of some serological markers with post-EVT haemorrhage has also been demonstrated, with, among these, low platelets and white blood cells, along with high neutrophil counts, being risk factors of HT [50].

A promising indicator of neuroinflammation that can effectively predict the radiological outcome in patients with AIS is the neutrophil-lymphocyte ratio (NLR). Its lower value and slower progression over time is an independent predictor of reduced risk and mortality from sICH [51]. Conflicting data exists on the association of hyperlipidemia and platelet-to-lymphocyte ratio (PLR) with HT. Both high and low values of these parameters have been indicated as risk factors for haemorrhagic AIS transformation by different authors [27, 29, 33, 50]. The exact mechanisms of how biochemical and serological parameters influence post-EVT HT occurrence is not well understood. Hyperglycaemia probably aggravates ischaemic injury by promoting neuronal death due to intracellular metabolic disturbances, while other abovementioned factors (e.g. NRL) may be surrogate markers for neuroinflammation.

Some authors have found women to have a higher risk of sICH after AIS endovascular management [52]. To the best of our knowledge, no other demographic factor has been found to influence HT after MT.

The factors affecting HT in patients treated with MT are set out in Table 2.

Cerebral oedema

Considering that COD is a natural consequence of focal brain ischaemia that primarily results from tissue necrosis (cytotoxic oedema), factors influencing post-treatment infarct size should also play a significant role in its formation and eventual extent.

This hypothesis is confirmed by several observations that include: the association of malignant brain oedema (MBO) incidence with size of diffusion-weighted imaging (DWI) infarct volume on pre-treatment imaging, the time to MT treatment initiation, and the degree of recanalisation achieved [44, 53]. Similarly, poor collateral flow has been found to be an independent risk factor of MBO [54]. Another factor observed to be an independent MBO predictor was LVO localisation, with intracranial ICA occlusion associated with its higher occurrence [54].

Another possible mechanism of COD formation is the reperfusion syndrome that results from bloodflow restoration into the area of the disrupted blood-brain barrier due to

prior ischaemia [4]. This process might be intensified by high post-reperfusion systemic BP. It has been shown that patients with higher mean SBP in the first 24 hours after recanalisation and those with its increased variability are at higher risk of MBO [44]. This finding may suggest that close BP monitoring and management after EVT can be potentially beneficial in terms of improving outcome.

Recently, another interesting factor has been shown to be associated with MBO. A lower ratio of cerebrospinal fluid to intracranial fluid volume has been found to be an independent risk factor [55].

Several comorbidities, such as hypertension, atrial fibrillation, and diabetes have been found to be associated with MBO formation, and a similar effect was described for hyperglycaemia [56].

Factors influencing COD magnitude among patients treated with MT are set out in Table 3.

Acute ischaemic stroke tissue sequelae and functional outcome

Prevention of FIV progression may at least partially explain the beneficial effect of MT on patients' prognoses [57]. This was confirmed by the pooled analysis of data from seven RCTs by Boers et al. that showed MT to be independently associated with lower FIV, which in turn appeared to be a predictor of functional outcome. Larger FIV was also associated with a poorer prognosis, with its threshold value of 133 mL. being highly specific for an unfavourable clinical outcome [58]. Similarly, post-MT parenchymal haematoma has been shown to be independently associated with lower rates of good neurological outcome and higher 90-day mortality [42, 59]. SICH is associated with poorer short- and long-term functional outcomes and higher mortality [60].

Another post-EVT unfavourable brain tissue consequence affecting clinical outcome is COD. Increased intracranial pressure secondary to oedema is associated with a poor prognosis, and in case of mass effect that can lead to brain herniation is an important element in determining the immediate risk of mortality. This is especially true for extensive hemispheric strokes or those located in the posterior cranial fossa. MBO, which occurs when most of the middle cerebral artery is involved, is associated with an up to 80% risk of death [61]. If conservative treatment fails, and the patient's condition deteriorates, this is an indication for urgent surgical decompression [62]. Therefore, COD is considered an important factor that may reduce the benefit/risk ratio of EVT [63].

The most common AIS sequelae affecting functional outcome in patients treated with EVT are set out in Table 4.

There are several limitations of our review: they primarily include the scarcity of evidence from prospective RCTs assessing presented associations. Secondly, there is limited data confirming that the modification of presented factors would affect tissue changes and clinical prognosis. The exact mechanisms of outlined interactions between presented factors and outcomes are mostly speculative, rather than based on solid evidence.

Conclusions

Post-MT treatment AIS sequelae can be noninvasively evaluated by neuroimaging studies. The most commonly assessed characteristics of brain tissue outcomes are: final volume of cerebral ischaemia, the extent of local oedema, and haemorrhagic complications. All of these show a strong association with functional outcome.

Data presented in our review emphasises the importance of identifying factors that influence these parameters. Potentially modifiable factors such as the choice of anaesthesia method, periprocedural systemic blood pressure, or blood glucose levels, are of particular importance. Further research on interventions modifying these parameters should be encouraged, as they may reduce the occurrence of unfavourable AIS tissue outcome that would improve prognosis.

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