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Review

Unraveling inflammation's role in cerebral aneurysm recanalization: A comprehensive systematic review

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Abstract. *Background:* Although significant progress has been made in identifying risk factors for cerebral aneurysms (CAs), the exact pathological mechanisms responsible for aneurysm recurrence remain poorly elucidated. Inflammation has increasingly been implicated in the progression and recurrence of aneurysms following endovascular coiling. This systematic review aims to evaluate the role of inflammatory factors in post-coiling recanalization across intracranial aneurysms based on literature from the last decade. *Methods:* The review was conducted in accordance with PRISMA 2020 guidelines. English-language studies published between 2015 and 2025 were retrieved using PubMed, SpringerLink, SagePub, and Google Scholar. Editorials, review articles, duplicate publications, and studies lacking a digital object identifier (DOI) were excluded. Articles were screened through a three-step process (title, abstract, and full-text review). *Results:* A total of 1,800 records were identified through database searches. After applying inclusion and exclusion criteria and conducting full-text analysis, seven eligible studies were included. The selected literature reported on inflammatory mediators such as tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β), matrix metalloproteinase (MMP-2) and MMP-9, macrophage subtypes, and complement system activation. These factors were associated with impaired thrombus stabilization, delayed endothelial repair, and increased likelihood of CA recanalization. *Conclusion:* Inflammatory responses are key contributors to aneurysm recurrence following coiling, acting through vessel wall degradation, extracellular matrix remodeling, and endothelial dysfunction. Macrophage infiltration, cytokine upregulation, MMP activation, and complement involvement are mechanistically linked to long-term instability of treated aneurysms. Future research should explore anti-inflammatory adjunct therapies, optimize biomaterial selection, and identify predictive inflammatory biomarkers to enhance the durability of endovascular aneurysm treatment. (www.actabiomedica.it)

Key words: cerebral aneurysm, coiling, recanalization, inflammation, cytokines, macrophage

Introduction

Cerebral aneurysm (CA) is an abnormal vascular bulging due to weakening of the arterial wall, arising from congenital or acquired etiologies. Its clinical presentation varies from asymptomatic cases to life-threatening rupture, making early identification and risk stratification crucial. Extensive research in clinical, genetic, and artificial intelligence fields has aimed to identify individuals at high risk of CA formation and rupture. Established clinical risk factors include a family history of CA, smoking, hypertension, and female sex. From a pathophysiological perspective, inflammation is recognized as a key contributor to

the formation, progression, and potential rupture of CA. The weakening of the arterial wall, endothelial dysfunction, and extracellular matrix (ECM) degradation are hallmark processes underlying aneurysm development (1,2). Despite advancements in understanding CA risk factors, the precise pathogenic pathways remain incompletely understood. Recent evidence has increasingly implicated inflammation in the pathogenesis of CA, with numerous experimental and animal studies highlighting its role in aneurysm formation and progression. Inflammatory processes have been associated with aneurysm development and rupture, with particular attention to the role of cytokines such as tumor necrosis factor-α (TNF-α). TNF-α is a central regulator of inflammation, influencing the behavior of key cell types involved in aneurysm pathology, including macrophages, endothelial cells, and smooth muscle cells. However, while studies have noted altered TNF-α levels in patients with CA, its direct role in aneurysm formation and rupture remains unclear (3-5). Endovascular treatment has emerged as a less invasive alternative to surgical clipping for managing CA, particularly for unruptured cases or patients unsuitable for neurosurgical intervention. Over the years, endovascular techniques have evolved to become the preferred treatment option in many cases. Coiling, the first widely adopted endovascular approach, remains the standard therapy for both ruptured and unruptured aneurysms, often performed alongside balloon-assisted techniques. However, despite technological advancements, recanalization—a major complication leading to aneurysm recurrence—remains a significant concern. Several risk factors for recanalization have been identified, including the initial occurrence of subarachnoid hemorrhage (SAH), aneurysm size, and posterior circulation location (6,7). Given the inconclusive findings regarding the specific inflammatory factors associated with recanalization after coiling, further investigation is needed. Understanding the relationship between inflammation and aneurysm recurrence could improve post-treatment monitoring and therapeutic strategies. Therefore, this systematic review aims to analyze the role of inflammatory factors in post-coiling recanalization rates of cerebral aneurysms.

Material and Methods

Protocol

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines to ensure transparency, reproducibility, and scientific rigor throughout the study process. The methodology was selected to enhance the consistency, precision, and reliability of the conclusions drawn from the literature. The study protocol was registered with PROSPERO (International Prospective Register of Systematic Reviews) on June 17, 2025, under the registration number CRD420251075493.

Criteria for eligibility

This review investigates the association between inflammatory markers and post-coiling recanalization of CA over the last decade. The objective was to synthesize relevant findings that could guide future treatment protocols. The inclusion criteria were as follows: (1) Studies published in English; and (2) Articles published between 2015 and 2025. The exclusion criteria included: (1) Editorials; (2) Papers lacking a digital object identifier (DOI); (3) Review articles previously published; and (4) Duplicate records across journals.

Search strategy

A systematic search was conducted using PubMed, SagePub, SpringerLink, and Google Scholar databases (Table 1). The search employed both MeSH and Boolean terms to capture all relevant studies. The complete search syntax was: *("Intracranial Aneurysm"[MeSH Terms] OR "intracranial aneurysm"[All Fields] OR "cerebral aneurysm"[All Fields] OR "brain aneurysm"[All Fields] OR "aneurysm"[All Fields]) AND ("Endovascular Procedures"[MeSH Terms] OR "endovascular procedure"[All Fields] OR "endovascular coiling"[All Fields] OR "endovascular embolization"[All Fields] OR "coiling"[All Fields]) AND ("Recanalization"[MeSH Terms] OR "recanalization"[All Fields] OR "aneurysm recurrence"[All Fields] OR "reopening"[All Fields] OR "reperfusion"[All Fields]) AND ("Inflammation"*

Table 1. Article Search Strategy

Database	Keywords	Hits
Pubmed	("Intracranial Aneurysm"[MeSH Terms] OR "intracranial aneurysm"[All Fields] OR "cerebral aneurysm"[All Fields] OR "brain aneurysm"[All Fields] OR "aneurysm"[All Fields]) AND ("Endovascular Procedures"[MeSH Terms] OR "endovascular procedure"[All Fields] OR "endovascular coiling"[All Fields] OR "endovascular embolization"[All Fields] OR "coiling"[All Fields]) AND ("Recanalization"[MeSH Terms] OR "recanalization"[All Fields] OR "aneurysm recurrence"[All Fields] OR "reopening"[All Fields] OR "reperfusion"[All Fields]) AND ("Inflammation"[MeSH Terms] OR "inflammation"[All Fields] OR "inflammatory response"[All Fields] OR "inflammatory mediators"[All Fields] OR "cytokines"[All Fields] OR "macrophages"[All Fields])	550
Springer Link	(((aneurysm) AND (coiling)) AND (recanalization)) AND (inflammation)	640
Sagepub	(((aneurysm) AND (coiling)) AND (recanalization)) AND (inflammation)	200
Google Scholar	(((aneurysm) AND (coiling)) AND (recanalization)) AND (inflammation)	410

[MeSH Terms] OR "inflammation"[All Fields] OR "inflammatory response"[All Fields] OR "inflammatory mediators"[All Fields] OR "cytokines"[All Fields] OR "macrophages"[All Fields]).

Data retrieval

Titles and abstracts were independently screened by multiple reviewers to determine eligibility according to predefined criteria. Studies that met all inclusion criteria and demonstrated relevance to the objective were selected for full-text review. The extracted data included article title, author names, year of publication, study region or aneurysm site, inflammatory markers studied, methodologies, and key outcomes. Articles failing to meet eligibility or lacking clinical relevance were excluded from further synthesis.

Quality assessment and data synthesis

Each eligible study was appraised for methodological quality using the Joanna Briggs Institute (JBI) critical appraisal checklist (Table 2). Multiple reviewers assessed each article, and disagreements were resolved by consensus. While all studies included met the minimum quality threshold, heterogeneity in biomarker assays and study design was noted. Results were qualitatively synthesized due to variation in experimental models and outcome measures.

Result and discussion

A total of 1,800 potentially relevant articles were initially retrieved through systematic searches of four electronic databases: PubMed, SpringerLink, SagePub, and Google Scholar. Following the removal of duplicates and a structured three-stage screening process (title review, abstract screening, and full-text evaluation), seven original research articles that directly met the inclusion criteria and addressed the central question of this review were selected for detailed analysis (Figure 1).

A comprehensive summary of the selected articles, including sample size, aneurysm location, inflammatory mediators studied, and principal conclusions, is presented in Table 3. This review included seven studies analyzing various aspects of inflammatory involvement in CA, including its general pathophysiological role in aneurysm formation and rupture and its specific contribution to post-coiling recanalization and aneurysm healing dynamics. Grüter et al. described the topographic accumulation of inflammatory mediators in aneurysm thrombi and neointima. Interestingly, their findings suggested that well-controlled inflammation reaction plays a crucial role in aneurysm healing after endovascular treatment, representing a context-specific reparative response rather than a purely destructive one (8). Wang et al highlighted the coexistence of inflammatory markers in recurrent aneurysms, such as CD68+ macrophages in aneurysm walls and granulation tissue, supporting the idea that

Table 2. Joanna Briggs Institute (JBI) *Critical appraisal of Study*

Parameters	Gruter (2023)	Wang (2021)	Lattanzi (2021)	Jin (2022)	Roa (2020)	Khasim (2020)	Brinjikji (2015)
1. Bias related to temporal precedence Is it clear in the study what is the "cause" and what is the "effect" (i.e., there is no confusion about which variable comes first)?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
2. Bias related to selection and allocation Was there a control group?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
3. Bias related to confounding factors Were participants included in any comparisons similar?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
4. Bias related to administration of intervention/exposure Were the participants included in any comparisons receiving similar treatment/care, other than the exposure or intervention of interest?	No	No	No	No	No	No	No
5. Bias related to assessment, detection, and measurement of the outcome							
Were there multiple measurements of the outcome, both pre and post the intervention/ exposure?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Were the outcomes of participants included in any comparisons measured in the same way?	No	No	No	No	No	No	No
Were outcomes measured in a reliable way?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
6. Bias related to participant retention Was follow-up complete and, if not, were differences between groups in terms of their follow-up adequately described and analyzed?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
7. **Statistical conclusion validity** Was appropriate statistical analysis used?	Yes	Yes	Yes	Yes	Yes	Yes	Yes

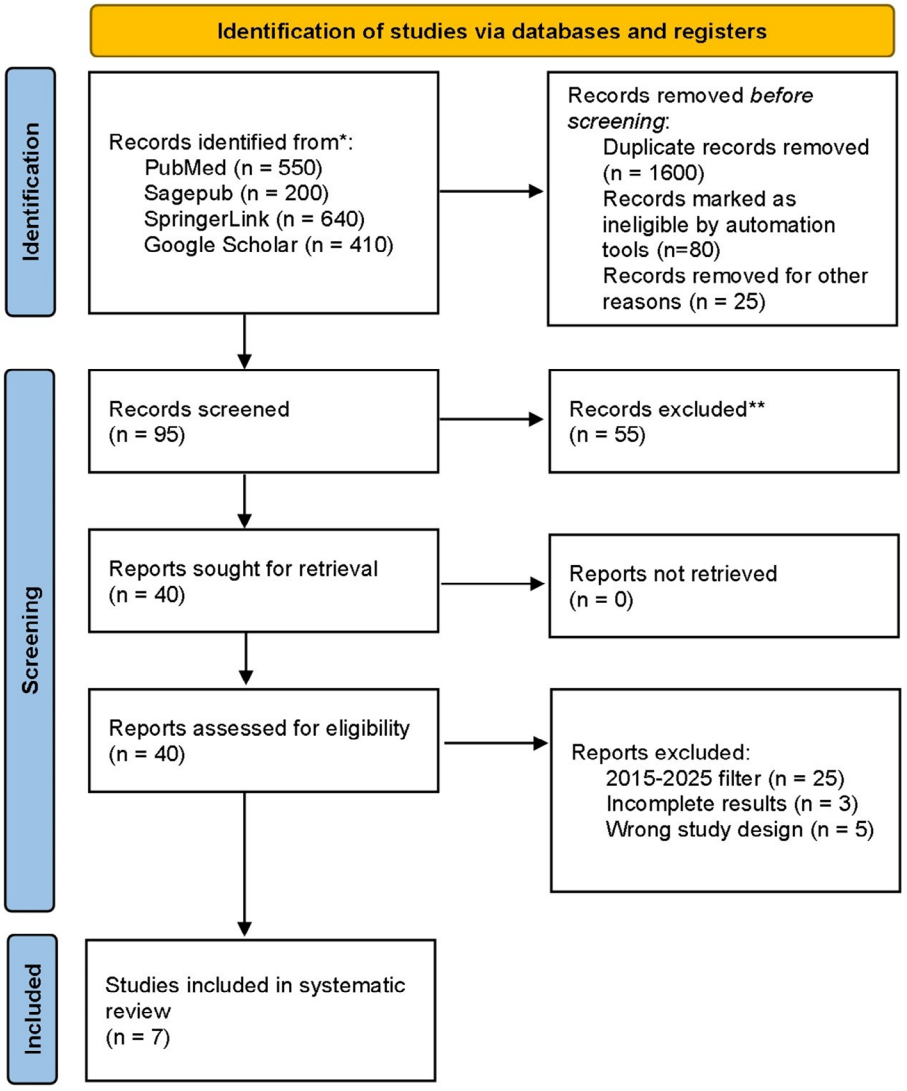

Figure 1. Article search flowchart.

chronic inflammation may contribute to recanalization (9). Khashim et al. specifically described macrophage polarization, with M1 macrophages being pro-inflammatory and M2 macrophages being pro-healing. This study proposed that M1 predominance was linked with poorer healing, while M2 response favored neointimal repair post-coiling (13). Brinjikji et al. provided indirect evidence by linking statin use with lower recurrence rates post-coiling, implicating anti-inflammatory modulation as a potential therapeutic strategy (14). On the other hand, Jin et al. and Roa et al. linked pro-inflammatory and cytotoxic immune responses with aneurysmal degeneration and rupture, suggesting context-dependent inflammatory pathways (11,12). Lattanzi et al. reported that elevated systemic inflammatory response index (SIRI) was associated with poor outcomes in ischemic stroke patients post-recanalization, indicating that peripheral inflammation may persist despite vascular reopening, though this was not specific to aneurysm pathology (10). The most commonly evaluated markers across the studies were TNF-α, IL-1β, matrix metalloproteinases

Table 3. The literature included in this study

Authors	Sample Size	Location or Type of Aneurysm	Inflammatory Factors Involved	Key Conclusion
Gruter et al., (8)	43	Saccular side wall aneurysms	Interleukin 6 (IL-6), matrix metalloproteinase (MMP- 2), MMP-9, TNF-α, Fibroblast growth factor-23 (FGF23), Vascular endothelial growth factor (VEGF)	A well-controlled inflammation reaction plays a crucial role in aneurysm healing after endovascular treatment.
Wang et al., (9)	9	Anterior communicating artery (ACOM), Posterior communicating artery (PCOM), Middle cerebral artery right (MCA R), Right & left carotid artery communicating segment	CD68+ antibody staining in the aneurysm wall; presence of granulation tissue	The coexistence of fresh thrombus, granulation tissue, and scar tissue is characteristic of recurrent aneurysms. Stable thrombus formation may be key to preventing aneurysm recurrence. Smooth muscle cell damage and inflammatory cell infiltration likely contribute to aneurysm recanalization.
Lattanzi et al., (10)	184	Not specified	Neutrophils, monocytes, inflammatory cytotoxic mediators; interactions between T cells, platelets, and endothelium; stress hormones (catecholamines and cortisol)	The systemic inflammatory response index (SIRI), reflecting the balance between innate and adaptive immunity, is an independent predictor of poor outcomes in acute ischemic stroke patients undergoing successful endovascular treatment. Patients with SIRI levels above 3.8 × 10^9/L had nearly double the risk of poor 3-month functional outcomes despite successful recanalization.
Jin et al., (11)	Not mentioned	Major arterial branch points of the Circle of Willis	TNF-α, IL-1β, IL-6, COX, MMP-9, NLRP3, and MCP-1	Pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6 are central mediators in the inflammatory cascade after aneurysm rupture. Their activation through pathways like TLR4/ NF-kB promotes immune cell infiltration, extracellular matrix degradation, and neuronal damage, which collectively compromise aneurysm stability and worsen clinical outcomes. Targeting these inflammatory pathways may provide therapeutic benefits in managing aneurysm SA.

Authors	Sample Size	Location or Type of Aneurysm	Inflammatory Factors Involved	Key Conclusion
Roa et al., (12)	13	Pericallosal, basilar artery (BA) tip, PCOM, ACOM, MCA	IL-17, IL-23, natural killer (NK) cells, VEGF, IL-6, TNF-α, MMP-9, Endothelin-1 CD8+CD161+ lymphocytes in cerebrospinal fluid, cells of the innate immune systems	The inflammatory factors contributing to aneurysm formation and progression include IL-17 and the IL-23/IL-17 axis, with CD8+CD161+ "Tc17" lymphocytes identified in the aneurysmal wall as significant sources of IL-17 and pro-inflammatory cytokines, suggesting their role in local vascular inflammation both before and after aneurysm rupture. Innate immune cells such as macrophages, neutrophils, and microglia infiltrate the aneurysmal wall and cerebrospinal fluid, releasing cytokines and proteases like MMP-9 that promote vascular damage and blood-brain barrier disruption. Elevated levels of VEGF, IL-6, TNF-α, and MMP-9 are associated with inflammatory responses and vasospasm after subarachnoid hemorrhage.
Khashim et al., (13)	23	Not specified	M1 and M2 macrophages	The polarization of macrophages from a pro- inflammatory M1 phenotype to an anti-inflammatory M2 phenotype correlates with improved healing of aneurysms after endovascular coil embolization.
Brinjikji et al., (14)	132	Not specified	MMPs	Statins are associated with lower rates of aneurysm recurrence following endovascular coiling of ruptured intracranial aneurysms.

(MMP-2 and MMP-9), and macrophage subtypes (M1/M2). However, methodological inconsistencies across studies—such as differences in immunohistochemistry protocols, patient cohort definitions, and outcome measurements—limited the ability to perform quantitative synthesis. Despite this, the qualitative evidence extracted from these studies supports the role of inflammation as a contributing factor to aneurysm recurrence after endovascular coiling. The role of inflammation in vascular pathology has been extensively studied, particularly in relation to aneurysm formation, progression, and rupture. Increasingly, post-coiling recanalization—a significant complication following endovascular treatment of intracranial aneurysms—has been linked to persistent inflammatory activity within the aneurysm wall. The infiltration of inflammatory cells, secretion of pro-inflammatory cytokines, and activation of matrix-degrading enzymes provide compelling evidence of biologically driven mechanisms underlying treatment failure. Although

coiling provides effective occlusion, long-term stability is often compromised by ongoing vascular remodeling and thrombus destabilization, as previously described in both experimental and clinical settings (15,16).

Macrophages are central mediators of this inflammatory response, infiltrating the aneurysm sac and releasing cytokines and proteolytic enzymes that weaken the vessel wall. Numerous studies, including those evaluated in this review, have observed that increased macrophage density is positively correlated with aneurysm recurrence. These cells produce MMPs that degrade elastin and collagen, vital components of the extracellular matrix (ECM) (17). Funakoshi et al. demonstrated that aneurysms with robust macrophage infiltration exhibited significantly higher recurrence rates post-coiling (18). TNF-α, a key pro-inflammatory cytokine, is frequently elevated in aneurysmal tissue and induces vascular endothelial dysfunction. It promotes leukocyte adhesion, smooth muscle cell apoptosis, and upregulates MMPs, notably MMP-9. Persistent TNF-α signaling following coiling can impair thrombus maturation and delay neointimal healing, increasing susceptibility to recanalization. Brinjikji et al. reported that TNF-α enhances MMP-9 expression, directly contributing to collagen IV degradation and aneurysm instability (14,19). Similarly, interleukin-1 beta (IL-1β) plays a pathogenic role by promoting inflammatory cell recruitment and further activating MMP pathways. Elevated IL-1β levels interfere with endothelial repair and neointimal coverage—both of which are essential for durable aneurysm occlusion. Grüter et al. demonstrated that IL-1β localized preferentially to regions of thrombus instability, supporting its role as a potential therapeutic target (8,20). MMP-2 and MMP-9 are particularly relevant to aneurysm biology, as they mediate the breakdown of the ECM, facilitating aneurysm recurrence post-treatment. These enzymes are secreted by macrophages, neutrophils, and vascular smooth muscle cells under inflammatory stimulation (21). Consistently elevated MMP-9 levels have been associated with poor occlusion durability. Experimental studies have shown that inhibition of MMP activity improves aneurysm healing outcomes and reduces recanalization risk (14). The complement cascade further compounds inflammatory injury in coiled aneurysms. Activation of components like C3 and C5b-9 induces endothelial injury, attracts leukocytes, and amplifies local inflammatory signaling. This contributes to thrombus destabilization and coil compaction, resulting in residual blood flow. Lei et al. and Morgan et al. highlighted complement-mediated inflammation as a contributor to post-coiling aneurysm instability (22,23). Chronic inflammation also disrupts endothelial regeneration and smooth muscle cell migration, two critical processes in aneurysm healing. TNF-α and IL-1β impair nitric oxide signaling and enhance leukocyte adhesion to the endothelial surface, limiting neointimal formation. Aneurysms with incomplete endothelial repair demonstrate a higher propensity for recurrence (24,25). Hemodynamic stress contributes to post-coiling recanalization by influencing inflammatory responses within the aneurysm sac. Regions of low wall shear stress promote leukocyte adhesion, complement activation, and MMP expression, exacerbating vessel wall degradation. Conversely, areas of high shear stress can induce endothelial damage, triggering an inflammatory cascade that undermines long-term aneurysm stability. Computational modeling studies have shown that inflammatory markers are predominantly localized in regions of disturbed flow within coiled aneurysms, supporting the hypothesis that hemodynamic forces modulate inflammation-driven recanalization (26). Given the multifactorial role of inflammation, targeted anti-inflammatory interventions are being investigated to improve the long-term efficacy of coil embolization. Experimental therapies—including TNF-α inhibitors, IL-1β antagonists, and MMP blockers—have demonstrated potential in reducing recurrence. Moreover, advanced embolic materials incorporating anti-inflammatory properties are under development. These innovations seek to promote stable thrombus organization, enhance neointimal coverage, and ultimately reduce recanalization risk. However, rigorous clinical validation remains necessary to determine their translational value (27,28). Future clinical strategies should focus on integrating inflammatory profiling into pre-treatment risk assessment, developing bioactive coils with anti-inflammatory properties, and designing adjunctive therapies targeting TNF-α, IL-1β, or MMP-9 in high-risk patients. Longitudinal trials assessing inflammatory biomarker trajectories and their

relationship with radiological outcomes are warranted to validate these approaches.

Conclusion

Inflammatory factors play a significant role in post-coiling recanalization of intracranial aneurysms by contributing to vessel wall degradation, delaying endothelial repair, and impairing stable thrombus organization. Key pathological mechanisms include macrophage infiltration, heightened TNF-α and IL-1β cytokine activity, matrix metalloproteinase overexpression, complement cascade activation, and the influence of hemodynamic stress. These elements collectively destabilize the aneurysm environment and compromise long-term treatment durability. Future research should prioritize the development of targeted anti-inflammatory therapies, innovation in coil materials with bioactive properties, and refined patient selection strategies guided by validated inflammatory biomarkers. A multidisciplinary approach integrating biological insights with advanced endovascular techniques holds promise for reducing recanalization rates and improving the long-term efficacy of aneurysm treatment.

Ethics Approval and Consent to Participate: According to national regulations, no ethical approval was required as this is merely a systematic review article. No written informed consent was obtained for publication.

Conflicts of Interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article".

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Abbreviations

CA: Cerebral aneurysm
ECM: Extracellular matrix
TNF-α: Tumor necrosis factor-α
SAH: Subarachnoid hemorrhage
PRISMA: Preferred reporting items for systematic review and meta-analysis
DOI: Digital object identifier
JBI: Joanna Briggs institute
IL-6: Interleukin 6
MMP: Matrix metalloproteinase
FGF23: Fibroblast growth factor-23
VEGF: Vascular endothelial growth factor
CD: Cluster of differentiation
HLA-DR: Human Leukocyte Antigen-DR isotype
ACOM: Anterior communicating artery
PCOM: Posterior communicating artery
MCAR: Middle cerebral artery right
SIRI: Systemic inflammatory response index
TXNIP: Thioredoxin-interacting protein
NLRP3: Nucleotide-binding domain, leucine-rich–containing family, pyrin domain–containing-3
IL-1β: Interleukin-1 beta
BA: Basilar artery
NK: Natural killer
M1 and M2: Macrophages
ECM: Extracellular matrix

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