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Meta-analysis of risk factors for restenosis after stent implantation in patients with ischemic cerebrovascular disease

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Abstract. – OBJECTIVE: The aim of this study was to investigate the risk factors for restenosis after stent implantation in patients with ischemic cerebrovascular disease (ICVD), and to provide a reference for potential measures to prevent ICVD.

MATERIALS AND METHODS: Relevant studies were identified by searching PubMed, ScienceDirect and Web of Science databases. Combined adjusted odds ratios and 95% confidence intervals were calculated.

RESULTS: Seven case-control studies were identified in the end. Diabetes mellitus and residual stenosis were the two main risk factors for restenosis (OR = 0.59, 95% CI: 0.39-0.91, $p = 0.01$; OR = 36.73, 95% CI: 19.72-70.02, $p < 0.001$). Gender, smoking, hypertension, dyslipidemia, and stent type were not significantly associated with restenosis (OR = 0.85, 95% CI: 0.53-1.38, $p = 0.52$; OR = 1.30, 95% CI: 0.91-1.86, $p = 0.15$; OR = 0.86, 95% CI: 0.16-4.66, $p = 0.86$; OR = 1.30, 95% CI: 0.58-2.91, $p = 0.53$; OR = 1.34, 95% CI: 0.72-2.48, $p = 0.35$).

CONCLUSIONS: The prevention of restenosis after stenting is particularly important for ICVD patients with diabetes or a high residual stenosis rate.

Key Words:
Meta-analysis, Risk factors, Stent implantation, Ischemic cerebrovascular disease.

Introduction

Ischemic cerebrovascular disease (ICVD) is a collective term for a large group of cerebrovascular diseases with focal neurological deficits due to insufficient perfusion of brain tissue. ICVD is a common clinical disease, including cerebral infarction, transient ischemic attack, cerebral thrombosis, and cerebral embolism[1]. The pathologic process is vascular wall lesion, blood composition change, and hemodynamic change. The main causes of ICVD are hypoxia, ischemia, and even necrosis of brain tissue caused by blood supply disorders to the brain. Continuous ischemia can lead to loss of nerve function and a higher risk of disability. A survey showed that the annual incidence of cerebrovascular accidents in Chinese adults is as high as 150-200/100,000, and ischemic cerebrovascular diseases are the most common (accounting for about 80%)[2]. Hemianesthesia, vision loss, and contralateral hemiplegia are common symptoms of ICVD. Cerebral artery thrombosis leads to intracranial artery occlusion, which is the main cause of ICVD. Cerebral tissue ischemia and hypoxia can cause neurological impairment in patients and even threaten their lives. The clinical recurrence rate and mortality of ICVD are high, which can seriously affect the life quality of patients, and its prevention and treatment is an important clinical task and challenge[3]. With the development of the economy, the demographic composition of human society is gradually aging, and the burden of cerebrovascular disease brought about by this is bound to increase further[4]. Existing data[5] indicate that serious adverse events such as disability and death caused by ischemic cerebrovascular diseases are becoming more and more serious.

One of the main treatments for ischemic cerebrovascular disease is stenting, which can rapidly open completely occluded or narrowed blood vessels through the application of puncture drugs, balloon dilation of blood vessels, and the corresponding stenting, thus saving patients' lives[6]. Stenting is gradually accepted by patients because of its minimal invasiveness, safety, high success rate, and fast recovery[7]. Stenting procedures are less invasive and can be done under local or general anesthesia. It has been reported[8] that the success rate of stenting is as high as 98.9%. The reason for this is that this type of surgery can directly act on the narrowed blood vessels, which can improve the blood flow in the area. At the same time, the ischemia of the brain tissue is relieved, and the acute and chronic damage caused by hypoxia and ischemia is reduced.

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Interventional stenting improves symptoms more rapidly than conservative treatment and reduces neurological damage. Successful stenting ensures that blood flow is unblocked and blood supply to the cranial tissue is restored. Furthermore, interventional stenting has been shown[9] to improve neurological function in a sustained manner. The relevant literature points out that the brain has the ability to reshape, especially after nerve damage, to constantly repair. The application of interventional stenting can improve the blood flow of stenotic vessels, which makes the remodeling of the injured cranial brain tissues obtain favorable conditions. The incidence of secondary neuronal injury is also reduced, which in turn accelerates the rate of neovascularization at the site of injury. Although cerebrovascular stenting is a minimally invasive procedure, this type of surgical treatment may cause multiple complications. Indeed, there are risks of restenosis, thrombosis, or cerebral hemorrhage after stenting, especially postoperative restenosis, the incidence of which is at a high level, which is a key clinical problem to be solved at present[10]. In-stent restenosis is an inflammatory process after arterial stenting due to factors such as surgical injury and long-term stimulation of the vessel wall by the stent. During this period, the local active factors and inflammatory substances in the blood vessels are abnormal, prompting the abnormal proliferation of vascular smooth muscle cells, thus inducing the abnormal proliferation of vascular endothelium, and ultimately resulting in localized vascular restenosis. Understanding the factors affecting restenosis after stenting for patients with ICVD is an important guide to its prediction and prevention[11].

The vast majority of studies in patients with ICVD have focused on risk factors for ischemic cerebrovascular disease and treatment modalities and outcomes, and no studies have focused on a meta-analysis of risk factors for restenosis after stent implantation in patients with ICVD[12]. Based on the above background, this study selected the recently published risk factors for restenosis after stent implantation in patients with ischemic cerebrovascular disease for meta-analysis, with a view to providing potential preventive measures and better treatment options for patients with this disease.

Materials and Methods

Search Strategy

All literature was obtained by searching PubMed, ScienceDirect, and Web of Science databases. The following keywords were used during the search: risk factors; risk; ischemic cerebrovascular disease; cerebral infarction; cerebral embolism; cerebral thrombosis; stent implantation and restenosis. Literature was searched from the time the database was created to July 2023. A manual search was also performed by reading the relevant published literature.

Inclusion and Exclusion Criteria for Literature

Literature inclusion criteria: (1) All the literature were randomized controlled trials or cohort studies related to stent implantation in patients with ICVD published in recent years without limiting the use of blinding; (2) Complete clinical information was available; (3) All the literature included studies of restenosis in patients after stent implantation.

Literature exclusion criteria: (1) Repeated publications and repeated searches; (2) non-English publications, dissertations, basic categories, reviews, abstracts, case reports and conference papers were excluded; (3) Literature with non-randomized controls or unclear observational indexes; (4) Literature with irrational trial designs or data that could not be extracted.

Literature Screening and Data Extraction

Literature search, screening, and data extraction were conducted independently by 2 professional evaluators according to literature inclusion and exclusion criteria. The evaluators first removed literature that was clearly inconsistent with the inclusion and exclusion criteria by reading the titles and abstracts. The initially screened literature was subjected to full-text search and evaluation to screen the literature further. The obtained literature was then cross-checked. In case of disagreement, consultation and discussion were carried out, or expert advice was sought for decision. The content extracted mainly included titles, authors, year of publication, type of study, and observational indicators.

Literature Quality Evaluation

Risk of bias analysis of the included literature was performed independently by two researchers using the Newcastle-Ottawa Scale (NOS), including nine entries on study subject selection, component comparability and outcome measures. The evaluation criteria were low-quality literature (1-3 points), medium-quality literature (4-6 points), and high-quality literature

(7-9 points). When subjective selection bias occurred between two researchers during the screening process, it was resolved by discussion or adjudicated by a third researcher.

Statistical Analysis

Meta-analysis was performed using Review Manager 5.4 software (RevMan 5.4, Nordic Cochrane Centre, Cochrane, Copenhagen, Denmark). The odds ratio was the effect indicator. Corresponding 95% confidence intervals (CI) were calculated and represented by forest plots. I^2 values represented the presence or absence of heterogeneity among studies. $p > 0.1$ and $I^2 < 50\%$ indicated that the included studies were statistically less heterogeneous, and a fixed-effects model was chosen; $p < 0.1$ and $I^2 > 50\%$ indicated that the included studies were statistically more heterogeneous, and a random-effects model was chosen. At this point, the causes of heterogeneity were analyzed, and subgroup analysis and sensitivity analysis were performed on factors that might trigger heterogeneity to exclude literature with higher sensitivity. Meanwhile, descriptive analysis was performed for those that could not be analyzed by meta-analysis. This systematic review and meta-analysis followed PRISMA guidelines and registered on PROSPERO (CRD42023466103).

Results

Literature Screening Process and Results

A total of 231 publications were obtained, all of which are publicly available. By removing 93 duplicate publications or duplicate detections, 138 documents were obtained; 71 case reports and abstracts were removed, and 67 publications were obtained. After reading the full text, 60 papers were not relevant, and 7 literatures[11,13-18] were finally included. The process of obtaining literature is shown in Figure 1.

Data Extraction and Quality Evaluation of the Included Literature

A total of seven papers[11,13-18] were included in the study, all of which were cohort studies. There was a total of 1,564 patients, including 194 patients with restenosis after stent placement and 1,370 patients without restenosis. The basic information of the included literature is shown in Table I.

Assessment of the Risk of Bias

Figure 2 indicates the results of the risk of bias assessment for the seven included studies[11,13-18]. Three studies[14,16,17] were assessed as having a high risk of bias in at least one area. However, all seven studies[11,13-18] had a low risk of bias in the outcome assessment domain.

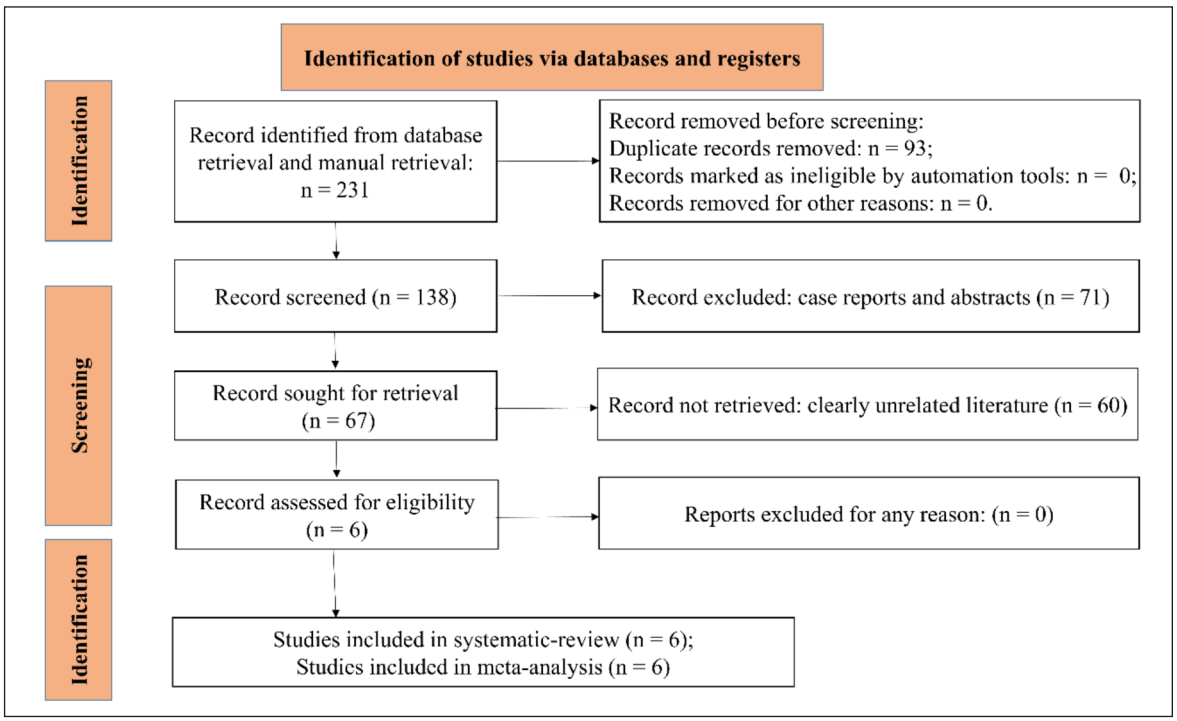

Figure 1. The process of literature search.

Table I. Basic information of the included literature.

Author	Year	Country	Study design	Restenosis	Total samples	Age	NOS score
Liu et al[13]	2023	China	Case-control	28	296	62 ± 6	8
Kuwabara et al[14]	2019	Japan	Case-control	15	293	73.9 ± 6.7	6
Song et al[15]	2022	China	Case-control	7	39	49.0-65.0	7
Zhang et al[16]	2021	China	Case-control	62	295	59.22 ± 9.11	8
Lai et al[17]	2013	China	Case-control	15	40	48.0 ± 11.7	5
Megaly et al[11]	2021	US	Case-control	25	148	72.8 ± 9.3	9
Wasser et al[18]	2012	Germany	Case-control	12	203	67.8 ± 6.6	4

Newcastle-Ottawa Scale (NOS).

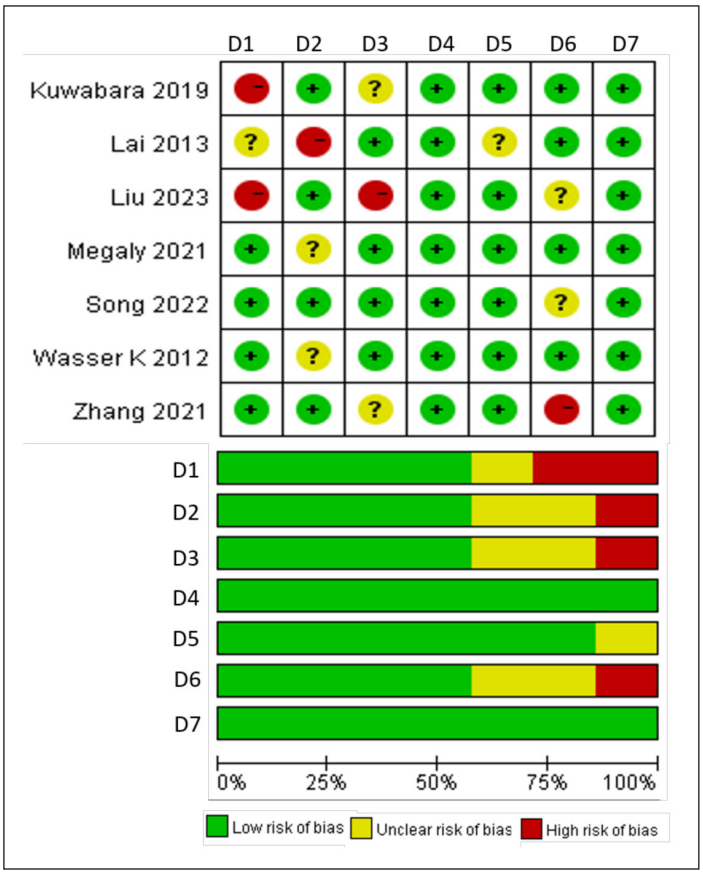

Figure 2. Bias risk assessment results from 7 included studies. D1: Random sequence generation (selection bias); D2: Allocation concealment (selection bias); D3: Blinding of participants and personnel (performance bias); D4: Blinding of outcome assessment (detection bias); D5: Incomplete outcome data (attrition bias); D6: Selective reporting (reporting bias); D7: Other bias.

Meta-Analysis of Risk Factors for Restenosis

At least 2 independent studies[13,15] mentioned 7 risk factors, including sex, diabetes, hypertension, dyslipidemia, smoking, and stent type. Therefore, these 7 factors were analyzed in this meta-analysis. The results are shown in Figure 3. Six studies[11,13-15,17,18] explored whether gender was a potential risk factor for restenosis (Figure 3A). Our results showed $I^2 = 29\% < 50\%$, no heterogeneity among the literature, and fixed-effect model analysis was considered. Forest plot results showed that the occurrence of restenosis after stent placement was not associated with gender (OR = 0.85, 95% CI: 0.53-1.38, $p = 0.52$).

Six included studies[11,13-15,17,18] explored whether diabetes was a potential risk factor for restenosis (Figure 3B). Our results showed $I^2 = 7\% < 50\%$, with no heterogeneity in the literature, considering a fixed-effect model analysis. Forest plot results showed that the occurrence of restenosis after stent placement was not associated with diabetes mellitus (OR = 0.59, 95% CI: 0.39-0.91, $p = 0.01$).

All included studies[11,13-18] explored whether smoking was a potential risk factor for restenosis

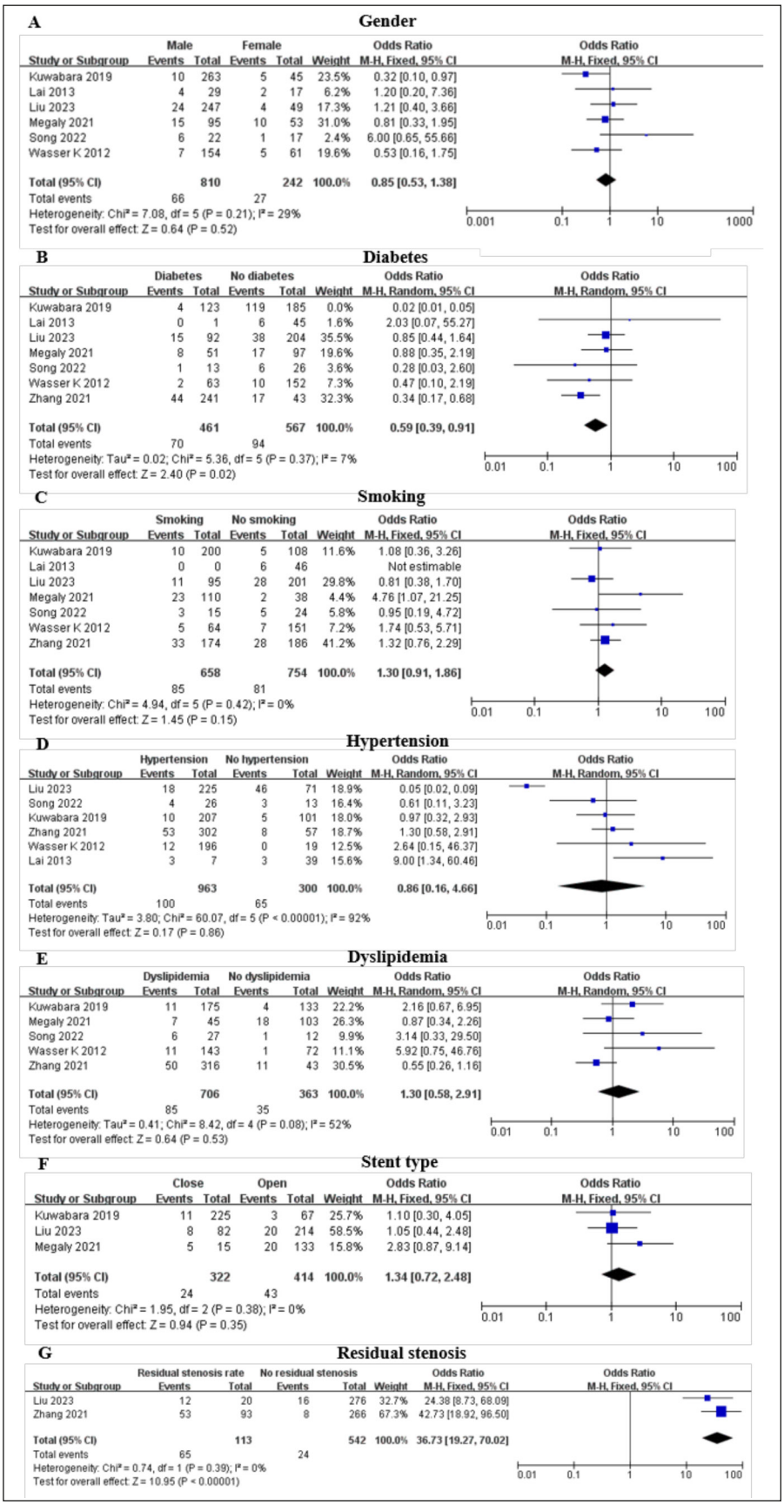

Figure 3. Odds ratio for restenosis with different factors. **A**, Diabetes mellitus; (**B**) residual stenosis; (**C**) gender; (**D**) smoking; (**E**) hypertension; (**F**) dyslipidemia; (**G**) stent type.

(Figure 3C). Our results showed $I^2 = 0 < 50\%$, and no heterogeneity in the literature, considering a fixed-effect model analysis. Forest plot results showed that the occurrence of restenosis after stent placement was not associated with smoking (OR = 1.30, 95% CI: 0.91-1.86, $p = 0.15$).

Six studies[13-18] explored whether hypertension was a potential risk factor for restenosis (Figure 3D). Our results showed $I^2 = 92\% > 50\%$, indicating heterogeneity among the literature, and a random-effects model was chosen for analysis. Forest plot results showed that the occurrence of restenosis after stent placement was not associated with the presence of hypertension (OR = 0.86, 95% CI: 0.16-4.66, $p = 0.86$).

Five studies[11,14-16,18] explored whether dyslipidemia was a potential risk factor for restenosis (Figure 3E). Our results showed $I^2 = 52\% > 50\%$, indicating heterogeneity among the literature, and a random-effects model was chosen for analysis. Forest plot results showed that the occurrence of restenosis after stent placement was not associated with dyslipidemia (OR = 1.30, 95% CI: 0.58-2.91, $p = 0.53$).

Three studies[11,13,14] explored whether stent type was a potential risk factor for restenosis (Figure 3F). Our results showed $I^2 = 0 < 50\%$, indicating no heterogeneity among the literature, and fixed-effects model analysis was considered. Forest plot results showed that the occurrence of restenosis after stent placement was independent of stent type (OR = 1.34, 95% CI: 0.72-2.48, $p = 0.35$).

Two studies[11,14] explored whether residual stenosis is a potential risk factor for restenosis (Figure 3G). Our results showed $I^2 = 0 < 50\%$, indicating no heterogeneity among the literature, and fixed-effects model analysis was considered. Forest plot results showed that the occurrence of restenosis after stent placement was independent of stent type (OR = 36.73, 95% CI: 19.72-70.02, $p < 0.001$).

Publication Bias Analysis

The funnel plot showed that most of the studies were distributed at the top, and the risk of publication bias in the included studies was low (Figure 4).

Discussion

ICVD is a phenomenon of brain tissue damage triggered by insufficient cerebral blood supply, which is mostly related to vascular wall lesions, local hemodynamic disorders and other causes. Statistical data show that atherosclerotic stenosis of intracranial and extracranial arteries is a key factor in the development of ICVD, accounting for more than 9/10 of cases. The causes of cerebral arterial stenosis are related to atherosclerosis in the arteries. For this reason, it is necessary to actively adopt the method of unblocking the narrowed arteries for ICVD patients. Cerebrovascular stenting is currently one of the most important means of stroke treatment and prevention. Surgery can remove the stenosis or even the occlusion of the blood vessels and restore cerebral blood perfusion and brain function. With the development of technology, the safety and efficacy of cerebrovascular stent implantation have been improved. However, there are still some patients with restenosis and occlusion in the stent after cerebrovascular stenting, which affects the prognosis of the patients. The exact mechanism of in-stent restenosis is unknown. Some researchers[19] suggest that it may be related

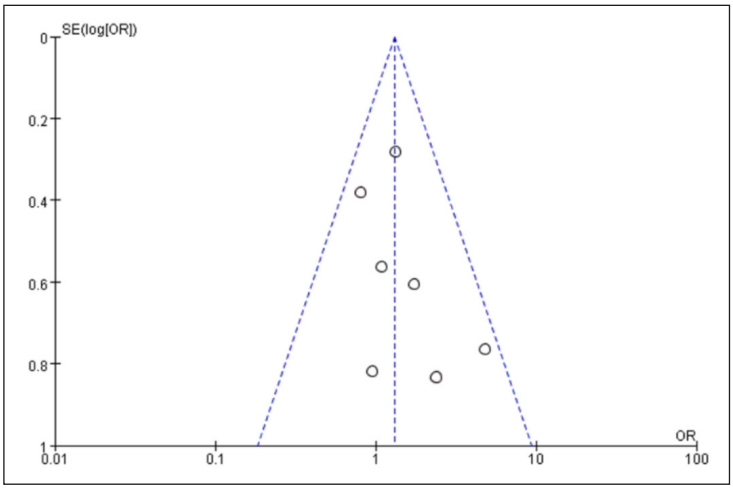

Figure 4. Funnel plot of restenosis risk factors.

to inflammatory mediators, cytokines, adhesion molecules, blood glucose and metabolites. Since the endothelial tissue is damaged to different degrees by surgical operation, blood plates were activated and attached locally to the stent, which promoted the synthesis of local inflammatory factors. This phenomenon aggravated the inflammatory response, multiplied smooth muscle cells, and resulted in the growth of the endovascular membrane, which led to in-stent restenosis. Therefore, the prevention of restenosis after treatment is of utmost importance. Focusing on the factors affecting restenosis events after stenting in patients with ICVD is an essential guide for its prediction and prevention. The current meta-analysis showed that the occurrence of restenosis after stenting in ICVD patients was not related to gender but was associated with the presence of diabetes mellitus. Diabetes has been shown[18] to be a strong predictor of restenosis after intracranial and coronary stenting in many early studies. There is growing evidence[20] that diabetic patients have a higher incidence of restenosis after coronary and intracranial stenting. Undoubtedly, the pathogenesis of restenosis after stent placement is characterized by intimal hyperplasia, which can be promoted by diabetes mellitus after coronary intervention. Diabetic patients have higher levels of insulin secretion, which can play a role in promoting the proliferation of vascular endothelial cells, resulting in a large amount of lipid deposition in endothelial cells and affecting endothelial function. Abnormal endothelial cells can produce procoagulant factors, which increase the degree of inflammation of endothelial cells and promote intimal hyperplasia, so restenosis is more likely to occur after surgery. Previous studies[21] have suggested that diabetes mellitus is a risk factor for the recurrence of carotid stenosis after carotid endarterectomy, which is consistent with our results. In diabetic patients, elevated blood glucose has been shown[22] to be associated with elevated levels of serum C-reactive protein and inflammatory cells, thus aggravating the inflammatory response at the stent implantation site and leading to in-stent restenosis.

Some studies[23] have shown that smoking or the presence of a history of smoking is an independent predictor of severe restenosis after arterial stenting. Smoking cessation can improve lipid, blood glucose, and blood pressure abnormalities, reduce oxidative stress and inflammation, and improve platelet aggregation, thereby reducing the risk of adverse events after stenting. Messner and Bernhard[24] revealed that cessation of smoking for only two weeks can reduce the series of oxidative stress induced by smoking, decrease the level of inflammatory factors in the body, and thus reverse vascular atherosclerosis to a certain extent in patients. However, some studies[25] have suggested that smoking may influence the occurrence of postoperative restenosis but is not an independent risk factor, and the results of the present study are consistent with this view, which may be related to the sample size of the study. In addition, this study also evaluated whether hypertension and dyslipidemia were risk factors. The results of the meta-analysis were not sufficient to demonstrate that the occurrence of restenosis after stenting is associated with hypertension and dyslipidemia.

Previous studies[26] have revealed an increased chance of restenosis in patients with closed stents compared to those with open stents. This is mainly due to the fact that open brackets have less physical metal in their composition compared to closed brackets. Less metal in an open stent means less contact with the carotid lumen. This results in less stimulation of the lumen, which may lead to a reduced response of the vascular system and a lower chance of endothelial hyperintensities. However, the results of the current study do not indicate a significant correlation between the occurrence of restenosis after stent placement and the stent type[27]. This may be related to the fact that only 3 of the included studies explored whether stent type is a risk. Of course, further studies are still needed to confirm this result. In addition, previous studies[28,29] on risk factors for developing restenosis after stent placement have reported that the higher the residual stenosis ratio after initial stent surgery, the more likely restenosis is to develop. In fact, previous studies[16] on risk factors for developing restenosis after stent placement have implied that the higher the residual stenosis ratio after initial stent placement, the more likely restenosis is to develop. For every 1% increase in residual stenosis, the risk of restenosis increased by 5%. This may be because a higher residual stenosis ratio is more likely to cause hemodynamic abnormalities, leading to abnormal platelet aggregation and restenosis.

In summary, this study identified only diabetes and residual stenosis as actual risk factors for restenosis after stent implantation. We believe that identifying these risk factors can significantly modify the prognosis after stent implantation. In addition, healthcare providers can remind patients of the importance of these interventions and provide value-added care through these findings. We also propose that in future studies, a larger

sample be included to identify additional risk factors and to assess the effectiveness of interventions to reduce the incidence and severity of postoperative restenosis. It must be recognized that the current meta-analysis also has some limitations: (1) Some special factors were only confirmed in individual studies, which may lead to bias problems and also mean that these factors could not be included in the final meta-analysis. (2) There was a large difference in sample size between the included studies, which may have had an impact on the results of the analysis; (3) The gray literature was not searched, and there was a possibility of publication bias; (4) The included studies were all example-control studies, and there were fewer cohort studies, and larger sample sizes and higher-quality cohort studies are still needed to provide more evidence. In future studies, we will collect more high-quality data and include more cohort studies to analyze and summarize the risk factors of restenosis after stenting more accurately. This will provide an early warning and evidence base for the prevention and intervention of postoperative restenosis so that clinical staff can identify the relevant risk factors and intervene at an early stage.

Conclusions

In conclusion, this meta-analysis found that the occurrence of restenosis after stenting in patients with ischemic cerebrovascular disease was associated with diabetes mellitus and residual stenosis and was not significantly associated with gender, smoking, hypertension, dyslipidemia, and stent type. It is still necessary to collect high-quality original studies on the influencing factors of restenosis after stenting to explore other influencing factors further so that the occurrence of restenosis can be better prevented and delayed in clinical practice.

Data Availability

The datasets generated during and/or analyzed during the current study are available in the manuscript.

Ethics Approval and Informed Consent

Not applicable.

Conflict of Interest

The authors declare that they have no conflict of interest.

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None.

Authors' Contributions

Zeyu Peng conceived the structure of manuscript. Yinghui Ji and Yu Li did the experiments and made the figures. Fei Wang reviewed and edited the manuscript. All authors read and approved the final manuscript.

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