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Latest Advances in Research on Spinal Cord Ischemia after Endovascular Repair of Type B Aortic Dissection

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Abstract

Stanford type B aortic dissection (TBAD) is a severe aortic emergency, with a sudden onset and rapid progression, which is a major threat to patient survival. Over the past decades, thoracic endovascular aortic repair (TEVAR) has become the main treatment for TBAD, because there were continuous improvements in endovascular devices, and the clinical decision-making has evolved, resulting in significant benefits both in short and long term survival. Even though there are these gains, spinal cord ischemia (SCI) after TEVAR—though it's rare—still a dreaded complication, which often causes permanent neurological deficits, making functional recovery and long-term quality of life significantly worse. Recent innovations in imaging, intraoperative neurophysiologic monitoring, and perioperative organ-sparing measures have been instrumental in fostering a better understanding of SCI in the context of TBAD. From a mechanistic viewpoint, there are three main factors that interact to either initiate or aggravate the spinal cord hypoperfusion. These factors include anatomical variations, technical variables that arise during the procedure, and patient-specific comorbidities. For diagnosis, combing a thorough neurologic examination with advanced imaging techniques (like diffusion-weighted MRI) and electrophysiologic studies (such as somatosensory and motor evoked potentials) has made detection more sensitive and timely. Management approaches encompass pharmacotherapy, surgical revascularization, and structured rehabilitation, each having its own rationale and patient selection criteria. Prevention is based on a rigorous preoperative risk evaluation, precise stent-graft sizing and positioning, and close postoperative hemodynamics and neurologic surveillance. This review has a distinctive feature, that it systematically categorizes the level of evidence supporting each recommendation, clearly distin-

guishing data from TBAD and TEVAR populations from those derived from thoracoabdominal aneurysm repairs, open surgery, or spinal injuries of non-aortic origin. Moreover, the review gives explicit time-based definitions (like immediate vs. delayed) and outcome-based classifications (transient vs. permanent) for SCI. It redefines the diagnostic imaging algorithm and emergency response pathway, and provides a balanced assessment of the predictive utility of various risk factors and clinical signs. By combing these up-to-date findings, the review provides clinicians with practical, evidence-based guidance to personalize therapy, reduce the frequency of SCI, and improve patient outcomes.

Keywords

B-Type Aortic Dissection, Thoracic Endovascular Aortic Repair, Spinal Cord Ischemia, Treatment, Prevention

1. Introduction

1.1. Research Background

TBAD is a vital cardiovascular emergency. It starts with a tear in the innermost layer of the aorta, enabling blood to flow into the middle layer. This results in a false lumen and divides the vessel wall into two parts [1]. This condition is usually marked by a sudden onset and rapid progression, with patients usually experiencing sudden, severe chest or back pain. Without timely intervention, mortality is extremely high [2]. In recent years, with the continuous development of Endovascular Aneurysm Repair (EVAR/TEVAR) techniques, their application in the treatment of TBAD has become more widespread. TEVAR restores the true lumen perfusion, and reduces the risk of dissection rupture by using covered stent grafts to seal the aortic intimal tear [2] [3]. But even though TEVAR has significantly improved survival rates, Spinal Cord Ischemia (SCI) is a serious postoperative complication that has a big impact on surgical outcomes and patient prognosis. Although SCI has a relatively low incidence, it might cause irreversible neurological deficits like paraplegia and sensory loss, which severely affects patients; quality of life [4]. According to the 2026 ESVS guidelines, protocolized SCI prevention strategies and staged repair can effectively reduce this risk [2]. So, a whole investigation is need to understand the pathogenesis, diagnostic approaches, and therapeutic strategies of SCI following TEVAR for TBAD.

1.2. Problem Statement

Endovascular repair has made a lot of progress in TBAD management, but the pathogenesis of postoperative spinal cord ischemia is still not clear, and standardized treatment regimens are not yet defined [5]. Previous studies show that spinal cord ischemia is linked to many factors, like anatomical variations, surgical techniques, and underlying comorbidities [6]. But because the spinal cord blood sup-

ply system is complicated and individuals vary, precisely predicting SCI risk is a big challenge. Moreover, although a variety of treatment modalities—like pharmacological therapy, surgery, and rehabilitation—have been explored, their specific indications, efficacy evaluation, and long-term prognosis require further validation [7]. So, strengthening research on SCI following endovascular repair for TBAD, especially by deeply exploring its pathogenesis and innovation in therapeutic strategies, is an important topic in the cardiovascular surgical field.

1.3. Research Objectives

The objectives of this study are to thoroughly review the latest advancements in the field of endovascular repair for tuberculosis (TBAD). The study emphasizes the innovations and breakthroughs that have emerged in the pathogenesis, diagnostic approaches, and therapeutic strategies. This work will summarize existing research gaps and propose future research directions based on recent findings. In the treatment domain, this study will focus a lot on the combination of multiple modalities, like pharmacological therapy, surgical intervention, and rehabilitation. It will also evaluate their clinical results to offer a more comprehensive and accurate reference for clinical practice. In this study, we want to provide theoretical support to improve the safety and efficacy of endovascular repair in TBAD, and build a solid foundation for better patient prognosis.

2. Literature Review

2.1. Theoretical Basis

The spinal cord has a unique blood supply characteristic. It is mainly supplied by the anterior and posterior spinal arteries, which distribute different segments of the cord: the anterior spinal artery provides the anterior two-thirds, and the posterior spinal artery provides the posterior third. As a supplementary vascular source, radicular arteries interconnect with anterior and posterior spinal arteries via branches like intercostal and lumbar arteries, forming a complex vascular network [4]. Covered stent grafts may cover or occlude intercostal and lumbar arteries while excluding the aortic intimal tear, thereby compromising spinal cord perfusion. Segmental arteries of the descending aorta, particularly those at the T8 - L2 level where the artery of Adamkiewicz arises, serve as the principal supply to the anterior spinal artery. Their occlusion may result in spinal cord ischemia. However, recent evidence suggests that extensive coverage of intercostal arteries alone is not an independent risk factor for symptomatic SCI. Instead, the primary mechanism entails compromise of at least two independent vascular territories—such as the left subclavian, intercostal or lumbar, and internal iliac arteries—coupled with prolonged intraoperative hypotension [8]. It should be noted that the aforementioned anatomical knowledge is derived primarily from basic anatomy and open surgical experience, rather than from TBAD-specific findings. Such changes in the anatomical relationships not only explain the potential mechanisms by which endovascular repair may affect spinal cord blood supply but also provide a

theoretical basis for optimizing stent selection and surgical techniques in clinical practice [3].

2.2. Review of Current Literature

In recent years, domestic and international scholars have conducted extensive research on spinal cord ischemia following endovascular repair for TBAD. Early studies primarily focused on exploring its pathogenesis; for example, reports have demonstrated the correlation between spinal cord ischemia and the coverage of important branch vessels by covered stent grafts, and have proposed the concept of the "high-risk zone for spinal cord ischemia [5]". With advancements in imaging technology, diagnostic methods have undergone significant improvements. Although traditional imaging techniques such as Digital Subtraction Angiography (DSA) and Computed Tomography Angiography (CTA) have been widely applied for postoperative monitoring, their invasive nature or high radiation exposure has gradually been supplemented by newer imaging technologies [9]. For instance, Diffusion-Weighted Imaging (DWI) and functional Magnetic Resonance Imaging (fMRI) have shown unique advantages in early detection of spinal cord ischemia and assessment of spinal cord function [10]. The 2026 ESVS guidelines clearly recommend protocolized SCI prevention strategies and emphasize that staged repair can significantly reduce the incidence of SCI. In treatment, strategies have progressed from initial conservative approaches to comprehensive ones. Pharmacological therapy, primarily vasodilators, anticoagulants, and neuroprotective agents, aims to enhance spinal cord perfusion and mitigate reperfusion injury [2]. Additionally, the application of interventional methods such as lumbar CSF drainage and vascular bypass surgery has provided new directions for SCI treatment. These research findings have not only enriched our understanding of this complication but also offered important references for clinical practice [11]. Nevertheless, it should be recognized that the evidence supporting many of the above recommendations—particularly for cerebrospinal fluid drainage, staged repair, and electrophysiological monitoring—is derived largely from Thoracoabdominal Aortic Aneurysm (TAAA) repair or open aortic surgery. Direct, high-quality evidence specific to TBAD-TEVAR remains limited.

2.3. Deficiencies in Current Evidence

Despite advances in the literature on SCI following endovascular repair for TBAD, several critical gaps remain. Current risk-assessment models for predicting SCI are predominantly based on baseline patient conditions and lesion characteristics, with limited consideration of individual anatomical variations, resulting in suboptimal predictive accuracy [12]. In addition, while existing studies have predominantly focused on evaluating the efficacy of single treatment modalities, comprehensive treatment strategies tailored to the specific conditions of different patients remain insufficient. Moreover, research on the long-term prognosis and rehabilitation outcomes of spinal cord ischemia is relatively limited, which is difficult to

meet clinical demands. More critically, the current literature generally lacks a systematic stratification of evidence sources for each recommendation. As a result, direct evidence and indirect inferences are often used interchangeably in clinical decision-making. Therefore, this study will integrate multidimensional data to construct a more precise risk-assessment system and explore individualized prevention and treatment strategies, aiming to provide new entry points and solutions for improving clinical diagnostic and treatment levels.

2.4. Definition and Classification of SCI Outcome Measures

To accurately interpret the risk factors, monitoring methods, and treatment effects reported in the literature cited in this review, it is necessary to clarify the classification criteria for SCI outcomes. This review categorizes SCI outcomes along two dimensions [11].

Time dimension:

1) Immediate SCI: occurs intraoperatively or within 24 hours after emergence from anesthesia, and is often directly related to intraoperative hemodynamic fluctuations, stent coverage, or embolic events.

2) Delayed SCI: occurs more than 24 hours postoperatively (typically from several hours to several days after surgery), and may be associated with spinal cord edema, delayed hypoperfusion, or thrombosis. These two types differ in their underlying mechanisms, required monitoring frequency, and optimal timing for intervention.

Outcome dimension:

1) Transient SCI: neurological deficits resolve completely or substantially within 24 to 48 hours, leaving no significant functional impairment.

2) Permanent SCI: neurological deficits persist beyond 72 hours or are irreversible, resulting in varying degrees of paraplegia, sensory loss, or sphincter dysfunction.

When interpreting the incidence and efficacy data cited in this review, it should be noted that the definitions of transient and permanent SCI are not uniform across studies. Most studies did not report the proportions of these two types separately, which directly affects the assessment of risk factors and the evaluation of treatment effectiveness.

3. Pathogenetic Mechanisms

3.1. Anatomical Factors

3.1.1. Spinal Cord Perfusion Characteristics

In aortic dissection, the formation of a false lumen due to intimal tear can compress the true lumen or directly occlude the ostia of the radicular arteries, thereby severely compromising spinal cord blood supply [11]. In addition, the spinal cord exhibits significant segmental heterogeneity in blood supply; the thoracic cord (T4-L1) lacks rich collateral circulation and is therefore considered the "watershed zone," rendering it particularly vulnerable to ischemia that can occur after endo-

vascular aortic repair [13]. The concept of the "watershed zone" is derived from basic anatomy and open surgical experience, and is not specific to TBAD. Therefore, the anatomical characteristics of the spinal cord blood supply system in aortic dissection determine its vulnerability and constitute an important anatomical basis for postoperative spinal cord ischemia.

3.1.2. Aortic Blood Supply to the Spinal Cord

The aorta, being the bodys largest artery, has a strong correlation between its blood supply areas and spinal cord perfusion. The intercostal and lumbar arteries originate from the thoracic aorta, which are the primary sources for the radicular arteries. These radicular arteries connect to the anterior and posterior spinal arteries through these branch vessels, providing continuous blood supply to the spinal cord [14]. However, while TEVAR effectively seals the aortic intimal tear via covered stent-graft deployment, it may inadvertently cover or partially occlude the intercostal and lumbar arteries, thereby compromising spinal cord perfusion. This is very relevant in the descending thoracic aorta, especially at T8 - L2 level, because stent-graft coverage might block critical radicular arteries and cause spinal cord ischemia [15]. These anatomical variations not only explain the mechanisms by which endovascular repair might damage spinal cord perfusion, but also give a theoretical basis to optimize stent-graft selection and improve procedural strategies in clinical practice.

3.2. Procedural Factors

3.2.1. Vascular Effects of Covered Stent-Grafts

Stent-graft deployment is effective in sealing aortic intimal tear, but it might also occlude or stenose aortic branch vessels, which would compromise spinal cord perfusion. Clinical studies indicate that selecting stent-graft length or diameter that is too long or too small can result in excessive coverage of intercostal or lumbar arteries, which may disrupt blood flow via the radicular arteries and increase the risk of spinal cord ischemia. Moreover, the stent-grafts have a rigid structure, which might compress nearby vessels, especially at the aortic angulation sites, which could lead to vascular wall injury or thrombosis, and further worsen spinal cord ischemia [16]. However, it's important to note that the relationship between coverage length and SCI risk is mainly based on TAAA repair data. Direct evidence regarding TBAD and TEVAR is limited. Therefore, selecting stent-graft type, length, and deployment site appropriately is crucial to prevent spinal cord ischemia during TEVAR.

3.2.2. Operative Duration and Hemodynamic Stress

Prolonged operative duration and intraoperative hemodynamic fluctuations are recognized as significant precipitating factors for spinal cord ischemia. Extended anesthesia and surgical manipulation may lead to systemic hemodynamic instability, thereby reducing spinal cord perfusion pressure and increasing the risk of ischemic injury [17]. Furthermore, marked intraoperative hemodynamic fluctua-

tions may further impair spinal autoregulation, particularly in patients with pre-existing vascular disease or insufficient collateral circulation [18]. Studies have demonstrated that maintaining intraoperative mean arterial pressure (MAP) between 80 and 100 mmHg effectively ensures spinal cord perfusion, whereas either excessive hypotension or hypertension may precipitate spinal cord ischemia [17]. This target value is derived largely from experience with open surgery and TAAA repair, and has not been specifically validated by RCTs in TBAD-TEVAR. Therefore, optimization of surgical workflow, reduction of operative duration, and strict intraoperative hemodynamic control are essential for reducing the incidence of spinal cord ischemia.

3.3. Patient-Specific Factors

3.3.1. Comorbidities

Underlying diseases like hypertension, diabetes mellitus, and atherosclerosis greatly increase the risk of postoperative spinal cord ischemia [6] [19]. Chronic hypertension might cause arterial wall stiffening and luminal narrowing, which harms the autoregulatory capacity of the spinal vasculature; diabetes mellitus also impairs spinal vascular function via microangiopathy and metabolic disturbances, making the spinal cord more prone to ischemic injury [5]. Large-scale prospective validation is specifically need for TBAD patients undergoing TEVAR. These comorbidities directly affect the functional integrity of the spinal vasculature, and indirectly promote spinal cord ischemia through various pathophysiological mechanisms. So, preoperative assessment should be done with care, and targeted interventions should be implemented, to reduce the risk of postoperative complications.

3.3.2. Individual Variability

Age, sex, genetic polymorphisms are factors that contribute a lot to the pathogenesis of spinal cord ischemia. Studies show elderly patients have a much higher incidence of postoperative SCI compared to younger individuals. This because the vascular elasticity and collateral circulation capacity in elderly patients are reduced [20]. Some studies show that male patients make up a high proportion. This might indicate that the disease has certain epidemiological characteristics, rather than having an independent predictive role of male sex. Therefore, male sex shouldn't be considered a definitive predictor of SCI after TEVAR [11]. Furthermore, genetic polymorphisms have been hypothesized to be potential modulating factors influencing susceptibility to spinal cord ischemia. For instance, there are certain gene variants linked to inflammatory response and oxidative stress, which might increase the risk of postoperative spinal cord ischemia [19]. However, the current evidence is limited to basic research or a small number of candidate gene association studies, which are not TBAD specific. Therefore, this review does not recommend adding such genetic markers to clinical risk assessment.

4. Diagnosis of SCI

4.1. Clinical Manifestations

4.1.1. Early SCI Manifestations

Early symptoms of spinal cord ischemia typically include lower-extremity numbness, weakness, and pain, reflecting combined sensory and motor deficits. These may be unilateral or bilateral and are often accompanied by varying degrees of sphincter dysfunction [21]. Studies have shown that anterior spinal artery syndrome represents a major manifestation of spinal cord ischemia. Its hallmark features include diminished lower-extremity motor function and reduced pain sensitivity [22]. Additionally, some patients may experience intermittent claudication or position-dependent symptom worsening, likely reflecting dynamic changes in spinal cord perfusion [21]. It should be noted that intermittent claudication and positional symptom exacerbation (e.g., worsening with change in body position) are not typical or specific early signs of SCI. They are more often suggestive of lumbar spinal stenosis or peripheral vascular disease. When these symptoms occur after TEVAR, other causes should be ruled out first. They should not be considered as hallmark symptoms of spinal cord ischemia. In clinical practice, early recognition of these symptoms is essential for timely intervention and improved prognosis. Delayed treatment may result in rapid progression to irreversible neurological deficits.

4.1.2. Progressive Manifestations

As spinal cord ischemia develops, patients might develop severe neurological deficits, like paraplegia or quadriplegia [22]. Paraplegia usually shows complete or partial bilateral lower-extremity motor loss, with sensory deficits and sphincter dysfunction. This pattern mainly shows the persistent ischemia in the anterior spinal artery territory [20]. In some instances, the cervical spinal cord might be involved, which could result in quadriplegia, a more severe neurological deficit, often with life-threatening complications like respiratory muscle paralysis [18]. Some patients might develop spinal shock, which presents with flaccid paralysis below injury level, no reflexes, urinary retention, and other symptoms [9]. It's important to note that the description of these advanced-stage manifestations is based on classical neurology, applies to all spinal cord injuries, and is not specific to aortic surgery. These progressive manifestations not only greatly affect the quality of life, but also make treatment more complex and challenging.

4.2. Imaging Studies

4.2.1. Conventional Imaging

Conventional imaging modalities, particularly DSA and CTA, remain fundamental to diagnosing spinal cord ischemia. They are important tools for evaluating aortic anatomy, extent of dissection, degree of true lumen compression, and involvement of spinal cord supply vessels—including intercostal arteries, lumbar arteries, and the Adamkiewicz artery. However, they are not a confirmatory diag-

nostic tool for SCI itself. DSA enables detailed visualization of aortic anatomy and branch vessels, including intercostal and radiculomedullary arteries. This allows direct assessment of spinal cord perfusion and identification of vascular compromise [5]. However, DSA is an invasive procedure associated with complications such as access-site hematoma and contrast-induced nephropathy. These risks limit its broader clinical adoption [13]. By contrast, CTA is non-invasive and provides rapid imaging. Three-dimensional reconstruction allows comprehensive evaluation of aortic dissection extent and branch-vessel involvement [5]. However, CTA is not sensitive to ischemic changes in the spinal cord parenchyma, and DSA cannot directly visualize the pathological status of spinal cord tissue. Hence, neither modality should be used as the sole basis for confirming SCI. Therefore, the choice of imaging method should be guided by the individual patient's clinical circumstances.

4.2.2. Novel Imaging Techniques

For patients who are clinically suspected of having a spinal injury (SCI), the recommended diagnostic approach involves using spinal MRI, which incorporates diffusion-weighted imaging (DWI) sequences, and integrating this with a systematic neurological physical examination. DWI is sensitive to changes in water molecule diffusion, and it effectively identifies pathological changes at the cellular level in the spinal cord. A lot of the early phase of acute SCI, it can show regions of restricted diffusion, giving important guidance for clinical management [10]. So, if there are suspicious neurological symptoms suggesting SCI after TBAD and TEVAR, spinal DWI and MRI should be done urgently. It needs to be combined with a whole neurological physical examination, which includes assessing sensory level, muscle strength grading, reflexes, and sphincter function, since this the main way to confirm the diagnosis and evaluate the conditions severity.

4.2.3. Functional Magnetic Resonance Imaging (fMRI) and Other Exploratory Techniques

FMRI uses changes in Blood Oxygen Level-Dependent (BOLD) signals to assess neuronal activity [23]. The author believes that fMRI holds a certain degree of theoretical appeal in the field of spinal cord ischemia research. However, it should be emphasized that the current application of fMRI in evaluating SCI after aortic surgery remains at an extremely preliminary exploratory research stage. The relevant studies cited in this review do not support the claim that fMRI can be used for real-time localization, immediate monitoring, or functional assessment of SCI following TEVAR. The subjects and paradigms of those studies are completely unrelated to post-operative SCI after aortic surgery. Furthermore, BOLD signals in the spinal cord are susceptible to interference from respiration, cardiac pulsation, and magnetic field inhomogeneity. Standardized acquisition and analysis protocols are lacking. No validated data exist to demonstrate its utility in clinical decision-making for SCI after TEVAR. Therefore, this review does not recommend the use of fMRI as a diagnostic or monitoring tool for SCI after TEVAR.

Substantial basic and clinical research is still needed before its clinical translation can be realized.

4.3. Electrophysiological Studies

4.3.1. Somatosensory Evoked Potentials (SSEPs)

SSEPs are a common electrophysiological modality. They record cortical potentials after peripheral nerve stimulation, which gives an indirect assessment of spinal sensory conduction pathway integrity. Moreover, there is a strong correlation between the SSEP waveform changes and the severity of spinal cord ischemia. Specifically, if the amplitude is reduced or the latency is prolonged, this usually indicates spinal cord dysfunction [24] [25]. Conversely, complete waveform loss signifies severe conduction impairment. Evidence shows that monitoring with SSEPs after TEVAR can detect SCI early, enabling timely treatment modification [12]. But SSEPs are prone to multiple confounding factors, like anesthetic depth, temperature changes, and electrode positioning. These may result in false positive or negative outcomes. Therefore, clinical interpretation necessitates the integration of complementary diagnostic modalities [26] [27]. It should be noted that much of the evidence for SSEPs is derived from TAAA repair and open spinal surgery. Direct data specific to TBAD-TEVAR are limited, and thus the evidence is largely indirect.

4.3.2. Motor Evoked Potentials (MEPs)

MEPs evaluate the spinal motor conduction. Responses are produced via transcranial magnetic or electrical stimulation of the motor cortex, and corresponding electrical activity is recorded at the spinal cord and peripheral neuromuscular junctions [28]. The MEPs provide a sensitive method to detect motor neuron excitability and conduction integrity in the spinal cord ischemia. Waveform changes directly relate to the severity of ischemic conditions [3]. For instance, a significant reduction in amplitude or a prolonged latency indicates an injury to the spinal motor pathway. Conversely, the absence of waveforms suggests a complete motor loss [20], but MEPs have significant limitations. They are very sensitive to anesthetics, and technical factors might prevent stable waveform acquisition in some patients [29]. Similarly, direct evidence for MEPs in TEVAR is less than that from TAAA/open surgery, so it should be considered indirect inference. So, although MEPs are useful for diagnosing spinal cord ischemia, they need to be combined with complementary modalities to improve diagnostic accuracy and reliability.

5. Treatment of Spinal Cord Ischemia

5.1. Acute Spinal Cord Injury Management Pathway

When SCI is confirmed or highly suspected after TBAD-TEVAR, the following acute management pathway should be followed, rather than applying isolated individual measures in a piecemeal fashion.

5.1.1. Immediate Neurological Assessment and Grading

Perform an urgent neurological physical examination to determine the sensory and motor levels of impairment, muscle strength (using the ASIA grading scale), and sphincter function. Simultaneously, complete spinal DWI-MRI to confirm the ischemic territory and exclude other causes, such as epidural hematoma.

5.1.2. Optimization of Spinal Cord Perfusion Pressure (SCPP)

Elevation of Mean Arterial Pressure (MAP) is the primary hemodynamic intervention for managing SCI. After excluding cardiac dysfunction and bleeding risk, it is recommended to raise MAP to a target of 90 - 100 mmHg (or an increase of 10% - 20% from baseline) and maintain this until neurological symptoms stabilize or improve. This measure directly increases spinal cord blood flow and serves as the cornerstone of any SCI management pathway. (Note: The recommendation of MAP ≥ 90 mmHg is derived from experience in TAAA repair and open surgery [11]. Specific RCT evidence in TBAD-TEVAR is lacking, but this target is widely accepted as a reasonable clinical practice.)

5.1.3. Correction of Hypoxemia and Anemia

Maintain SpO_2 ≥ 95%; administer oxygen therapy or non-invasive/invasive ventilation when necessary. Maintain hemoglobin at ≥80 - 100 g/L, adjusted according to the patient's baseline status. Severe anemia can reduce oxygen delivery and exacerbate ischemic injury [11].

5.1.4. Indications and Implementation of Cerebrospinal Fluid Drainage (CSFD)

CSFD reduces intraspinal pressure and increases SCPP [4]. It should be clarified that direct, high-quality prospective evidence for CSFD in TBAD-TEVAR is lacking. Its recommendation is mainly extrapolated from evidence in open and endovascular TAAA repair [30]. Therefore, in TBAD-TEVAR patients, CSFD should be reserved for the following situations: 1) moderate to severe SCI (e.g., ASIA grade B or lower) with poor response to vasopressor therapy; 2) delayed SCI with no contraindications on imaging (*i.e.*, no coagulopathy, no intracranial hypertension, and no infection at the puncture site). During implementation, strict aseptic technique should be followed. The drainage rate should be controlled at 10 - 15 mL/h to avoid intracranial hypotension due to over-drainage [31]. Clinical outcome assessment relies mainly on neurological function scores and imaging findings, such as changes in spinal cord edema on MRI [32].

5.1.5. Assessment of Reversible Vascular Etiologies

If clinically feasible and there is high suspicion of mechanical vascular obstruction—such as stent coverage of critical intercostal artery ostia or progressive false lumen enlargement compressing the true lumen—CTA or DSA can help identify target lesions that may be amenable to interventional (e.g., branch artery stenting) or surgical (e.g., vascular bypass) treatment. However, such procedures are complex and advanced, requiring multidisciplinary team evaluation. They should not be used as first-line routine management.

5.2. Pharmacological Interventions

5.2.1. Vasodilators

Clinical experience suggests that continuous infusion of alprostadil can prevent postoperative spinal cord thrombosis and promote neurological recovery [21]. Currently, there is a lack of high-quality clinical evidence to support its recommended use in cardiothoracic surgery.

5.2.2. Anticoagulants

Anticoagulants are mainly used to manage spinal cord ischemia to avoid thrombosis and enhance local perfusion. Heparin and low molecular weight heparin are the most commonly used agents [33]. Heparin potentiates antithrombin III activity, inhibits thrombin and multiple coagulation factors, producing a rapid and potent anticoagulant effect [5]. Studies show that early heparin administration after TEVAR reduces spinal cord thrombosis risk and improves outcomes in patients with SCI [11]. Compared to low-molecular weight heparin, it has a longer half-life and more predictable anticoagulant effect. It also shows a lower risk of bleeding, which has made it suitable for widespread clinical use. But the available direct evidence is limited to traumatic spinal injury or the prevention of peripheral vascular thrombosis. There is no direct supporting data for SCI following TEVAR, and the bleeding risk must be carefully considered. A lot of the anticoagulant therapy requires close monitoring of coagulation parameters, like activated partial thromboplastin time and international normalized ratio, to avoid bleeding complications from over-anticoagulation [34]. Also, low molecular weight heparin dosage needs careful adjustment in patients with renal insufficiency to prevent drug accumulation and adverse effects.

5.2.3. Neuroprotective Agents

Neuroprotective agents are crucial in the management of spinal cord ischemia. They reduce ischemia-reperfusion injury and keep neuronal function, so it supports neurological recovery. Edaravone is a free radical scavenger, it neutralizes reactive oxygen species (ROS) and blocks lipid peroxidation. These actions can limit cellular membrane damage and neuronal apoptosis [20]. Animal experimental studies indicate that edaravone might help alleviate ischemia-reperfusion injury; however, there is a lack of clinical RCT verification. Gangliosides constitute another crucial class of neuroprotective agents. They integrate into the neuronal cell membranes, stabilize the membrane architecture, and promote the release of nerve growth factor, thus accelerating neuronal repair and regeneration [20]. From clinical experience, it's found that gangliosides not just reduce spinal cord ischemia-reperfusion injury, but also improve long-term outcomes. These benefits are especially strong when combined with other therapeutic modalities [18]. But the mentioned drugs don't have well-designed clinical RCTs to prove their effectiveness in improving long-term neurological outcomes after TEVAR-related SCI. They are not recommended to be routine or first-line therapy. Their precise mechanisms of action are not yet understood, and their long-term safety

and effectiveness need to be validated by large-scale clinical trials.

5.2.4. Overall Role of Pharmacological Therapy

Pharmacological therapy must be used in combination with the aforementioned management measures (vasopressor support, CSFD, and oxygenation maintenance). It should always be placed in an adjunctive role and should not be relied upon as a standalone treatment.

5.3. Vascular Bypass Surgery and Other Interventional Modalities

Vascular bypass surgery is a effective way to treat moderate to severe spinal cord ischemia by rebuilding spinal cord perfusion. Its fundamental principle is to restore blood supply via vascular anastomosis [35]. Common bypass configurations include intercostal-to-lumbar and internal iliac-to-lumbar artery bypasses. These procedures anastomose patent donor vessels to spinal segmental arteries, rerouting blood flow around diseased or obstructed segments to restore spinal cord perfusion [35]. Surgical indications include severe spinal cord ischemia refractory to conservative management, as well as high-risk patients with significant vascular anatomical abnormalities [6]. It's important to note that such procedures are limited to a few case reports in TBAD-TEVAR. The main experience is from open TAAA repair. In recent years, TEVAR and MISACE have become new strategies for preventing SCI [36]-[38]. These methods can greatly reduce the risk of SCI. But they were developed mainly for complex TAAA repair. In TBAD, their use is exploratory or limited to case reports, and they are not a standard recommendation.

6. Prevention of Spinal Cord Ischemia

6.1. Preoperative Assessment

6.1.1. Imaging Assessment

Preoperative high-resolution imaging is crucial for the prevention of spinal cord ischemia. It allows for a comprehensive assessment of the extent of aortic dissection, the anatomical distribution of spinal vascular anatomy, and the condition of branch-vessel. CTA and MRI are the main clinical modalities. They precisely define dissection anatomy, like intimal entry site and false lumen extent, and provide detailed visualization of arteries supplying spinal cord [39]. For instance, CTA accurately determines whether major spinal supply vessels, such as intercostal and lumbar arteries, are affected by dissection. MRI is very sensitive for detecting morphological changes in spinal tissue, this helps early identification of ischemic risk [3]. Also, DSA is usually reserved for complex cases, because it's invasive and has a lot of radiation. So, it mainly serves a supplementary tool. Overall, these imaging modalities are integrated well, which supports precise preoperative planning, and reduces the risk of spinal cord ischemia by a lot.

6.1.2. Risk Assessment Models

Current models for predicting spinal cord ischemia (SCI) risk combine clinical features, imaging results, and procedural factors to inform postoperative risk

stratification. But these models have limited predictive accuracy and clinical usefulness. They usually rely on single isolated variables, which doesn't account for patient heterogeneity and the complex interactions among multiple risk factors [6]. 2026 ESVS guidelines emphasize the development of multivariable models incorporating the extent of left subclavian artery coverage, operative time, and anesthetic depth [2]. Recent advances in big data analytics and artificial intelligence have enabled more refined individualized risk prediction. Integrating multidimensional variables like hypertension, diabetes, disease extent, left subclavian artery coverage, and stent graft coverage improves predictive accuracy by a lot [7]. But none of the existing models have been validated externally in TBAD cohorts, so their generalizability is not confirmed. Moreover, combing dynamic intraoperative hemodynamic monitoring with postoperative neurological assessments improves the risk evaluation frameworks. Future studies should focus on developing comprehensive prediction tools that include more variables during the perioperative period. Such tools will support the accurate SCI risk stratification and allow for individualized patient management.

6.2. Intraoperative Strategies

6.2.1. Stent Graft Selection

In the TEVAR for aortic dissection, selecting the right stent graft is vital to maintain spinal cord perfusion. Stent configurations like bare metal, covered, and branched designs have a lot of differences in their effect on spinal blood supply. So, selection must be tailored to the patients anatomy. Bare metal stents have higher porosity and lower radial force, which usually keeps branch vessel patency. But they are less effective at excluding intimal tear [21]. Compared to covered stent grafts, they have more effective intimal tear exclusion. Their rigid structure might block critical branch vessels, like intercostal and lumbar arteries, which could increase the risk of spinal cord ischemia [40]. Recently, new branched stent grafts, such as the Castor device, offer a new solution to this problem. Such designs allow for the reconstruction of the left subclavian artery, while also maximizing the preservation of other critical branch vessels. There is evidence indicating that the use of Castor stent-graft is associated with a low incidence of spinal cord ischemia. But it's important to note that the current data are mainly obtained from single-center retrospective studies, and there is still a need for more evidence [41]. Moreover, combing a single-wire guidewire technique with branched stent graft deployment reduces the operative time and improves the procedural precision. This strategy provides a safer and more effective clinical alternative.

6.2.2. Optimization of Operative Technique

Optimizing operative techniques is crucial to avoid spinal cord ischemia. Key strategies include reducing operative time, maintaining stable intraoperative hemodynamics, and minimizing the coverage of critical branch vessels. Longer operative time can cause spinal cord hypoperfusion, which increases ischemic risk

[42]. So, surgeons need to be very technical and plan stent deployment strategically. This reduces the number of unnecessary procedural steps. Keeping intraoperative hemodynamics stable is also important for preventing SCI. Blood pressure fluctuations result in significant variations in the perfusion pressure of the spinal cord. These changes might result in ischemic injury. Intraoperative blood pressure requires close monitoring. Vasoactive agents should be titrated to keep mean arterial pressure within the target range of 80 - 10 mmHg [16]. The evidence supporting this target value is partly based on experience with TAAA and open surgical approaches. Avoiding the excessive coverage of critical branch vessels is crucial. Intraoperative stent-graft selection should be decided by preoperative high-resolution imaging, like CTA or DSA, to get the optimal length and size. This method ensures the patency of intercostal and lumbar arteries, as well as other spinal supply vessels. Collectively, these integrated measures significantly reduce the risk of spinal cord ischemia.

6.3. Postoperative Monitoring and Management

6.3.1. Vital Signs Monitoring

Postoperative vital signs monitoring is vital for the early detection of spinal cord ischemia. Blood pressure, heart rate, and oxygen saturation have abnormal fluctuations, which might suggest spinal hypoperfusion or systemic metabolic derangement, so we need to intervene immediately. Studies show that postoperative hypertension causes aortic wall stress to increase, which might worsen dissection or cause new intimal tears. Conversely, hypotension further reduces spinal perfusion pressure, thereby increasing ischemic risk [43]. Moreover, heart rate and oxygen saturation fluctuations may indicate overall systemic health. Tachycardia might suggest hypovolemia or pain. Hypoxemia can lead to tissue hypoxia, and this can exacerbate spinal cord injury [9]. Multimodal monitoring allows for the early detection of potential complications, enabling targeted interventions to prevent spinal cord ischemia.

6.3.2. Neurological Monitoring

Postoperative neurological assessment is crucial for the early detection of spinal cord ischemia. Close monitoring of limb sensation, motor function, and sphincter tone allows for early detection of neurological deficits, enabling timely intervention. Studies show that lower-extremity numbness, weakness, and pain are common initial signs of SCI, so they should be recognized quickly [9]. Also, sphincter dysfunction, like urinary retention or fecal incontinence, is another important sign of spinal cord ischemia. Its presence is usually a sign of involvement in the conus medullaris or cauda equina [44], Postoperative neurological monitoring should include the time aspect (immediate vs. delayed) and should be done for at least 72 hours post-surgery to account for the window during which delayed SCI may occur. To enhance the sensitivity and specificity of neurological monitoring, we can integrate electrophysiological techniques like SSEPs and MEPs into a comprehensive assessment. These modalities provide real-time insights into the integ-

rity of spinal cord conduction, offering objective data to facilitate prompt diagnosis. With the help of multimodal surveillance, clinicians can detect early signs of SCI quickly, and implement timely interventions, thus significantly improving patient outcomes.

7. Conclusions

7.1. Summary of Key Findings

SCI is a severe complication that arises after the TEVAR (Type B Aortic Dissection) procedure. The pathogenesis of SCI is complex and involves multiple factors. Anatomical factors, like spinal cord blood supply architecture and its connection to the aorta, determine whether endovascular repair might damage spinal perfusion. Operative factors include stent-graft effects on branch vessels, the duration of the operation, and intraoperative hemodynamic fluctuations. Comorbidities and patient-specific variations further elevate the risk of postoperative SCI. The main point is that the evidence from the TBAD-TEVAR population is limited in the current evidence base. Many clinical practice recommendations are derived from TAAA repair, open aortic surgery, and basic neuroscience. So, we need to be very cautious when translating these findings into clinical practice. For diagnosis, we combine early recognition of clinical manifestations with conventional imaging techniques (like DSA and CTA) and novel techniques (such as DWI and fMRI), as well as electrophysiological studies (like SSEPs and MEPs), to improve both the accuracy and timeliness of the diagnosis. Treatment includes pharmacological and surgical treatments. Vasodilators, anticoagulants, and neuroprotective agents are able to enhance spinal perfusion and reduce reperfusion injury. Cerebrospinal fluid drainage and vascular bypass surgery directly restore spinal blood supply by intervention. Prevention is based on a comprehensive preoperative assessment, precise risk assessment models, correct stent-graft selection, optimized operative technique, and vigilant postoperative monitoring of vital signs and neurological status. Collectively, these integrated strategies are crucial for reducing the occurrence of spinal cord ischemia.

7.2. Future Perspectives

Even though there were a lot of recent advances in SCI research after TEVAR for TBAD, a lot of unresolved challenges remain. Future studies need to further clarify the specific pathogenetic mechanisms of SCI, especially focusing on molecular and genetic determinants. But genetic polymorphisms should be added to risk models only after accounting for confounding factors; currently, they are not considered clinical predictors. AI-assisted risk prediction models also need further development. In the diagnostic field, the imaging and electrophysiological modalities that are more sensitive and specific are required. For exploratory techniques like fMRI, we first need to validate the standardized acquisition protocols in animal models or healthy volunteers, and then perform clinical evaluation. Such advances would allow for early and accurate diagnosis, enabling better clinical deci-

sion-making. Therapeutic initiatives should prioritize the development of novel pharmacological and surgical innovations. This includes the use of targeted neuroprotective agents and the implementation of anatomically optimized stent-graft designs to mitigate postoperative complications. Rehabilitation optimization is a crucial future direction, which involves integrating intelligent devices and virtual reality technologies to enhance recovery outcomes and quality of life. Preventive strategies should prioritize refined risk assessment models, and combining them with artificial intelligence might allow for individualized prediction and management. To sum up, multidisciplinary collaboration and cross-disciplinary research are promising for enhancing therapeutic efficacy and long-term outcomes. Such methods will offer crucial guidance for the clinical management of this complex complication.

Author Contributions

Chaohui Huang: Conceptualization, methodology, investigation, data curation.

Shiguan Luo: Conceptualization, supervision, funding acquisition, project administration, writing review & editing.

Weining Xu: Formal analysis, validation, resources, writing review & editing (Assisted in the study).

AI-Assisted Tool Disclosure

AI was used to assist with the language polishing of this manuscript, improving its fluency and clarity of expression.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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