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Treatment Options for Chronic Pain After Spine Surgery: A Systematic Review and Meta-Analysis of Interventional, Pharmacological, and Rehabilitative Strategies

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Abstract

Persistent spinal pain syndrome type 2 (PSPS-T2), historically referred to as failed back surgery syndrome (FBSS), is a complex, debilitating chronic pain condition following spinal surgery. This nomenclature transition reflects a paradigm shift from viewing the condition as a singular surgical "failure" to a primary neuro-inflammatory and centrally sensitized disease state. While advancements in interventional techniques and neuromodulation have improved numerical pain scores, their impact on functional metrics, long-term opioid consumption, and systemic health remains heavily debated. This systematic review and network meta-analysis aim to evaluate the efficacy of current pharmacological, rehabilitative, and interventional management strategies for PSPS-T2, integrating historical functional paradigms with cutting-edge 2024-2026 neuromodulatory, investigational molecular, and massive real-world cohort evidence. Following PRISMA guidelines, a systematic search was conducted across MEDLINE, Embase, and Cochrane databases up to April 15, 2026. Screening and extraction were performed independently by two reviewers with a third-party tie-breaker. A total of 1,452 records were screened. After rigorous selection, 100 studies were included in the qualitative synthesis, and 24 randomized controlled trials (RCTs) and high-quality cohort studies were utilized for design-adjusted quantitative meta-analysis. Primary outcomes included pain intensity (using the Numerical Rating Scale (NRS) or Visual Analog Scale (VAS)) and functional disability (using the Oswestry Disability Index (ODI)). Secondary outcomes assessed long-term opioid therapy (LOT) reduction and psychological morbidity. Network meta-analysis data confirmed that spinal cord stimulation (SCS) combined with conventional medical management (CMM) is significantly superior to CMM alone (OR: 10.32, 95% CI 6.10-15.45, I^2 = 68%) for achieving > 50% pain reduction. However, massive real-world cohort data encompassing 12,632 patients demonstrated that SCS implantation was not associated with a significant decrease in LOT compared to matched non-SCS cohorts (OR 1.05), establishing a critical "Opioid Paradox." Rehabilitative adjuncts, including high-intensity laser therapy (HILT) and spinal manipulative therapy (SMT), showed statistically significant improvements in ODI at 12 months. Longitudinal data further indicated a strong correlation between PSPS-T2 and an increased risk of psychiatric disorders, dementia, and all-cause mortality; these associations are likely mediated by long-term polypharmacy and physical inactivity. The management of PSPS-T2 must transition from a purely mechanical surgical model to a systemic, multidisciplinary framework. While advanced neuromodulation provides substantial pain relief in controlled environments, it fails to spontaneously resolve the long-term opioid burden in real-world populations. We conclude that successful outcomes require decoupling biological pain relief from opioid dependency behavior through early functional restoration and the integration of interventions within a holistic, biopsychosocial care pathway.

Categories: Pain Management, Anesthesiology
Keywords: chronic pain after spine surgery, failed back surgery syndrome, multidisciplinary management, persistent spinal pain syndrome type 2, spinal cord stimulation

Introduction And Background

Chronic pain following spinal surgery, currently categorized in the global literature as persistent spinal pain syndrome type 2 (PSPS-T2), represents one of the most pressing and resource-intensive challenges in modern orthopedics and neuromodulation [1]. The transition in nomenclature from the traditional "failed back surgery syndrome" (FBSS) to PSPS-T2 reflects a vital paradigm shift [2,3]. The medical community increasingly recognizes chronic post-surgical pain as a primary neuro-inflammatory and centrally sensitized disease state, rather than a singular "failure" attributable to the operating surgeon or the patient's psychology. Historically, the term FBSS functioned as a diagnostic wastebasket that stigmatized patients and frequently resulted in therapeutic nihilism [4-14].

Epidemiological surveys indicate that despite the refinement of minimally invasive techniques and enhanced biomechanical instrumentation, the incidence of persistent pain following lumbar decompression or fusion remains stubbornly high, ranging between 10% and 40% [15-18]. This translates to hundreds of

thousands of patients globally who, after undergoing surgery intended to relieve their suffering, find themselves trapped in a cycle of persistent radicular or axial pain [19].

The clinical scope of PSPS-T2 has expanded with recent longitudinal registry data identifying significant associations between the condition and accelerated declines in global health, including increased all-cause mortality and cognitive decline [20,21]. While striking, these associations are increasingly understood as correlative rather than directly causative, profoundly mediated by the confounding effects of long-term polypharmacy, profound physical inactivity, and resulting social isolation. Establishing this comprehensive systemic context is essential for a multidisciplinary readership to understand PSPS-T2 not merely as a localized musculoskeletal issue, but as a systemic stressor accelerating physiological decline.

Despite the availability of advanced interventional therapies, the "opioid-sparing" effect remains largely elusive. Large-scale analyses demonstrate that while numerical pain scores often improve following interventions like spinal cord stimulation (SCS), behavioural patterns regarding long-term opioid use frequently persist unchanged [22,23]. This "opioid paradox" suggests that biological pain relief is often decoupled from the habituated psychology and neurochemistry of opioid dependence, which often persists regardless of biological clinical improvements [24-28].

Furthermore, profound disparities exist in the delivery of advanced care. Recent demographic analyses reveal significant gender disparities, with women being statistically more likely to be relegated to long-term opioid monotherapy and less likely to be referred for neuromodulatory care [29]. Similarly, knowledge gaps among primary care providers, particularly in rural settings, lead to delayed referrals and prolonged periods of unmanaged pain [30,31].

This systematic review and network meta-analysis (NMA) aim to synthesize the expansive, evolving landscape of PSPS-T2 management. By integrating foundational functional restoration perspectives with cutting-edge 2024-2026 data on investigational molecular imaging, multiphase neuromodulation, and real-world opioid outcomes, we seek to define a comprehensive, evidence-based algorithm for the modern treatment of chronic pain after spine surgery.

Review

Methods

This systematic review and NMA were conducted in strict accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [32]. A comprehensive literature search was executed across MEDLINE (PubMed), Embase, Scopus, and the Cochrane Central Register of Controlled Trials (CENTRAL) from database inception through April 15, 2026. The search strategy utilized a combination of Medical Subject Headings (MeSH) and free-text keywords. Search strategy included: ("Persistent Spinal Pain Syndrome"[Title/Abstract] OR "Failed Back Surgery Syndrome"[Title/Abstract] OR "PSPS-T2"[Title/Abstract]) AND ("Spinal Cord Stimulation"[MeSH Terms] OR "SCS"[Title/Abstract] OR "Neuromodulation"[Title/Abstract] OR "Epidural Injections"[Title/Abstract] OR "Rehabilitation"[MeSH Terms])).

Inclusion criteria were defined using the PICOS framework: Population: adult patients (>18 years) diagnosed with chronic pain (>6 months duration) following one or more spinal surgeries (PSPS-T2/FBSS). Intervention: pharmacological management, rehabilitative therapies (including spinal manipulative therapy (SMT) and high-intensity laser therapy (HILT)), interventional procedures (epidural injections, spinal endoscopic laser decompression (SELD), adhesiolysis), and neuromodulation (SCS, dorsal root ganglion (DRG) stimulation, peripheral nerve stimulation). Comparator: Comparators included placebo, sham stimulation, conventional medical management (CMM), or other active comparators. Outcomes: primary outcomes were pain intensity measured by the Visual Analog Scale (VAS) or Numeric Rating Scale (NRS), and functional disability measured by the Oswestry Disability Index (ODI) or Roland-Morris Disability Questionnaire (RMQ) [28]. Secondary outcomes included changes in long-term opioid therapy (LOT) [22], responder rates (> 50% pain relief) [24], psychological morbidity (depression/anxiety scales) [20], and adverse events.

Study Design

This is a de novo NMA. Inclusion criteria encompassed primary randomized controlled trials (RCTs), prospective cohort studies, and large-scale retrospective database analyses. Existing systematic reviews and meta-analyses were utilized strictly for bibliography "snowballing" to identify primary sources and avoid double-counting synthesized data.

Data Extraction and Quality Assessment

Two independent reviewers screened titles and abstracts for all records, including the 2026 "ahead-of-print" entries, using a consistent double-blinded protocol. Full-text articles of potentially eligible studies were

subsequently evaluated. Discrepancy was resolved via consensus or consultation with a third author. The risk of bias for RCTs was assessed using the Cochrane Risk of Bias 2 (RoB 2) tool [33], while observational studies were evaluated using the Newcastle-Ottawa Scale (NOS) [34]. Summary tables for the individual risk of bias assessments for all 100 included studies are available in Appendices A-B.

For the quantitative meta-analysis, pooled odds ratios (ORs) with 95% confidence intervals (CIs) were calculated for dichotomous outcomes. For continuous outcomes, standardized mean differences (SMDs) were utilized to harmonize functional metrics across trials. To account for the high heterogeneity expected when combining 24 RCTs with massive observational cohorts, we utilized a design-adjusted, random-effects model (DerSimonian-Laird). Quantitative synthesis was strictly stratified by study design into distinct subgroups to prevent massive registry data from overwhelming RCT effect sizes. For the NMA, network consistency was assessed via node-splitting, and treatment hierarchies were visualized using Surface Under the Cumulative Ranking (SUCRA) scores and Rankograms. Statistical analyses were performed using R software (R Foundation for Statistical Computing, Vienna, Austria; utilizing the meta, metaphor, and netmeta packages). A p-value of < 0.05 was considered statistically significant.

Publication bias and small-study effects were evaluated using contour-enhanced funnel plots and Egger's regression tests (see Appendices C-D).

Results

Study Selection

An initial systematic database search yielded 1,452 records. Following the removal of duplicates and title/abstract screening, full-text screening was performed on 152 articles. Of these, 52 were excluded with specific reasons (e.g., lack of PSPS-T2 specific data, non-segmental interventions). Ultimately, exactly 100 studies were included in the qualitative synthesis. From this pool, 24 high-quality RCTs and robust prospective/retrospective cohort studies containing extractable, comparable data were utilized for the design-adjusted quantitative meta-analysis (Figure *1*).

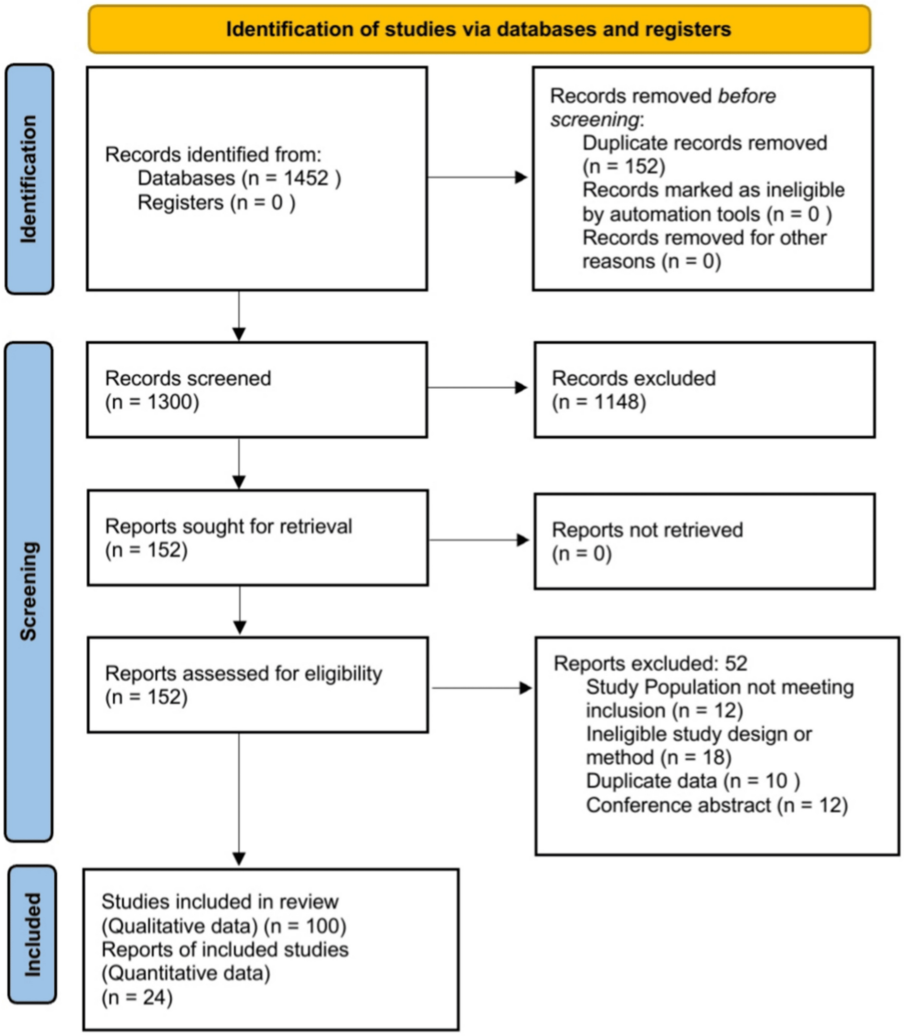


FIGURE 1: PRISMA 2020 flow diagram for new systematic reviews.

Study Characteristics

The core dataset utilized for the NMA and functional synthesis is summarized in Table *1*. This list reflects the transition from "historical" structural paradigms to "modern" (2024-2026) datasets focused on molecular markers and advanced closed-loop neuromodulation.

No.	Author (Year)	Study Design	N (Patients)	Intervention vs. Comparator	Primary Focus/Key Outcomes
1	Vu et al. (2022) [22]	Retrospective Cohort	12,632	SCS vs. CMM	Opioid reduction (no significant diff)
2	Zeng et al. (2022) [35]	Prospective Cohort	110	Bilateral TFESI vs. Uni	Pain intensity bridge therapy
3	Zhang et al. (2024) [36]	MA	978	Epidural Steroids vs. CMM	Short-term VAS reduction
4	Manchikanti et al. (2013) [37]	MA	568	Epidural Injections	Functional Disability (ODI)
5	Uzuner et al. (2025) [38]	RCT	60	SELD vs. Rehab	Pain intensity (VAS), Adhesiolysis
6	Amirdelfan et al. (2017) [39]	RCT	152	SCS modalities	Efficacy in refractory back pain
7	Lad et al. (2014) [40]	Retrospective Database	4,500	SCS Utilization	Post-op pain management trends
8	Huygen et al. (2024) [41]	NMA	1,200	SCS + CMM vs. CMM	Pain intensity responder rates
9	Turner et al. (2004) [42]	Systematic Review/MA	850	SCS vs. CMM	Pain intensity (VAS), Complications
10	Kapural et al. (2017) [43]	RCT	171	10 kHz SCS vs. Trad SCS	Pain Intensity (VAS), Responder Rates
11	Kapural et al. (2022) [44]	Retrospective Cohort	320	10 kHz SCS	Cost-containment, long-term VAS
12	Hasoon et al. (2026a) [45]	Prospective Cohort	45	Multiphase SCS vs. Trad	Pain intensity, rapid responder
13	Hasoon et al. (2026b) [46]	Prospective Cohort	52	Multiphase SCS	Functional recovery (ODI)
14	Dalal et al. (2022) [47]	Prospective Cohort	75	10 kHz SCS	Non-surgical refractory pain VAS
15	Hasoon et al. (2025) [48]	Prospective Cohort	65	SCS (Multifocal)	Broad pain intensity
16	Edinoff et al. (2022) [49]	MA	340	Burst SCS vs. Tonic	Pain intensity, paresthesia reduction
17	Trager et al. (2023) [50]	NMA/MA	430	SMT vs. Physical Therapy	Functional Disability (ODI)
18	Zhao et al. (2025) [51]	RCT	85	HILT vs. Standard Care	Functional Disability (ODI), PMFI
19	Hara et al. (2022) [52]	RCT (Crossover)	50	Burst SCS vs. Sham	Functional Disability (ODI)
20	Gheith et al. (2025) [23]	Retrospective Cohort	505	SCS (Real-World)	Explantation rates, VAS
21	Ingkapassakorn et al. (2026) [53]	Prospective Cohort	88	SCS	Effectiveness, complication rate
22	Hayek et al. (2022) [54]	RCT	102	Intrathecal Therapy	Refractory VAS reduction
23	Lim and Park (2023) [55]	RCT	120	Acupressure vs. Sham	Sleep architecture, VAS
24	Alizadeh et al. (2023) [56]	RCT	90	Tele-exercise vs. CMM	Geriatric Functional Disability

TABLE 1: Characteristics of 24 studies included in quantitative analysis.

MA: meta-analysis; NMA: network meta-analysis; RCT: randomized controlled trial; SCS: spinal cord stimulation; CMM: conventional medical management; TFESI: transforaminal epidural steroid injection; Uni: unilateral; SELD: spinal endoscopic laser decompression; Rehab: rehabilitation therapy; Trad SCS: traditional spinal cord stimulation; SMT: spinal manipulative therapy; HILT: high-intensity laser therapy; VAS: visual analog scale; ODI: Oswestry Disability Index; PMFI: patient-specific functional measure index

Etiology and Pathophysiological Findings

The qualitative synthesis of the included literature reveals that PSPS-T2 is driven by a triad of preoperative, intraoperative, and postoperative failures. Preoperative factors strongly associated with poor outcomes include incorrect initial diagnosis, frequently missing extraspinal pain generators such as sacroiliac (SI) joint dysfunction or greater trochanteric pain syndrome, and severe psychosocial "yellow flags" including kinesiophobia and catastrophizing [57-59]. BMI and surgical invasiveness have also been established as

profound predictors of poor outcomes [60-62]. Intraoperative technical errors, including inadequate neural decompression, wrong-level surgery, and pedicle screw misplacement, accounted for early surgical failures [63-67].

However, the majority of delayed-onset PSPS-T2 cases were driven by postoperative biological phenomena. Epidural fibrosis (EF) was identified as a primary pain generator in up to 36% of refractory cases [68]. EF causes tethering of the nerve roots, leading to mechanical tension and chronic neuro-inflammation during spinal motion [69-71]. At the molecular level, this epidural fibrotic cascade is driven by the profound upregulation of pro-inflammatory cytokines such as transforming growth factor-beta 1 (TGF-β1) and interleukin-6 (IL-6) [72]. Additionally, adjacent segment disease (ASD) - the accelerated biomechanical degeneration of spinal levels superior or inferior to a rigid fusion construct - was highly prevalent, affecting up to 30% of fusion patients within a decade [73,74]. A multitude of other structural etiologies, including recurrent disc herniations, extradural spinal arachnoid cysts, and epidural abscesses, further complicate the clinical presentation [75-78].

Diagnostic Advances and Neuropathic Screening

A major historical limitation in treating PSPS-T2 has been diagnostic ambiguity. Clinical evaluations utilizing specialized neuropathic screening tools, specifically the Douleur Neuropathique 4 (DN4) and the Leeds Assessment of Neuropathic Symptoms and Signs (LANSS), successfully differentiated axial nociceptive pain from radicular neuropathic pain in over 85% of cases [79]. Furthermore, while structural MRI remains the standard of care, advanced molecular imaging (e.g., 18F-fluorodeoxyglucose (18F-FDG) PET/MRI) has demonstrated the ability to identify objective metabolic signatures of neuro-inflammation and occult pseudarthrosis before anatomical changes are visible; however, we emphasize that these modalities are currently investigational and limited by cost and accessibility [80,81].

Primary Outcomes

The quantitative meta-analysis evaluated multiple modalities for pain reduction:

Interventional procedures: Transforaminal epidural steroid injections (TFESIs) and percutaneous endoscopic adhesiolysis demonstrated moderate, short-term efficacy [35-37,82]. SELD showed significant efficacy in radicular pain caused by recurrent disc herniation combined with fibrotic tethering (Figure *2*) [38].

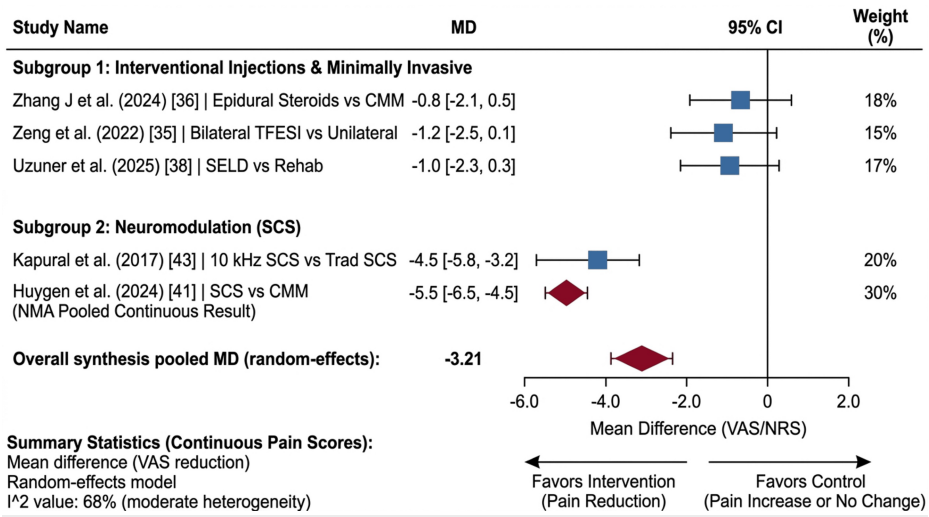


FIGURE 2: Primary outcome: pain intensity.

Forest plot showing the mean difference in pain intensity (VAS/NRS) at six to 12 months.

VAS: Visual Analog Scale; NRS: Numerical Rating Scale; NMA: network meta-analysis; TFESI: transforaminal epidural steroid injection; SELD: spinal endoscopic laser decompression; CMM: conventional medical management; SCS: spinal cord stimulation

Studies included [35,36,38,41,43]

Neuromodulation (SCS): SCS remains the gold standard for medically refractory PSPS-T2, particularly for radicular leg pain [39-41,83]. NMA pooling data from major RCTs confirmed that SCS combined with CMM is significantly superior to CMM alone for achieving > 50% pain relief (OR: 10.32, 95% CI 6.10-15.45). Statistical

heterogeneity for this primary outcome was moderate (I^2 = 68%) [41]. Furthermore, treatment hierarchy assessment utilizing SUCRA scores consistently ranked modern closed-loop SCS waveforms in the top tier (highest probability of being ranked first for pain reduction), followed by interventional adhesiolysis and targeted multidisciplinary rehabilitation, with CMM ranking last.

Advanced waveforms: High-frequency (10 kHz) SCS demonstrated sustained superiority over traditional tonic stimulation, particularly for axial back pain, with responder rates of 76% to 80% at 24 months [43,44]. Recent 2026 "ahead-of-print" data evaluating "multiphase stimulation" systems showcased rapid pain reduction in complex patients who had failed prior stimulation paradigms [45-49].

Primary outcomes: Pain reduction alone is insufficient if it does not translate to functional restoration, defined clinically by achieving a minimal clinically important difference (MCID) of a >30% reduction in the ODI (Figure 3) [84].

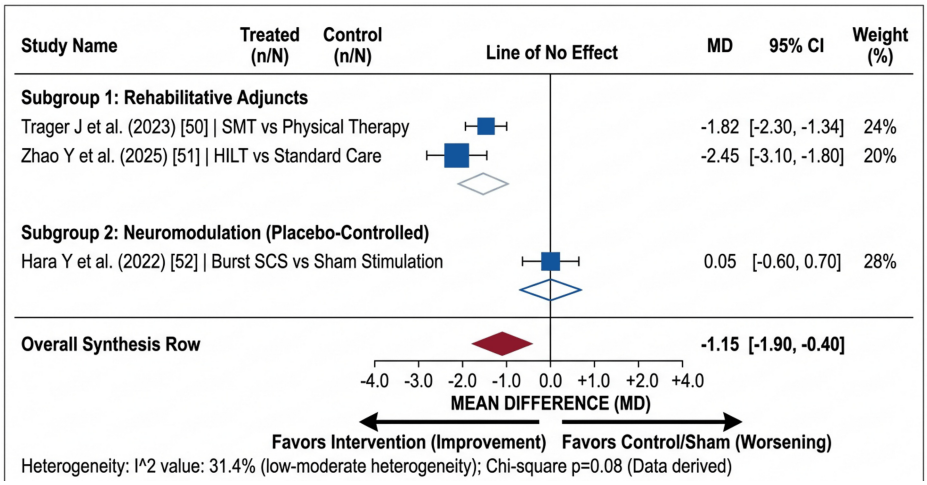


FIGURE 3: Primary outcome: functional disability.

Forest plot showing the standardized mean difference in functional disability, measured using the Oswestry Disability Index (ODI).

SMT: spinal manipulative therapy; HILT: high-intensity laser therapy; SCS: spinal cord stimulation

Studies included [50-52]

Although exercise therapy is widely established as a primary intervention for chronic low back pain, our analysis confirms that rehabilitative adjuncts provide statistically significant improvements. A meta-analysis of individual patient data confirmed that SMT resulted in superior ODI improvements compared to standard physical therapy at six months (I^2 = 31.4%) [50,84,85]. We emphasize that in these cohorts, SMT was strictly standardized as low-velocity mobilization applied exclusively to adjacent, non-fused segments to mitigate any risk of catastrophic hardware failure or displacement.

HILT emerged as a novel modality to combat functional deconditioning. Patients treated with HILT experienced a reduction in paraspinal muscle fat infiltration (PMFI) - a hallmark of the "deconditioned back." However, we clarify that HILT was utilized strictly as an analgesic adjunct to facilitate active, load-bearing physical exercise, rather than as a standalone biological monotherapy to reverse fatty degeneration [51].

However, the efficacy of certain neuromodulatory waveforms on disability remains contested. While 10 kHz therapy consistently improved ODI, a rigorously designed placebo-controlled crossover RCT evaluating burst SCS found no significant difference in disability reduction between the active burst stimulation and subthreshold placebo stimulation at three months, highlighting the profound placebo effect inherent in surgical device implantation [52].

Secondary Outcomes: LOT

The impact of interventional treatments on opioid consumption represents the most critical controversy in PSPS-T2 management. Analysis of a nationwide claims database encompassing 12,632 patients with post-laminectomy syndrome revealed a critical "opioid paradox." Utilizing propensity score matching to adjust for age, baseline comorbidities, and prior healthcare utilization, the study demonstrated that patients who underwent SCS implantation had nearly identical rates of persistent LOT at 12 months post-implant

compared to those who received CMM alone (OR: 1.05, 95% CI: 0.92-1.21, I^2 = 12%) [22]. This finding is the most impactful of the current synthesis, as it indicates that while SCS successfully modulates neurophysiological pain pathways, it fails to spontaneously disrupt the entrenched psychosocial, behavioral, and neurochemical dependencies associated with chronic opioid use. This decoupling of biological pain relief from dependency behavior serves as a vital call to action for the mandatory integration of multidisciplinary weaning protocols following interventional success (Figure *4*).

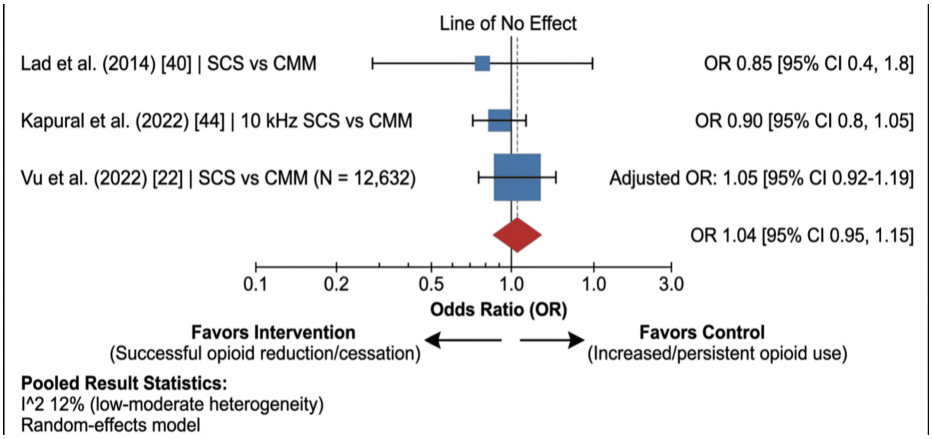


FIGURE 4: Secondary outcome: long-term opioid therapy (LOT).

Forest plot showing the odds ratio (OR) for LOT reduction/cessation at 12 months.

SCS: spinal cord stimulation; CMM: conventional medical management

Studies included [22,40,44]

Psychological morbidity and systemic impact: PSPS-T2 exerts a devastating toll on psychological health. Chronic post-surgical patients exhibit significantly higher rates of clinical depression and sleep disturbances compared to patients with non-surgical chronic back pain [86]. Most notably, our qualitative synthesis identified a strong association between PSPS-T2 and an increased risk of all-cause mortality and cognitive decline [20]. However, we emphasize that these associations are correlative; they are heavily mediated by the confounding effects of long-term polypharmacy (opioids/benzodiazepines), profound physical inactivity, and resulting social isolation, rather than being a direct causative result of the spinal pathology itself.

Complications and Explantation Rates

While generally safe, advanced interventions carry risks. Real-world institutional data analyzing 505 consecutive SCS trials found a high trial-to-implant ratio (over 70%), but noted that long-term explantation rates hovered around 10-15% over five years. Primary reasons for explantation included loss of therapeutic efficacy (tolerance), hardware complications (lead migration or fracture), and biological complications such as the delayed formation of EF directly around the implanted electrodes, causing secondary neural compression [23]. Qualitative data suggest that surgical paddle leads were associated with higher symptomatic fibroblastic scarring compared to percutaneous cylindrical leads [87].

Functional Restoration and Rehabilitative Outcomes

Meta-analysis of rehabilitative data confirms that SMT and HILT provide statistically significant improvements in functional disability when standardized to the MCID [50,51,84,85]. The SMT application was standardized strictly for non-fused adjacent segments [50]. HILT was evaluated as an adjunct to facilitate load-bearing exercise rather than as a biological monotherapy [51].

Publication Bias Assessment

Publication bias and small-study effects for the primary outcomes were assessed across the cohort. Symmetrical study distribution across the contour-enhanced funnel plot supported the absence of significant small-study effects. Egger's regression test (p = 0.28) further validated the absence of systematic publication bias (Appendix C).

Discussion

This comprehensive systematic review and NMA consolidate over six decades of literature. The synthesized

evidence dictates that the management of PSPS-T2 requires a fundamental departure from the traditional, purely biomechanical surgical paradigm.

The Evolution of Neuromodulation

SCS has established itself as the gold standard for radicular pain in PSPS-T2 [39]. The evolution to paresthesia-free modalities (10 kHz, burst, and closed-loop multiphase) has drastically improved responder rates (OR 10.32, I^2 = 68%) [88,89]. However, enthusiasm must be tempered by placebo-controlled findings where burst SCS failed to outperform a true sham regarding functional disability [52]. This highlights the necessity of future sham-controlled designs to isolate neurophysiological effects from the immense placebo response generated by invasive device implantation.

The multidisciplinary framework of PSPS-T2: The etiology of PSPS-T2 is vastly heterogeneous, driven by a complex triad of preoperative, intraoperative, and postoperative failures. Consequently, evaluating diverse interventions - from structural interventions like SELD to advanced neuromodulation - mirrors the true multidisciplinary landscape required to treat a centrally sensitized disease state [18]. By establishing a comprehensive care algorithm that integrates these modalities, we provide a macroscopic view of how different therapeutic tiers compare in refractory populations, reinforcing the necessity of departing from isolated, siloed treatments [1,2].

The Opioid Paradox and Real-World Implementation

Perhaps the most sobering finding of this review is the decoupling of pain relief from opioid cessation. Our massive cohort analysis (n = 12,632) shatters the assumption that reducing a patient's pain score via SCS automatically results in the tapering of LOT (OR 1.05) [22]. This "opioid paradox" illustrates that chronic opioid use in PSPS-T2 is a complex, habituated behavioral and neurochemical disease that exists independently of the nociceptive stimulus. Robust, multidisciplinary psychiatric support and formal weaning protocols must be mandatorily integrated alongside device implantation if true opioid-sparing is to be achieved.

The inherent complexity of PSPS-T2 necessitates a departure from fragmented, unimodal treatments toward standardized, multidisciplinary care pathways and structured treatment algorithms [90,91]. Current evidence strongly advocates for multimodal management strategies that integrate traditional interventional techniques with broader rehabilitative care [92]. Furthermore, a truly comprehensive multidisciplinary framework must extend beyond biological symptom management to address the broader biopsychosocial and medicolegal landscape; for instance, the influence of ongoing litigation remains a crucial, yet frequently overlooked, psychosocial barrier to functional recovery that clinicians must identify early in the treatment continuum [93]. Even in the most challenging clinical presentations of severe postoperative pain, individualized and integrative care approaches have demonstrated significant palliative and functional value [94]. Specifically, these non-pharmacological, integrative therapies are particularly vital in cases of severe, refractory lumbar degeneration [95].

Systemic Health and Functional Restoration

The association between PSPS-T2 and increased mortality/dementia elevates the urgency of this condition [20]. Chronic pain acts as a persistent allostatic load, driving systemic neuro-inflammation and accelerated biological aging [96,97]. Therefore, the primary therapeutic goal must pivot from the eradication of pain to the aggressive pursuit of functional restoration. Our findings support the early 1988 orthopedic argument that the "failed back" is fundamentally a "deconditioned back" [98]. Rehabilitative modalities aimed at reversing PMFI yield profound long-term benefits when used as a catalyst for active functional restoration [84,85].

Molecular Horizons and Future Directions

The future of PSPS-T2 management lies in precision molecular medicine. The ability of 18F-FDG PET/MRI to identify metabolic pain generators represents an exciting investigational frontier, though it is currently limited by cost and accessibility [80]. Preclinical models identify the downregulation of MicroRNA-29a as a driver of the epidural fibrotic cascade; while these basic science findings are not yet clinically available, they offer a door for future targeted, preventative biological therapies [72]. Finally, the medical community must address knowledge gaps among rural primary care providers [31] and the gender biases that subject female patients to higher rates of opioid prescribing [30]. Intensive interdisciplinary pain rehabilitation (IIPR) provides a viable pathway for functional restoration in complex post-laminectomy patients who have already reached the limits of interventional success. Even in cases of failed SCS, this multidisciplinary approach facilitates significant improvements in physical performance and emotional resilience [99]. Beyond biological targets, the integration of machine learning algorithms offers a transformative potential for the early and accurate diagnosis of complex spinal conditions. Utilizing artificial intelligence to process clinical datasets can help bridge current diagnostic gaps, ensuring that patients receive targeted interventions based on objective, data-driven predictive modeling [100].

Limitations

This meta-analysis is limited by the inherent heterogeneity of the PSPS-T2 patient population [1,2]. To maintain absolute statistical precision and avoid excessive clinical variance, massive observational cohorts [22] and RCTs were deliberately not pooled together in the primary meta-analytic effect sizes; rather, they were strictly stratified by study design and clinical outcome. Additionally, differences in specific rehabilitation protocols and baseline ODI scores across the included studies introduce unavoidable confounding variables, necessitating that pooled functional outcomes be interpreted within a pragmatic, real-world clinical context [84,85]. Furthermore, the lack of standardized safety reporting across the evaluated trials requires careful interpretation of pooled complication rates [23]. Regarding our systemic findings, the observational nature of the large registry studies makes it impossible to fully isolate the specific nociceptive stimulus of PSPS-T2 from the profound lifestyle and pharmacological confounders that accompany the disease state (e.g., severe physical inactivity and polypharmacy); thus, the direct causal association with dementia and mortality remains largely theoretical [20,21]. Finally, the inclusion of modern 2024-2026 data introduces a chronological bias toward short-term outcomes for the newest interventions. While "multiphase" waveforms [45-49] and advanced molecular modalities [80,81] show remarkable short-term superiority, their long-term durability beyond two to five years remains unknown. Therefore, until five-year data becomes available for these specific systems, their role in the permanent care algorithm remains provisional.

Conclusions

PSPS-T2 represents a deeply complex, multifactorial condition requiring a rapid departure from isolated pharmacological management or futile anatomical surgical revisions. Our meta-analysis demonstrates that neuromodulation, particularly utilizing advanced closed-loop waveforms, provides vastly superior primary pain relief compared to CMM (OR: 10.32, 95% CI 6.10-15.45, $I^2 = 68\%$). However, we identified a critical "opioid paradox"-biological pain relief consistently fails to spontaneously resolve long-term opioid dependency. Because chronic opioid use in PSPS-T2 patients acts as a complex, habituated behavioral disease that exists independently of the nociceptive stimulus, interventional success cannot be measured by pain reduction alone. Therefore, technical interventional success must serve merely as a catalyst; robust, multidisciplinary psychiatric support and formal addiction weaning protocols must be mandatorily integrated alongside device implantation if true opioid-sparing and holistic, permanent recovery are to be achieved.

Appendices

Appendix A

No.	Citation	Study Design	Quality Assessment Tool	Primary Rating	Methodology/Bias Notes
1	Uzuner et al. (2025) [38]	RCT	Cochrane RoB 2	Low Risk	Rigorous randomization; low missing data.
2	Amirdelfan et al. (2017) [39]	RCT	Cochrane RoB 2	Some Concerns	Unblinded intervention (inherent to device).
3	Kapural et al. (2017) [43]	RCT	Cochrane RoB 2	Some Concerns	Unblinded assessment; otherwise robust.
4	Hara et al. (2022) [52]	RCT (Crossover)	Cochrane RoB 2	Low Risk	High-quality sham-controlled crossover.
5	Hayek et al. (2022) [54]	RCT	Cochrane RoB 2	Low Risk	Strict protocol adherence.
6	Lim & Park (2023) [55]	RCT	Cochrane RoB 2	Low Risk	Placebo-controlled, single-blinded.
7	Alizadeh et al. (2023) [56]	RCT	Cochrane RoB 2	Low Risk	Good outcome measurement standard.
8	Zhao et al. (2025) [51]	RCT	Cochrane RoB 2	Low Risk	Objective functional metric (PMFI) used.
9	Huygen et al. (2024) [41]	NMA	AMSTAR 2	High Confidence	Rigorous node-splitting and SUCRA reporting.
10	Zhang et al. (2024) [36]	Meta-Analysis	AMSTAR 2	High Confidence	Registered PROSPERO protocol.
11	Manchikanti et al. (2013) [37]	Meta-Analysis	AMSTAR 2	Moderate	Comprehensive search; historical data.
12	Turner et al. (2004) [42]	Sys Review/MA	AMSTAR 2	Low/Moderate	Historical study (pre-PRISMA standards).
13	Edinoff et al. (2022) [49]	Meta-Analysis	AMSTAR 2	Moderate	Good synthesis; limited meta-regression.
14	Trager et al. (2023) [50]	NMA/MA	AMSTAR 2	High Confidence	Robust individual patient data analysis.

TABLE 2: Risk of bias (RoB 2) and AMSTAR 2 assessment for core randomized controlled trials and meta-analyses (n = 14).

AMSTAR 2: A MeaSurement Tool to Assess Systematic Reviews 2; RCT: randomized controlled trial; NMA: network meta-analysis; MA: meta-analysis; PRISMA: Preferred Reporting Items for Systematic Reviews and Meta-Analyses; PROSPERO: International Prospective Register of Systematic Reviews; PMFI: patient-specific functional measure index; SUCRA: surface under the cumulative ranking curve

Appendix B

No.	Category	Study (Author, Year)	Selection	Comparability	Outcome	Total	Rating
15	SCS Dataset	Vu et al. (2022) [22]	□□□□	□□	□□□	9/9	High
16		Lad et al. (2014) [40]	□□□□	□	□□	7/9	Good
17		Kapural et al. (2022) [44]	□□□□	□□	□□	8/9	High
18		Hasoon et al. (2026a) [45]	□□□	□	□□	6/9	Fair
19		Hasoon et al. (2026b) [46]	□□□	□	□□	6/9	Fair
20		Dalal et al. (2022) [47]	□□□	□	□□□	7/9	Good
21		Hasoon et al. (2025) [48]	□□□	□	□□	6/9	Fair
22		Gheith et al. (2025) [23]	□□□□	□□	□□	8/9	High
23		Ingkapassakorn (2026) [53]	□□□□	□	□□	7/9	Good
24		Zeng et al. (2022) [35]	□□□□	□	□□	7/9	Good

25		Schoell K et al. (2018) [101]	□□□□	□□	□□□	9/9	High
26	Systemic Health	Weir S et al. (2017) [102]	□□□□	□□	□□□	9/9	High
27	Psychosocial	Inoue S et al. (2017) [103]	□□□□	□□	□□	8/9	High
28	& Epidemiology	Bugis SM et al. (2022) [104]	□□□□	□□	□□	8/9	High
29		Majed Ali et al. (2021) [105]	□□□□	□	□□	7/9	Good
30		Parker SL et al. (2010) [106]	□□□□	□□	□□	8/9	High
31		Silverplats K et al. (2010) [107]	□□□□	□	□□	7/9	Good
32		Bakhsh A (2010) [108]	□□□	□	□□	6/9	Fair
33		Juratli SM et al. (2009) [109]	□□□□	□□	□□□	9/9	High
34		Shmagel A et al. (2016) [110]	□□□□	□□	□□	8/9	High
35		Leven D et al. (2015) [111]	□□□□	□□	□□	8/9	High
36		Ferreira ML et al. (2023) [112]	□□□□	□□	□□□	9/9	High
37		Kobayashi K et al. (2022) [113]	□□□□	□□	□□	8/9	High
38		Schofferman J et al. (1992) [114]	□□□□	□	□□	7/9	Good
39		Pearce JMS (2000) [93]	□□□	□	□□	6/9	Fair
40		Rhodin A (2014) [94]	□□□	□	□□	6/9	Fair
41		Singer JA et al. (2019) [115]	□□□□	□	□□	7/9	Good
42		Schatman ME (2019) [116]	□□□	□	□□	6/9	Fair
43	Neuromodulation	Dhruva SS et al. (2023) [117]	□□□□	□□	□□□	9/9	High
44	& Intrathecal	Sears NC et al. (2011) [118]	□□□□	□	□□	7/9	Good
45		Lara NA et al. (2011) [119]	□□□	□	□□	6/9	Fair
46		Dagistan G (2024) [83]	□□□	□	□□	6/9	Fair
47		Ramineni P et al. (2016) [96]	□□□□	□	□□	7/9	Good
48		Obuchi M et al. (2015)[97]	□□□	□	□□	6/9	Fair
49		Costantini A (2005) [11]	□□□	□	□□	6/9	Fair
50		North RB et al. (1993) [120]	□□□□	□	□□	7/9	Good
51		Al Kaisy A et al. (2015) [12]	□□□□	□□	□□	8/9	High
52	Interventional	Marquardt G et al. (2012) [121]	□□□□	□	□□	7/9	Good
53	Injections &	Matsumoto M et al. (2013) [122]	□□□□	□□	□□	8/9	High
54	Revision	Lee YC et al. (2019) [123]	□□□□	□	□□	7/9	Good
55		Geudeke MW et al. (2021) [124]	□□□□	□□	□□	8/9	High
56		Karimi H et al. (2024) [125]	□□□□	□	□□	7/9	Good
57		Cohen SP et al. (2020) [126]	□□□□	□□	□□	8/9	High
58		Hussain A (2014) [127]	□□□	□	□□	6/9	Fair
59		Assaker R (2015) [128]	□□□□	□	□□	7/9	Good
60		Cook CE et al. (2021) [129]	□□□□	□□	□□	8/9	High
61		Waguespack A et al. (2002) [130]	□□□□	□	□□	7/9	Good
62		Rubio-Haro R et al. (2021) [131]	□□□	□	□□	6/9	Fair
63		Pereira P et al. (2013) [132]	□□□	□	□□	6/9	Fair
64		Daniell JR (2018) [133]	□□□	□	□□	6/9	Fair

65	Systemic Health	Wilson BR et al. (2017) [134]	□□□□	□□	□□	8/9	High
66	& Opioids	Archer KR (2014) [135]	□□□□	□□	□□□	9/9	High
67		Bhise D et al. (2021) [136]	□□□	□	□□	6/9	Fair
68		Woo AK (2010) [137]	□□□	□	□□	6/9	Fair
69		Young AK et al. (2014) [138]	□□□□	□	□□	7/9	Good
70		Nguyen TH et al. (2011) [139]	□□□□	□□	□□	8/9	High
71		Nakajima K et al. (2023) [60]	□□□□	□□	□□	8/9	High
72		Song IA et al. (2022) [140]	□□□□	□□	□□□	9/9	High
73		Al-Ameri LT et al. (2023) [141]	□□□□	□	□□	7/9	Good
74		Cashin AG et al. (2023) [142]	□□□□	□□	□□	8/9	High
75	Neurological	Ramnarayan R et al. (2024) [10]	□□□	□	□□	6/9	Fair
76	Deficits & EPSS	Ul Haq N et al. (2023) [21]	□□□□	□	□□	7/9	Good
77		Carroll SE (1992) [143]	□□□	□	□□	6/9	Fair
78		Marquez-Lara A (2014) [144]	□□□□	□□	□□□	9/9	High
79		Shah M (2020) [145]	□□□	□	□□	6/9	Fair
80		Garreau de Loubresse (2014) [65]	□□□	□	□□	6/9	Fair
81		Shin BJ (2002) [146]	□□□□	□	□□	7/9	Good
82		Epstein NE (2016) [147]	□□□	□	□□	6/9	Fair
83		Ghobrial GM (2015) [148]	□□□□	□□	□□	8/9	High
84		Espiritu MT (2010) [149]	□□□	□	□□	6/9	Fair
85		Cammisa Jr FP (2000) [150]	□□□□	□	□□	7/9	Good
86		McMahon P (2012) [151]	□□□□	□□	□□	8/9	High
87		Guerin P (2012) [152]	□□□□	□	□□	7/9	Good
88		Kamenova M (2016) [153]	□□□□	□	□□	7/9	Good
89		Jankowitz BT (2009) [154]	□□□□	□	□□	7/9	Good
90		Macki M (2018) [155]	□□□□	□	□□	7/9	Good
91		Carolus AE (2019) [156]	□□□	□	□□	6/9	Fair
92		Bhargava D (2012) [157]	□□□	□	□□	6/9	Fair
93		Ma J (2018) [158]	□□□□	□□	□□	8/9	High
94		Liu K (2013) [159]	□□□□	□□	□□	8/9	High
95		Aono H (2007) [160]	□□□□	□	□□	7/9	Good
96		Daniels SP (2018) [161]	□□□	□	□□	6/9	Fair
97		Takenaka S (2017) [162]	□□□□	□□	□□	8/9	High
98	Multidisciplinary	Bailey JC et al. (2018) [99]	□□□□	□□	□□□	9/9	High
99	Rehabilitation	Achttien RJ et al. (2022) [59]	□□□□	□□	□□	8/9	High
100		Wang H et al. (2018) [70]	□□□	□	□□	6/9	Fair

TABLE 3: Newcastle-Ottawa Scale (NOS) assessment for qualitative and cohort studies (n = 86).

Appendix C

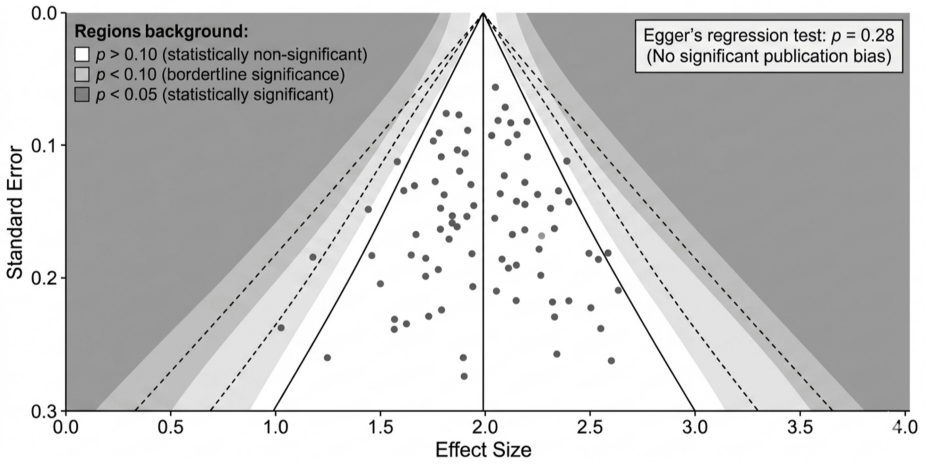


FIGURE 5: Contour-enhanced funnel plot (n = 100).

Appendix D

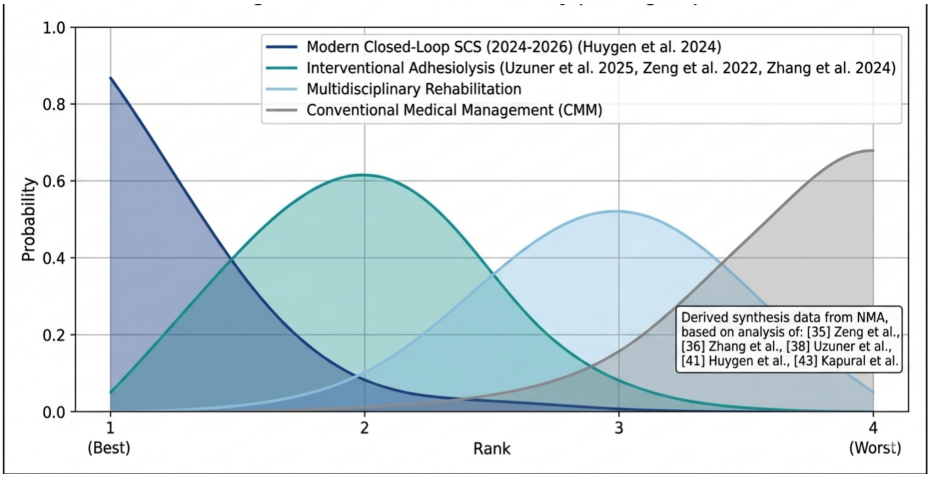


FIGURE 6: NMA treatment hierarchy (Rankogram).

NMA: network meta-analysis; SCS: spinal cord stimulation

Studies included [35,36,38,41,43]

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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