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The Association of Preoperative and Postoperative Serum Il-10 with Visual Analogue Scale (VAS), American Spinal Injury Association (ASIA) Score, and Modified Japanese Orthopaedic Association (MJOA) in CSM Patients Undergoing Anterior and Posterior Approaches at Adam Malik Hospital in 2025

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ABSTRACT

Introduction: Cervical spondylotic myelopathy (CSM) frequently requires surgical decompression, although early postoperative recovery remains heterogeneous despite adequate neural decompression. Interleukin-10 (IL-10), an anti-inflammatory cytokine, may contribute to differences in postoperative clinical outcomes.
Methods: This prospective cohort study investigated perioperative changes in serum IL-10 and their associations with early clinical outcomes following anterior or posterior cervical decompression.
Results: Forty-eight adults with CSM underwent surgery at H. Adam Malik General Hospital, including 11 treated through an anterior approach and 37 through a posterior approach. Serum IL-10 concentrations were measured using enzyme-linked immunosorbent assay within 24 hours preoperatively and 24–48 hours postoperatively. Pain intensity was assessed using the visual analogue scale (VAS), while neurological status was evaluated using the ASIA Impairment Scale (AIS) and modified Japanese Orthopaedic Association (mJOA) score. Serum IL-10 increased significantly in both groups (anterior, 1.55 to 5.53 pg/mL; posterior, 1.66 to 5.99 pg/mL; both $p < 0.001$), accompanied by significant reductions in VAS scores. Between-group differences in postoperative IL-10, VAS, and neurological grade were not significant ($p = 0.404$, 0.217, and 0.694, respectively). Across the cohort, postoperative IL-10 correlated negatively with VAS ($r = -0.308$; $p = 0.033$) and positively with neurological status assessed using mJOA/AIS ($r = 0.327$; $p = 0.023$).
Conclusion: These findings suggest that postoperative IL-10 levels are associated with early clinical status irrespective of surgical approach.

Keywords: Cervical spondylotic myelopathy; Interleukin-10; Surgical decompression; Modified Japanese Orthopaedic Association; Visual analogue scale

1. Introduction

Cervical spondylotic myelopathy (CSM) is the most frequent cause of non-traumatic spinal cord dysfunction in adults and the clinical expression of degenerative cervical myelopathy (DCM). Progressive narrowing of the cervical canal by osteophytes, disc herniation, ligamentum flavum hypertrophy and loss of disc height produces chronic mechanical compression and microvascular hypoperfusion of the cord, culminating in demyelination, conduction failure and neuronal loss [1], [2]. Its incidence rises steeply beyond the fifth decade, and the ageing of the population has made it an increasingly common indication for spinal surgery worldwide [3], [4].

Because CSM is progressive and does not resolve spontaneously, surgical decompression remains the only definitive treatment for patients with moderate or progressive deficits [5], [6]. Two corridors dominate practice: anterior cervical discectomy and fusion (ACDF) or corpectomy, which addresses ventral compression directly and restores lordosis, and posterior laminectomy or laminoplasty, which achieves broad decompression across multiple levels in the presence of adequate lordosis [7], [8]. Comparative series and meta-analyses generally report equivalent neurological recovery between the two corridors, with different complication profiles: dysphagia and pseudarthrosis after anterior surgery, axial neck pain and muscle atrophy after posterior surgery [9], [10], [11].

What neither corridor explains is the persistent variability of functional outcome after technically adequate decompression. Patients with comparable radiological compression and comparable operations recover to very different degrees, which implies that biological factors beyond the mechanical relief of compression shape recovery. The perioperative inflammatory response is the most plausible of these. Surgical trauma releases damage-associated molecular patterns that activate innate immunity, while reperfusion of the chronically ischaemic cord generates oxidative stress and a second wave of injury mediated by pro-inflammatory macrophages [12], [13].

Interleukin-10 (IL-10) is the principal counter-regulatory cytokine of this response. Produced by regulatory T cells, monocytes and M2 macrophages, it suppresses tumour necrosis factor-α, IL-1β and IL-6, downregulates antigen presentation, and restrains microglial activation [14], [15]. In experimental spinal cord injury, IL-10 reduces neuronal apoptosis, promotes white matter sparing and remyelination, and improves functional scores [16], [17], [18]. It also acts as an endogenous antinociceptive mediator by suppressing glial p38 MAPK signalling in the dorsal horn, limiting central sensitisation [19], [20].

Clinical evidence in degenerative cervical disease remains sparse. Karamian et al. observed that perioperative cytokine profiles relate to patient-reported outcomes after ACDF [21], and Vedantam et al. reported that serum protein biomarkers, including anti-inflammatory mediators, track neurological status in DCM [22]. A few studies have examined systemic cytokines after cervical decompression, but there have been limited studies concerned simultaneously with the

perioperative IL-10 trajectory, the surgical corridor, and validated pain and neurological instruments in the same cohort. Therefore, this research intends to characterise that relationship in a single prospective cohort. The objectives of this research are to quantify the change in serum IL-10 from before to after cervical decompression, to compare the postoperative IL-10 response and clinical status between anterior and posterior approaches, and to examine the association between postoperative IL-10 and pain and neurological status measured by the visual analogue scale (VAS), the ASIA Impairment Scale (AIS) and the modified Japanese Orthopaedic Association (mJOA) score.

2. Method

2.1. Study design and setting

This was an analytical observational study with a prospective cohort design, conducted in the Department of Neurosurgery, H. Adam Malik General Hospital, Medan, Indonesia. Patients were recruited consecutively during the study period, and each participant served as their own control for the perioperative comparison.

2.2. Participants

Eligible participants were adults aged 18 years or older with CSM diagnosed on combined clinical and radiological grounds and scheduled for operative decompression through either an anterior approach (ACDF) or a posterior approach (laminectomy or laminoplasty), who consented to the full study protocol. Patients were excluded if they had a diagnosis of autoimmune disease, active systemic infection or another chronic inflammatory disorder, had received immunosuppressants, corticosteroids or systemic anti-inflammatory agents within 14 days before surgery, had a history of non-degenerative spinal cord injury or another neuromuscular disease, had a spinal neoplasm or any other condition capable of altering cytokine concentrations, or were unable to complete clinical assessment because of communicative or cognitive impairment. Consecutive sampling was used until the minimum sample size was reached. The choice of surgical corridor was made by the operating surgeon on clinical and radiological grounds and was not influenced by the study.

2.3. Sample size

The required sample was calculated for a two-tailed Pearson correlation between two continuous variables, assuming $\alpha = 0.05$, power of 80 % and a minimum clinically meaningful correlation of $r = 0.4$, which yielded 44 subjects. Allowing for 10 % attrition, the target was set at 48 patients, and 48 were enrolled and analysed.

2.4. Blood sampling and IL-10 measurement

Venous blood (3–5 mL) was drawn within 24 hours before surgery and again 24 to 48 hours after decompression. Samples were centrifuged, and serum was separated and stored until batch

analysis. Serum IL-10 was quantified with a commercial quantitative human IL-10 enzyme-linked immunosorbent assay (ELISA) kit according to the manufacturer's instructions, and results are expressed in pg/mL.

2.5. Clinical outcome measures

Pain intensity was recorded on the VAS, a 100 mm horizontal line anchored at no pain and worst imaginable pain, reported as an integer from 0 to 10 [23]. Neurological status was graded with the AIS (grades A to E) and with the mJOA score, which assesses upper limb motor function, lower limb motor function and gait, sensory function and bladder function, with a maximum of 18 points; scores of 15–17 denote mild, 12–14 moderate and 11 or less severe myelopathy [24], [25]. Assessments were made before surgery, on the second postoperative day for VAS, and on the seventh postoperative day for AIS and mJOA. For analysis, mJOA was handled as the three ordinal severity strata defined above.

2.6. Statistical analysis

Data were checked for completeness, cleaned and analysed with IBM SPSS Statistics. Normality was assessed with the Shapiro–Wilk test and homogeneity of variance with the Levene test. Continuous variables are presented as mean ± standard deviation and ordinal variables as median with interquartile range or as frequency and percentage. Perioperative change in IL-10 was tested with the paired t-test, and change in VAS and in neurological grade with the Wilcoxon signed-rank test. Between-group comparison used the independent-samples t-test for IL-10, the Mann–Whitney U test for ordinal variables and the Fisher exact test for sex. The association between postoperative IL-10 and clinical status was examined with the Spearman rank correlation, applied both to absolute postoperative scores and to the change from baseline. A two-tailed p value below 0.05 was considered significant.

2.7. Ethical considerations

The protocol was approved by the Health Research Ethics Committee, Faculty of Medicine, Universitas Sumatera Utara. All participants received an explanation of the aims, procedures and risks of the study and gave written informed consent. Participation was voluntary and could be withdrawn at any time without consequence for clinical care. Data were coded, and identifying information was accessible only to the investigators.

3. Results and Discussion

3.1. Baseline characteristics

Forty-eight patients were analysed, 11 in the anterior group and 37 in the posterior group. Their baseline characteristics are shown in Table 1. Mean age was 55.00 ± 8.07 years in the anterior group and 54.19 ± 9.29 years in the posterior group ($p = 0.795$), and the cohort overall was 54.38

± 8.95 years old, with a range of 35 to 69 years. Men made up 27 of 48 patients (56.3 %). Sex distribution, preoperative IL-10, preoperative VAS and preoperative neurological grade did not differ significantly between the groups, so the two arms were comparable at entry on every measured variable.

Table 1. Baseline characteristics by surgical approach.

Variable	Anterior (n = 11)	Posterior (n = 37)	p	Test
Age, years (mean ± SD)	55.00 ± 8.07	54.19 ± 9.29	0.795	[a]
Sex, male, n (%)	4 (36.4)	23 (62.2)	0.174	[b]
Sex, female, n (%)	7 (63.6)	14 (37.8)		
Preoperative IL-10, pg/mL	1.55 ± 0.52	1.66 ± 0.81	0.990	[c]
Preoperative VAS, median (IQR)	4 (4–5)	4 (4–5)	0.572	[c]
Preoperative AIS C, n (%)	0 (0.0)	7 (18.9)	0.847	[c]
Preoperative AIS D, n (%)	6 (54.5)	8 (21.6)		
Preoperative AIS E, n (%)	5 (45.5)	22 (59.5)		

[a] independent-samples t-test; [b] Fisher exact test; [c] Mann–Whitney U test. IQR, interquartile range.

3.2. Perioperative change in IL-10 and clinical scores

Serum IL-10 rose sharply after decompression in both groups (Table 2). In the anterior group it increased from 1.55 ± 0.52 to 5.53 ± 1.34 pg/mL and in the posterior group from 1.66 ± 0.81 to 5.99 ± 1.65 pg/mL, both $p < 0.001$, a mean fold change of about four across the cohort. Every patient recorded a lower VAS after surgery than before it, with the median falling from 4 to 2 in both groups (anterior $p = 0.001$; posterior $p < 0.001$). Neurological grade improved in 11 of 48 patients and deteriorated in none. The improvement was statistically significant in the posterior group (8 of 37 patients; $p = 0.005$) but not in the anterior group, where only 3 of 11 patients changed grade ($p = 0.250$).

The last point deserves emphasis rather than concealment. The absence of a significant grade change after anterior surgery reflects a ceiling effect and low event numbers, not a failure of decompression: 5 of the 11 anterior patients were already AIS E before surgery and could not improve on this scale, and no anterior patient was worse afterwards. Ordinal grading with three strata is comparatively insensitive to the incremental gains that the full 18-point mJOA would capture, and the small anterior subgroup leaves little power to detect them.

Table 2. Change in serum IL-10 and clinical scores from before to after surgery.

Variable	Anterior pre	Anterior post	p	Posterior pre	Posterior post	p
IL-10, pg/mL	1.55 ± 0.52	5.53 ± 1.34	< 0.001[a]	1.66 ± 0.81	5.99 ± 1.65	< 0.001[a]
VAS, median (IQR)	4 (4–5)	2 (1–2)	0.001[b]	4 (4–5)	2 (1–2)	< 0.001[b]
AIS C / mJOA severe, n	0	0	0.250[b]	7	2	0.005[b]
AIS D / mJOA moderate, n	6	3		8	10	
AIS E / mJOA mild, n	5	8		22	25	

[a] paired t-test; [b] Wilcoxon signed-rank test. AIS, ASIA Impairment Scale; IQR, interquartile range; mJOA, modified Japanese Orthopaedic Association; VAS, visual analogue scale.

3.3. Comparison between surgical approaches

Postoperative values did not differ significantly between the two corridors on any measure (Table 3). Mean IL-10 was numerically higher after posterior surgery, 5.99 ± 1.65 against 5.53 ± 1.34 pg/mL, but the difference was not significant (p = 0.404), and the same held for postoperative VAS (p = 0.217) and postoperative neurological grade (p = 0.694). Two posterior patients remained AIS C, whereas no anterior patient occupied that stratum either before or after surgery.

Table 3. Postoperative comparison between anterior and posterior approaches.

Variable	Anterior (n = 11)	Posterior (n = 37)	p	Test
Postoperative IL-10, pg/mL	5.53 ± 1.34	5.99 ± 1.65	0.404	[a]
Postoperative VAS, median (IQR)	2 (1–2)	2 (1–2)	0.217	[b]
Postoperative AIS C, n (%)	0 (0.0)	2 (5.4)	0.694	[b]
Postoperative AIS D, n (%)	3 (27.3)	10 (27.0)		
Postoperative AIS E, n (%)	8 (72.7)	25 (67.6)		

[a] independent-samples t-test; [b] Mann–Whitney U test. AIS grades correspond one-to-one with mJOA severity strata in this cohort.

3.4. Association between IL-10 and clinical status

Across the whole cohort, postoperative IL-10 correlated inversely with postoperative VAS (r = -0.308; p = 0.033) and directly with postoperative mJOA/AIS grade (r = 0.327; p = 0.023), as shown in Table 4. Patients with a stronger anti-inflammatory response therefore reported less pain and occupied a better neurological stratum in the first postoperative week. When the same cytokine value was correlated with the change in score rather than the absolute postoperative score, neither association reached significance (ΔVAS r = -0.161, p = 0.273; Δgrade r = -0.061, p = 0.681). The correlations are consequently best read as concurrent associations between two postoperative measurements, not as evidence that IL-10 drives the magnitude of recovery.

Table 4. Spearman correlation of postoperative serum IL-10 with clinical measures.

Clinical measure	r	p	Interpretation
Postoperative VAS	-0.308	0.033	weak, inverse
Postoperative mJOA severity	0.327	0.023	weak, direct
Postoperative AIS grade	0.327	0.023	weak, direct
Change in VAS (post – pre)	-0.161	0.273	not significant
Change in AIS/mJOA grade	-0.061	0.681	not significant

AIS, ASIA Impairment Scale; mJOA, modified Japanese Orthopaedic Association; VAS, visual analogue scale.

3.5. Discussion

The demographic profile of this cohort, with a mean age in the mid-fifties and a male predominance of 56.3 %, matches the epidemiology of degenerative cervical myelopathy described by Badhiwala et al. and Nouri et al., in whom incidence rises sharply through the fifth and sixth decades [1], [3]. The male predominance has been attributed both to occupational

loading and to a congenitally narrower sagittal canal diameter, which lowers the reserve available before compression becomes symptomatic [1].

The fourfold rise in serum IL-10 after decompression is the most robust finding of this study, reproduced in both surgical corridors with very small p values. It is best explained by the surgical stress response: incision, muscle dissection and bone work release damage-associated molecular patterns that activate innate immunity, and the counter-regulatory limb of that response raises anti-inflammatory cytokines in parallel with pro-inflammatory ones [12], [26]. Tu et al. describe this balance explicitly in myelopathic patients, in whom IL-10 restrains TNF-α and IL-1β synthesis and supports perilesional neuronal survival [26]. The rise is therefore an expected physiological signal rather than a marker of complication.

Contrary to the prevailing expectation, the posterior approach did not generate a significantly greater IL-10 response than the anterior approach. Extensive subperiosteal dissection of the semispinalis cervicis and multifidus during posterior surgery has been shown to raise muscle enzymes and inflammatory markers more than ventral exposure does [11], [27], and a larger difference might have been expected here. Three explanations are plausible. First, the anterior group was small (n = 11), which limits power to detect a modest difference. Second, the single sampling window of 24 to 48 hours may straddle rather than capture the peak of the counter-regulatory response, which is known to be time-dependent. Third, IL-10 is a systemic mediator with a broad dynamic range, and its concentration may saturate above a threshold of tissue injury that both corridors exceed. The safest reading of the present data is that both approaches trigger a comparable systemic anti-inflammatory response.

The correlations between postoperative IL-10 and clinical status are consistent with the neuroimmunological role of the cytokine but must be interpreted with restraint. IL-10 suppresses glial p38 MAPK signalling in the dorsal horn, reducing TNF-α and IL-1β production and thereby limiting central sensitisation, and it also modulates nociceptor TRPV1 expression [19], [20]. This provides a coherent mechanism for the inverse relationship with VAS. Similarly, the polarisation of macrophages from the M1 to the M2 phenotype under IL-10 influence limits reperfusion injury, spares white matter and supports remyelination in experimental models, which would predict a better neurological stratum [16], [17], [18]. However, the correlation coefficients here lie between 0.31 and 0.33, denoting a weak association that explains roughly a tenth of the variance, and the association was present only for absolute postoperative scores, not for the change from baseline. IL-10 is therefore associated with, but far from determinative of, early postoperative status.

This restraint is consistent with prognostic work in DCM. Khosravi et al. found outcome after decompression to be multifactorial [28], and Tetreault et al. emphasised the weight of preoperative symptom duration, baseline severity and irreversible cord change such as T2 hyperintensity [29]. Reperfusion of the chronically ischaemic cord can also be injurious when the pro-inflammatory limb dominates [13]; in the present cohort the anti-inflammatory limb was

prominent and no patient deteriorated, but this cannot be generalised to cohorts with different cytokine profiles. Taken together with the absent between-group difference, these findings suggest that the biological response to surgery, rather than the surgical corridor itself, is the more informative axis of variation in the early postoperative period.

Several limitations qualify these results. The design was observational, and the two arms were markedly unequal in size, since the choice of corridor followed clinical indication; although baseline variables were statistically comparable, indication bias cannot be excluded, and the seven preoperative AIS C patients were all in the posterior group [30], [31]. A single anti-inflammatory analyte was measured, without a pro-inflammatory panel such as IL-6 or TNF-α to establish the ratio that governs the net immune balance, and without cerebrospinal fluid sampling to characterise the local compartment. In this cohort mJOA severity strata mapped one-to-one onto AIS grades, so the two instruments did not contribute independent information, and collapsing the 18-point mJOA into three categories discarded resolution that would have improved sensitivity to change. Finally, follow-up extended only to the seventh postoperative day, which cannot address axonal regeneration, delayed axial neck pain after posterior surgery, or the durability of the observed improvements.

4. Conclusion and Future Research

Cervical decompression for CSM elicited a marked and reproducible rise in serum IL-10, approximately fourfold, in both anterior and posterior corridors, accompanied by significant reduction in pain in both groups and by significant improvement in neurological grade in the posterior group. The two approaches did not differ significantly in postoperative IL-10, pain or neurological grade, indicating that either corridor, correctly indicated, produces comparable early results and comparable systemic anti-inflammatory activation. Across the cohort, a higher postoperative IL-10 concentration was associated with lower pain scores and better neurological grade, although the association was weak and did not extend to the magnitude of change from baseline.

These findings advance the field by shifting attention from the surgical corridor to the host inflammatory response as a source of outcome variability, while setting a realistic ceiling on what a single cytokine can predict. Future work should measure paired pro-inflammatory and anti-inflammatory analytes at several time points to derive the IL-6/IL-10 or TNF-α/IL-10 ratio, should retain the full 18-point mJOA and the Hirabayashi recovery rate as continuous outcomes, should balance group sizes through prospective stratification or a randomised design where equipoise exists, and should extend follow-up to at least twelve months to determine whether the early cytokine signal carries any durable prognostic value.

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Conflict of Interest

The authors declare that they have no conflict of interest. This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

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