ORIGINAL ARTICLE

Adiposity, lumbosacral lordosis and systemic inflammation in chronic low back pain: An analytical cross-sectional study

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ABSTRACT

Background and Aim: Chronic low back pain (cLBP) reflects spinal mechanics and low-grade inflammation, yet most studies treat body mass index (BMI) only as a baseline descriptor. To our knowledge, few studies have examined adiposity, lumbosacral alignment, inflammatory markers, pain intensity and disability side by side in a single cLBP cohort, which is the gap the present analysis sets out to fill.

Methods: Forty-two adults with non-specific mechanical cLBP were assessed in one visit. BMI was recorded and the lumbosacral lordotic angle (LLA) measured on sagittal T2 MRI by the Cobb method. Neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR) and erythrocyte sedimentation rate (ESR) came from one venous sample. Pain used the Numerical Pain Rating Scale (NPRS) and disability the Indonesian Oswestry Disability Index (ODI). Spearman correlation was primary (Pearson reported alongside); subgroups (BMI <25 vs ≥25; men vs women) were compared with Mann-Whitney U.

Results: Mean BMI was 25.2 kg/m²; 47.6% overweight or obese. BMI correlated positively with disability (Spearman r=0.37, *p*=0.016). A modest association with ESR appeared only in the Pearson analysis (r= 0.38,

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p=0.012) and did not hold under the primary Spearman test (r=0.21, *p*=0.183). Pain and LLA links were positive but non-significant; NLR and MLR were unrelated to BMI. Above BMI 25, median ODI, NPRS and ESR were higher but non-significant. ESR was far higher in women than men (median 17 vs 8 mm/h; *p*=0.002).
Conclusion: Higher BMI tracked with greater disability, whereas its link with ESR was inconsistent across statistical methods and should be read as tentative; the inflammatory ratios were unmoved. Adiposity is an inexpensive, modifiable correlate alongside spinal alignment, not a replacement; ESR should be interpreted by sex. The modest sample (n=42) may have limited the power to detect the weaker associations; longitudinal work is needed before clinical use. (www.actabiomedica.it)

Key words: chronic low back pain, body mass index, adiposity, lumbosacral lordosis, erythrocyte sedimentation rate, Oswestry Disability Index

Introduction

Chronic low back pain (cLBP) pain between the lower costal margin and the gluteal folds persisting beyond twelve weeks has held its place as the leading global contributor to years lived with disability through successive Global Burden of Disease reports (1,2). It is rarely traceable to one lesion. The clinical picture instead grows out of several overlapping strands: degenerative change in the discs and facet joints, altered loading of the spine, a low-grade systemic immune signal, psychosocial strain, and individual differences in how pain is processed (2,3). Body weight cuts across more than one of those strands, which is part of what makes it interesting. A large meta-analysis by Shiri and colleagues found that overweight and obese adults carry a higher prevalence and incidence of low back pain, with the strongest link seen for care-seeking and for chronic, rather than transient, pain (4). Two mechanisms are usually invoked. The first is mechanical: extra mass, and abdominal mass in particular, shifts the line of gravity forward and changes how axial load is delivered into the lumbosacral junction, which can accentuate the lordotic curve and load the posterior elements (4,5). The second is metabolic. Adipose tissue is no longer regarded as inert storage; it behaves as an endocrine and immune organ, releasing leptin, interleukin-6, tumour necrosis factor-α and other mediators that raise the circulating inflammatory tone as fat mass climbs (6-8). Both routes the biomechanical and the inflammatory could feed the peripheral nociceptive input that, against a background of central sensitisation, keeps chronic pain going. The structural side of this story is captured well by the lumbosacral lordotic angle (LLA), the Cobb-method angle between the superior endplate of L1 and that of S1 on sagittal imaging (9). Both flattened and accentuated curves have been tied, in clinical and cadaveric work, to uneven disc loading and compensatory muscle activity (10,11). The inflammatory side is often probed with inexpensive haematology indices the neutrophil-to-lymphocyte ratio (NLR), the monocyte-to-lymphocyte ratio (MLR) and the erythrocyte sedimentation rate (ESR) which have been examined as surrogates of systemic inflammatory burden in disc degeneration and other spinal disorders, with mixed results (12-14). What is less often done is to place body mass at the centre of the analysis and ask whether it threads through both axes at once. In a companion report from the same cohort, we examined the LLA and these three markers as direct correlates of pain and disability, and found that an abnormal angle and a raised ESR carried most of the signal. Here we turn the lens around. Taking BMI as the organising variable, we set out to describe how adiposity relates, within one well-characterised Indonesian cLBP cohort, to lumbosacral alignment, to the three inflammatory markers, and to pain and disability and, as a secondary question, whether the inflammatory profile differs by sex. Our intention is descriptive and hypothesis-generating rather than confirmatory.

Materials and methods

Study design and setting

This was an analytical, cross-sectional observational study conducted at Dr. Wahidin Sudirohusodo General Hospital, Hasanuddin University Teaching Hospital, and their affiliated locations in Makassar, South Sulawesi. Recruitment occurred from October 2025 to March 2026 at the outpatient neurology and pain clinics. The reporting adheres to the STROBE standard for cross-sectional studies (15). The cohort, recruitment period, and primary metrics are consistent with our supplementary study of the same patients; the two publications tackle distinct enquiries and are designed to be read in conjunction. The companion research focuses on the lumbosacral lordotic angle as its main variable, while the current analysis is structured around body mass index and incorporates comparisons between lean and higher adiposity, as well as sex differences, as detailed below. To prevent repetition, the erythrocyte sedimentation rate is examined solely in relation to body mass and sex, whereas its correlation with clinical outcomes is detailed in the accompanying study.

Participants

Eligible participants were people aged 23 to 79 years with non-specific cLBP of mechanical origin (symptoms persisting for a minimum of twelve weeks) who underwent a lumbosacral MRI as part of standard therapy. We excluded individuals with suspected or proven spinal cancer or infection, prior lumbar surgery, spondyloarthropathy or other inflammatory arthritis, active or recent (<4 weeks) febrile illness, haematological disorders, ongoing corticosteroid or immunosuppressive medication, and pregnancy. Consecutive sampling was employed. In a correlational design anticipating a modest effect ($r \approx 0.45$), with a two-sided $\alpha = 0.05$ and power of 0.80, the minimum sample size required was 36; we enrolled 42 to accommodate subgroup analysis (16).

Data collection and measurements

Following consent, each participant underwent a systematic history and examination, along with a single peripheral venous blood sample, which was processed on the same day at the ISO 15189-accredited hospital laboratory. Height and weight were assessed during the patient's visit when they were attired in light clothing and barefoot, with BMI computed as weight in kilos divided by height in meters squared. The WHO cut-offs established are as follows: underweight is defined as below 18.5, normal weight ranges from 18.5 to 24.9, overweight is classified between 25.0 and 29.9, and obesity is designated as 30.0 kg/m² or more. For the subgroup analysis, we categorised the cohort into a lean group (BMI < 25) and a higher-adiposity group (BMI ≥ 25). The LLA was quantified on the optimal mid-sagittal T2-weighted imaging utilising the Cobb method, defined as the angle between lines drawn at the superior endplates of L1 and S1, employing RadiAnt DICOM Viewer (Medixant, Poznań, Poland). Measurements were conducted by three senior neurology residents under the oversight of three consultant neurologists (MYA, LTL, YG, each possessing over ten years of experience) and subsequently verified independently by a consultant neuroradiologist (JB) who was blinded to the clinical data; discrepancies exceeding 3° were resolved by consensus. An angle under 30° was designated as hypolordosis, 30–50° as normal, and above 50° as hyperlordosis (17). The Neutrophil-to-Lymphocyte Ratio (NLR) and Monocyte-to-Lymphocyte Ratio (MLR) were calculated using the percentages of neutrophils and monocytes divided by the percentage of lymphocytes; the Erythrocyte Sedimentation Rate (ESR) was assessed via the Westergren method and expressed in mm/h. Pain experienced throughout the prior week was assessed using the 11-point Numerical Pain Rating Scale (NPRS), categorising intensity as mild (1-3), moderate (4-6), or severe (7-10). Disability was evaluated using the Indonesian-validated Oswestry Disability Index (ODI v2.1a) and represented as a percentage (18).

Statistical analysis

Analyses were conducted using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY). Continuous data are presented as mean ± SD when normally distributed and as median (interquartile range, IQR) otherwise; categorical variables are

reported as counts and percentages. The Shapiro-Wilk test was employed to assess normality. Due to the non-normal distribution of NPRS, ODI, NLR, MLR, and ESR, Spearman rank correlation was utilised as the principal method for continuous pairs, with Pearson correlation given in instances where a linear relationship was justifiable. The Mann-Whitney U test was employed to compare BMI subgroups (lean versus higher-adiposity) across the two sexes. A two-tailed p-value of less than 0.05 was considered significant. Considering the exploratory objective and the limited sample size, we did not implement formal corrections for multiple comparisons, although we interpreted borderline results with caution.

Results

Participant characteristics and body-composition profile

Forty-two adults completed the protocol, with no missing values for any primary variable. Women were slightly in the majority (23 of 42, 54.8%) and the mean age was 49.9 ± 14.6 years, spanning 23 to 79. Most were still in work at recruitment (59.5%). On body composition the cohort sat, on average, just over the overweight threshold: mean BMI 25.2 ± 4.4 kg/m² (range 17.8-35.9). Just under half were of normal weight (20 patients, 47.6%), with 16 (38.1%) overweight, 4 (9.5%) obese and 2 (4.8%) underweight (Table 1). Splitting the cohort at a BMI of 25 produced two groups of similar size 22 lean and 20 higher-adiposity patients which we used for the subgroup contrasts below. Clinically, disability clustered in the moderate ODI band, as is usual for a tertiary pain clinic, and pain fell mostly in the moderate-to-severe range.

Distribution of body mass, lumbosacral lordosis and inflammatory markers

Descriptive statistics for the continuous variables are set out in Table 2. BMI and the LLA were both close to normally distributed (Shapiro-Wilk p=0.110 and p= 0.115), whereas the inflammatory markers and the two clinical scores were right-skewed and failed the normality test (all p<0.01). ESR carried the longest tail a median of 13 mm/h but a maximum of 58 and NLR behaved similarly, with most values near 2.0 and a single reading as high as 10.0. This skew is the reason Spearman correlation was chosen as the primary test, and it matters again when the Spearman and Pearson estimates for ESR diverge later on.

Body mass index and lumbosacral alignment

The relationship between BMI and the LLA was positive but loose. Heavier patients tended to sit at higher lordotic angles, yet the trend stayed well short of significance (Spearman r=0.15, p=0.333; Pearson r=0.23, p=0.136; Figure 1A). The direction is what one would predict from the mechanical argument forward shift of load with rising abdominal mass but the strength of the link in our data is modest, and a good part of the LLA spread clearly owes nothing to body mass.

Table 1. Demographic, body-composition and clinical characteristics of the study participants (n = 42).

Variable	Category	n	%
Sex	Female	23	54.8
	Male	19	45.2
Working status	Working	25	59.5
	Not working	17	40.5
BMI category	Underweight (< 18.5)	2	4.8
	Normal (18.5-24.9)	20	47.6
	Overweight (25.0-29.9)	16	38.1
	Obese (≥ 30.0)	4	9.5
Adiposity group	Lean (BMI < 25)	22	52.4
	Higher (BMI ≥ 25)	20	47.6
NPRS category	Mild (1-3)	8	19.0
	Moderate (4-6)	21	50.0
	Severe (7-10)	13	31.0
ODI category	Moderate (21-40%)	34	81.0
	Severe (41-60%)	7	16.7
	Crippled (61-80%)	1	2.4

Abbreviations: BMI, body mass index; NPRS, Numerical Pain Rating Scale; ODI, Oswestry Disability Index. Percentages are rounded to one decimal place.

Table 2. Distribution of the continuous variables (n = 42).

Variable	Mean	SD	Median	Min-Max	S-W $_p$
BMI (kg/m²)	25.22	4.35	24.96	17.8-35.9	0.110
Age (years)	49.88	14.60	48.5	23-79	0.536
LLA (degrees)	45.02	10.90	45.05	10.5-71.6	0.115
NLR	2.34	1.61	2.00	0.7-10.0	< 0.001
MLR	0.21	0.12	0.20	0.09-0.80	< 0.001
ESR (mm/h)	17.17	12.86	13.0	4-58	< 0.001
NPRS	5.33	2.03	5.0	2-10	0.009
ODI (%)	33.55	9.84	31.0	22-66	< 0.001

S-W *p*, Shapiro-Wilk *p*-value for normality. *Abbreviations* as in Table 1; LLA, lumbosacral lordotic angle; NLR, neutrophil-to-lymphocyte ratio; MLR, monocyte-to-lymphocyte ratio; ESR, erythrocyte sedimentation rate.

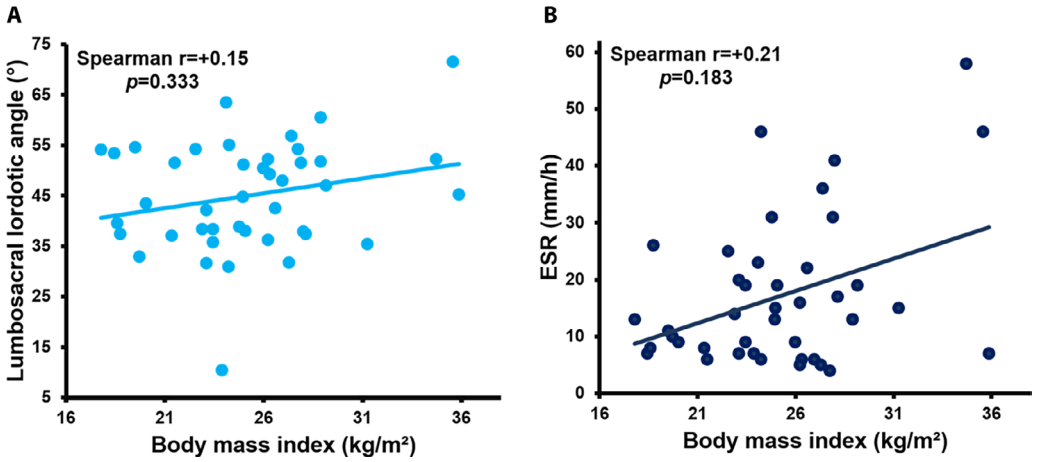

Figure 1. Body mass index against (A) the lumbosacral lordotic angle and (B) the erythrocyte sedimentation rate. Each point is one participant; the line is the least-squares fit and the inset gives the Spearman statistic (n = 42).

Body mass index and the systemic inflammatory markers

Of the three markers, only ESR moved with body mass to any convincing degree. The Pearson correlation between BMI and ESR was moderate and significant (r= 0.38, *p*=0.012), although the rank-based Spearman estimate was weaker and not significant (r=0.21, *p*=0.183), a gap that again reflects the long ESR tail (Figure 1B). The two leukocyte-derived ratios were essentially flat against BMI: NLR (Spearman r=0.05, *p*=0.763) and MLR (Spearman r=0.004, *p*=0.979) showed no meaningful relationship in either direction (Table 3). In other words, the inflammatory signal that adiposity carried in this cohort was confined to the acute-phase marker and did not reach the differential-count ratios.

Body mass index, pain intensity and functional disability

This is where the clearest finding sat. BMI correlated positively and significantly with disability: patients with a higher body mass reported higher ODI scores (Spearman r=0.37, *p*=0.016; Figure 2A), the Pearson estimate sitting just at the margin (r=0.30, *p*=0.055). The relationship with pain pointed the same way but was weaker and not significant (Spearman r=0.18, *p*=0.256; Figure 2B). Read together, the pattern suggests that

body mass tracked the functional consequences of cLBP more closely than the raw pain rating a dissociation that is worth keeping in mind, since disability and pain intensity are not interchangeable outcomes.

Lean versus higher-adiposity subgroups: a categorical view

Dichotomising at a BMI of 25 told a consistent but quieter story (Table 4, Figure 3). On every clinical and inflammatory measure the higher-adiposity group sat above the lean group a higher median ODI (34.3% vs 29.0%), a higher NPRS (5.5 vs 5.0) and a higher ESR (15.5 vs 12.0 mm/h) but none of these contrasts reached significance, with the ODI difference coming closest (Mann–Whitney p=0.111). The LLA followed the same upward direction (48.6° vs 40.9°, p=0.326). The lesson is mostly statistical: cutting a continuous variable into two blunt categories discards information, and in a sample of this size the graded BMI-ODI association of Section 3.5 is the more trustworthy reading than any threshold contrast.

Table 3. Correlation of body mass index with lumbosacral alignment, inflammatory markers and clinical outcomes.

Variable pair	Spearman r	p	Pearson r	p
BMI vs LLA	+0.153	0.333	+0.234	0.136
BMI vs NLR	+0.048	0.763	+0.184	0.244
BMI vs MLR	+0.004	0.979	+0.129	0.415
BMI vs ESR	+0.210	0.183	+0.383	0.012 *
BMI vs NPRS	+0.179	0.256	+0.174	0.272
BMI vs ODI	+0.369	0.016 *	+0.298	0.055

Spearman is the primary test given the non-normal distributions; Pearson is shown for comparison. * p<0.05.

Sex as a modifier of the inflammatory profile

A clear sex difference surfaced in just one place the ESR. Women had markedly higher values than men (median 17 vs 8 mm/h; Mann–Whitney U=94, p=0.002), the single strongest group contrast in the whole analysis (Table 5, Figure 4). Women also tended to carry a slightly higher BMI (median 26.0 vs 24.1 kg/m², p=0.090), so the heavier ESR readings in the female group sit alongside, and are hard to disentangle from, both their greater adiposity and the well-recognised tendency for ESR reference values to run higher in women on physiological grounds. What did not differ by sex was the clinical burden itself: men and women reported almost identical pain (median NPRS 5 in both) and disability (ODI 31% vs 32%, p=0.761), and their lordotic angles and leukocyte ratios were comparable. The sex signal, then, is real but narrow it lives in the ESR, not in the outcomes and it cautions against reading a raised ESR in a woman with cLBP the same way one would in a man.

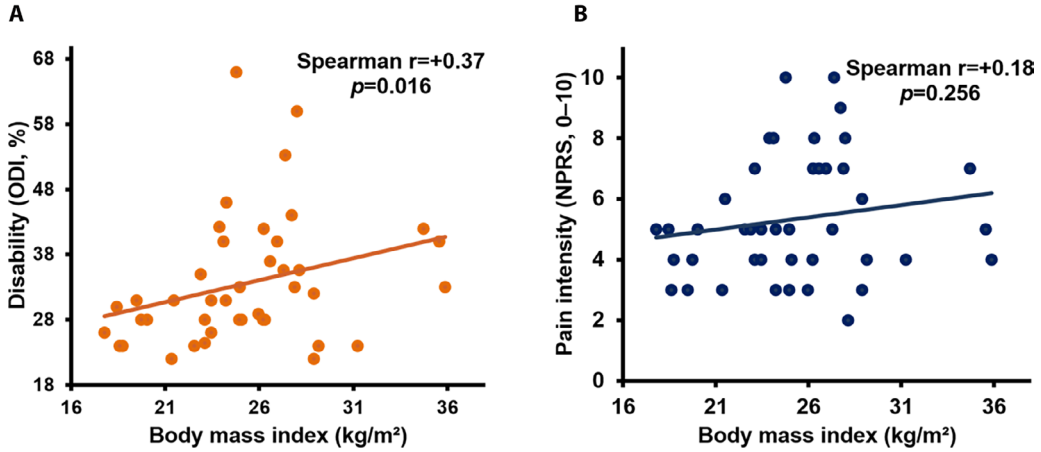

Figure 2. Body mass index against (A) pain intensity (NPRS) and (B) functional disability (ODI). The BMI-ODI correlation was the only significant continuous association in the analysis (Spearman r=0.37, p=0.016).

Table 4. Alignment, inflammation and clinical outcomes compared between lean and higher-adiposity subgroups

Measure	Lean, median (IQR)	Higher, median (IQR)	U	*p*
LLA (°)	40.9 (37.2–53.0)	48.6 (38.0–52.2)	180	0.326
ESR (mm/h)	12.0 (8.0–19.8)	15.5 (6.8–24.2)	204	0.696
NPRS	5.0 (4.0–5.0)	5.5 (4.0–7.0)	182	0.331
ODI (%)	29.0 (26.0–32.5)	34.3 (28.0–40.5)	156	0.111

(Mann-Whitney U test). IQR, interquartile range (25th-75th percentile).

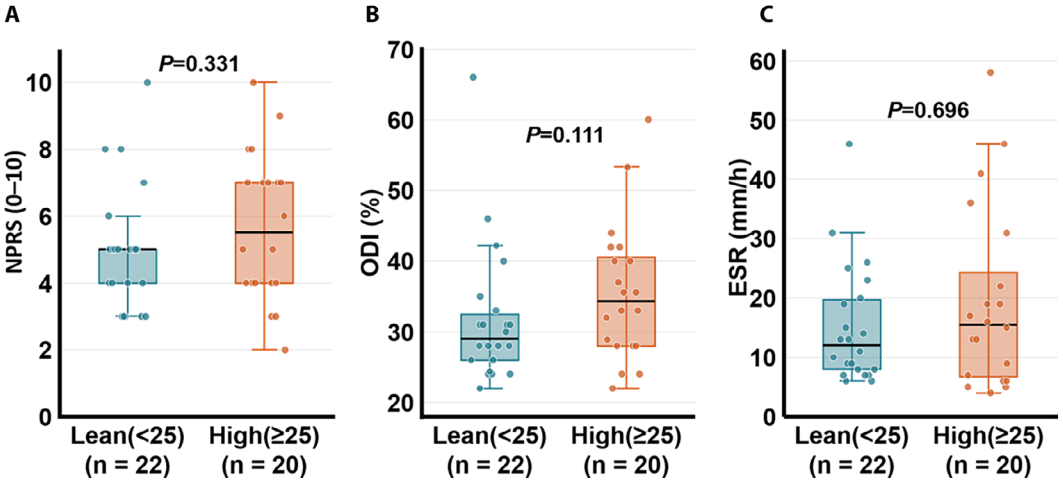

Figure 3. Pain (A), disability (B) and ESR (C) in lean (BMI < 25, n = 22) versus higher-adiposity (BMI ≥ 25, n = 20) participants. Boxes show the median and interquartile range; points are individual values; p-values are from the Mann-Whitney U test. All three measures trended higher with adiposity without reaching significance.

Table 5. Body composition, alignment, inflammation and clinical outcomes by sex

Measure	Men, median (IQR)	Women, median (IQR)	U	*p*
BMI (kg/m²)	24.1 (20.8–26.4)	26.0 (23.1–28.5)	151	0.090
LLA (°)	42.5 (35.6–52.5)	47.1 (38.2–52.2)	176	0.289
NLR	2.0 (1.4–2.7)	2.0 (1.7–2.5)	194	0.535
ESR (mm/h)	8.0 (6.0–13.0)	17.0 (13.0–28.5)	94	0.002 *
NPRS	5.0 (4.0–7.0)	5.0 (4.0–7.0)	236	0.672
ODI (%)	31.0 (28.0–38.5)	32.0 (25.2–37.8)	231	0.761

(Mann-Whitney U test). * $p<0.05$.

Discussion

Placing body mass at the centre of the analysis, three points emerge. First, of all the continuous variables we examined, BMI was the one that carried a significant association with disability patients with more body mass reported higher ODI scores. Second, that link did not extend cleanly to pain intensity, and among the inflammatory markers it touched only the ESR, leaving the leukocyte ratios unmoved. Third, the sharpest group difference in the dataset was not driven by adiposity at all but by sex, with women showing substantially higher ESR than men. Each of these deserves a closer look.

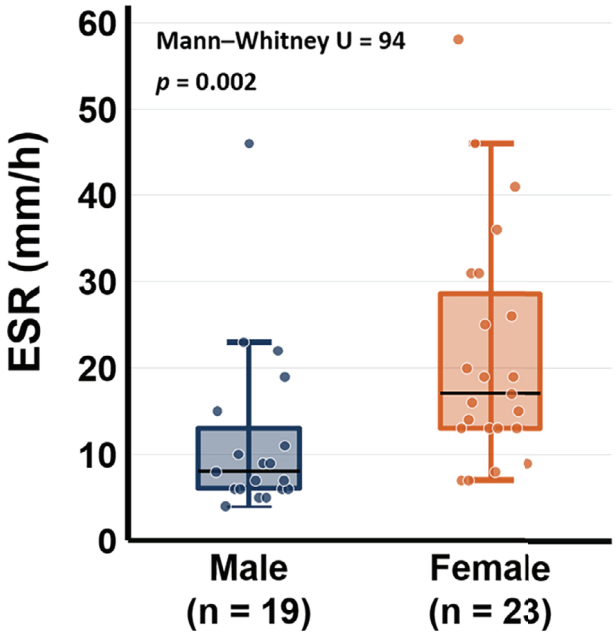

Figure 4. Erythrocyte sedimentation rate by sex. ESR ran substantially higher in women than in men (median 17 vs 8 mm/h; Mann-Whitney p=0.002). Boxes show the median and interquartile range; points are individual values.

Adiposity and the burden of chronic low back pain

That a higher BMI went with greater disability fits a sizeable body of work. Shiri and colleagues, pooling dozens of studies, found overweight and obesity to raise both the prevalence and the incidence of low back pain, with the firmest association seen for chronic and care-seeking pain rather than for transient episodes (4). Urquhart and co-workers went further, reporting that greater fat mass specifically predicted higher pain intensity and disability (19,20), and a more recent Indonesian study tied central adiposity, alongside lordosis and stress, to back pain in young adults (5). Our finding sits comfortably in that line. What is perhaps more interesting is the dissociation we saw between disability and pain: BMI tracked the ODI but not the NPRS. One reading is mechanical and functional carrying more body mass makes the very activities the ODI probes (lifting, walking, sitting tolerance, self-care) physically harder, quite apart from how intense the pain feels at rest. Disability, in other words, may be where adiposity leaves its mark most visibly, which is a useful reminder that the two outcomes should not be collapsed into one.

Why ESR, and not the leukocyte ratios, moved with body mass

The split we observed among the inflammatory markers is biologically coherent. Adipose tissue is an active endocrine and immune organ; as fat mass grows, adipocytes and resident macrophages release more leptin, interleukin-6 and tumour necrosis factor-α, and these in turn drive hepatic synthesis of acute-phase proteins such as fibrinogen (6,7,8,21). ESR, a Westergren reading that depends heavily on fibrinogen and immunoglobulins, is well placed to register that slow, smouldering rise (14). The neutrophil-to-lymphocyte and monocyte-to-lymphocyte ratios answer to a different rhythm: they shift with acute or substantial immune challenges and are comparatively deaf to the low-grade inflammation that accompanies adiposity and chronic musculoskeletal pain (12,13,22). Thus, the fact that BMI correlated with ESR but not with NLR or MLR is less a contradiction than a clue to which marker is sensitive at the low-grade end of the spectrum. The caveat is the by-now-familiar one: the BMI–ESR association was robust under the Pearson assumption but softer under Spearman, because a handful of high-ESR patients stretch the distribution. We therefore present it as suggestive rather than settled.

The sex gradient in ESR

The most striking single contrast in our data ESR roughly twice as high in women as in men has to be read with care. Part of it is almost certainly physiological: ESR reference intervals are conventionally set higher for women and rise further with age, reflecting differences in haematocrit and plasma protein profile rather than disease. Part of it may be adiposity, since the women in our cohort also carried a somewhat higher BMI. And part may be the kind of sex-linked difference in pain biology and inflammatory response that others have flagged in low back pain (23,24). We cannot separate these threads in a sample of this size. The practical message is more modest but still useful: an ESR value that looks elevated in a woman with cLBP should be interpreted against sex-specific norms before it is read as a marker of inflammatory burden, and the same number means something different in a man.

Body mass alongside, not instead of, spinal alignment

It would be tempting to cast adiposity as the upstream factor that explains both the lordotic angle and the inflammatory tone, with disability as the common downstream result. Our data are too thin to support that tidy a model. BMI's link to the LLA was only a weak positive trend, and the lean-versus-higher contrast, while uniformly in the expected direction, never reached significance once the continuous variable had been chopped into categories. What the cohort does support is more measured: body mass is one inexpensive, modifiable correlate of the disability seen in cLBP, sitting alongside spinal geometry and inflammation rather than displacing either. In the companion analysis of these same patients, an abnormal LLA and a raised ESR each carried independent signal for the clinical outcomes; the present results add that body mass deserves a place in that same short list of cheap, routinely available markers. A composite that weighs BMI, an alignment measure and a low-cost inflammatory marker together is the natural next step, and one we intend to test.

Strengths and limitations

The study's strengths are a prospectively recruited, well-phenotyped cohort with no missing data; LLA measurements made by a single standardised technique under layered supervision and independent neuroradiological check; and a transparent reporting of both rank-based and parametric statistics, so readers can judge the robustness of each claim. The limitations are real and constrain how far the findings travel. The design is cross-sectional, so we cannot say whether higher body mass precedes or follows the disability it accompanies. Prospective and life-course evidence will be needed to settle direction: unmeasured occupational loading is one such contributor (25), and longitudinal studies have tied overweight and obesity to incident low back disorders and to sciatica (26,27). The sample of 42 is modest, the obese stratum small, and the analysis deliberately stopped short of multivariable adjustment which means we cannot formally disentangle the overlapping contributions of BMI, sex and age to ESR. BMI itself is a crude gauge of adiposity that does not separate fat from lean mass or capture central distribution, which is the depot most implicated in inflammation; waist circumference or imaging-based fat measures would sharpen the question. The inflammatory panel was limited to three routine markers, and more specific mediators (IL-6, TNF-α, hs-CRP, leptin) would test the mechanism we have invoked more directly. Finally, we did not measure psychosocial contributors to disability, which are known to weigh heavily on ODI scores.

Conclusions

In this Indonesian cross-sectional cohort of 42 adults with chronic low back pain, a higher body mass index was associated with greater self-reported disability while its association with the erythrocyte sedimentation rate was inconsistent across statistical methods and should be interpreted cautiously, while showing no relationship with the neutrophil- or monocyte-to-lymphocyte ratios and only a weak trend with lumbosacral alignment. The clearest single difference in the data was a markedly higher ESR in women, which should be read against sex-specific norms. Taken together, the findings position adiposity as one inexpensive and modifiable correlate of the functional burden of cLBP worth recording alongside spinal alignment and a low-cost inflammatory marker, but not a substitute for either. Larger, longitudinal studies with better measures of body fat, more specific inflammatory mediators and validated psychosocial tools are needed before these associations can be turned into stratification tools.

Ethical Approval and Informed Consent: Approved by the Health Research Ethics Committee, Faculty of Medicine, Hasanuddin University (approval 882/UN4.6.4.5.31/PP36/2025). Procedures followed the Declaration of Helsinki (2013 revision). Written informed consent was obtained from every participant before any study procedure.

Conflict of Interest: Each author declares no commercial associations (consultancies, stock ownership, equity interest, patent or licensing arrangements) that might constitute a conflict of interest in connection with this article.

Author Contributions: IS: conceptualisation, design, LLA measurement, NPRS and ODI administration, data collection, analysis and drafting. MYA: conceptualisation, supervision, senior clinical validation of all measurements, interpretation, revision and final approval. NH: methodology and revision. JB: independent neuroradiological validation of all LLA measurements. LTL and YG: senior clinical supervision and validation, interpretation and revision. All authors read and approved the final manuscript.

Declaration on the Use of AI: The authors used AI-assisted language tools only for minor grammar and language checks. No AI tool was used to generate scientific content, perform analyses, interpret data or prepare references. All scientific judgements and conclusions are those of the authors.

Data Availability. All data are available from the corresponding author on reasonable request (MYA).

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