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ORIGINAL ARTICLE

Peripheral Blood Inflammatory Ratios and Prostate Volume in Benign Prostatic Hyperplasia: An Equivalence-Tested Cross-Sectional Study at a Tertiary Indonesian Referral Centre

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ABSTRACT

Background: Chronic inflammation contributes to benign prostatic hyperplasia (BPH), and complete blood count-derived ratios have been proposed as inexpensive surrogates of prostatic inflammatory burden.

Objective: To determine whether neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR) and lymphocyte-to-platelet ratio (LPR) are associated with ultrasonographically measured prostate volume in Indonesian men with BPH.

Methods: This cross-sectional study included 45 consecutive men with BPH at Dr. Mohammad Hoesin General Hospital, Palembang (March–June 2026). Spearman correlation and Mann–Whitney tests were complemented by pre-specified equivalence tests, Bayes factors, minimum-detectable-effect analysis and sensitivity analyses.

Results: Mean age was 70.31 ± 7.98 years and mean prostate volume was 58.14 ± 29.49 mL. NLR was elevated in 77.8% and MLR in 53.3%. Correlations with prostate volume were ρ = 0.055, 0.056 and 0.027 for NLR, MLR and LPR, respectively, each explaining <0.32% of variance. Equivalence held to $|\rho| < 0.30$ for NLR and LPR and to $|\rho| < 0.35$ for all three markers; Bayes factors favoured the null 5.0–5.2:1. Prostate volume did not differ across NLR or MLR categories.

Conclusion: Systemic inflammation was prevalent yet quantitatively unrelated to prostate volume. Blood-count ratios should not be used to estimate gland size or select an operative approach.

1. Introduction

Benign prostatic hyperplasia (BPH) is the commonest benign proliferative disorder of ageing men and one of the highest-volume conditions in general and urological surgical practice. Global Burden of Disease analyses record a steep rise in prevalence from the sixth decade of life, with the greatest burden between 60 and 79 years and a continuing worldwide increase driven by population ageing.[1,2] Lifetime risk in men above 40 years is substantial across all world regions, and the disease clusters with cardiometabolic comorbidity in ways that magnify its surgical impact.[3,4] Clinically it presents as progressive lower urinary tract symptoms (LUTS) arising from static obstruction by an enlarging transition zone and from dynamic smooth-muscle tone. Contemporary surgical management — transurethral resection, endoscopic enucleation, robot-assisted or open simple prostatectomy, and the newer minimally invasive therapies — is stratified above all by measured gland volume.[5,6,7] In a tertiary referral system serving a large and ageing catchment, any inexpensive predictor of that volume would have immediate operational value.

Chronic inflammation is now recognised as a core component of BPH pathogenesis rather than an incidental finding.[8] Macrophage- and lymphocyte-derived cytokines, principally interleukin-6 (IL-6) and tumour necrosis factor-α (TNF-α), sustain a stromal proliferative programme that operates alongside, and partly independently of, androgen signalling.[9,10] Mechanical and inflammatory cues converge on YAP1 to promote cell survival and fibrosis in prostatic stroma,[11] and even remote inflammatory foci can drive prostatic hyperplasia through the IL-6/IL-6R axis.[12] Circulating IL-6 and TNF-α concentrations track prostatic disease activity,[13] and higher IL-6 has been linked directly to LUTS severity in men with BPH.[10] Together these observations make a peripheral inflammatory readout an attractive proposition.

The complete blood count offers exactly such a readout at no additional cost. NLR, MLR and the platelet- or lymphocyte-based ratios have been evaluated extensively in urology, but the evidence is neither uniform nor easy to reconcile. In the prospective TCLSIH cohort of 15,783 Chinese men free of BPH at baseline, higher NLR predicted incident BPH defined as a total prostate volume of 30 mL or more.[14] A propensity score-matched analysis of 8,727 middle-aged men similarly reported associations of NLR, PLR and the lymphocyte-to-monocyte ratio with prevalent BPH.[15] Among 698 men with BPH/LUTS, complete blood count-derived indices were independently associated with symptom severity on the International Prostate Symptom Score (IPSS),[16] and in 2,709 men in the National Health and Nutrition Examination Survey (NHANES) the NLR was associated with LUTS with an odds ratio of 1.81 (95% CI 1.31–2.49).[17] Against this, an NHANES analysis of 22,343 adults found the relationship between NLR and overactive bladder to be U-shaped rather than monotonic,[18] and a small case-control series reported near-perfect discrimination between men with BPH and healthy controls — a magnitude difficult to reconcile with the population-scale estimates.[19]

Two structural problems limit what this literature can support. First, the estimand is usually the *presence* of BPH, dichotomised at a volume threshold and heavily confounded by age, rather than measured volume treated as a continuous outcome.[14,15,17] Second, associations are typically reported as p-values without effect sizes, confidence intervals or any statement of the magnitude the study could have detected, so a reader cannot distinguish a study that found nothing from one

that could never have found anything.[20] Where a relationship with prostate volume has been sought directly, the signal has been narrow: among 671 men with diabetes, MLR correlated with prostate volume only within the diabetic stratum,[21] and metabolic loading is itself an established determinant of prostatic growth.[22,23]

No study has evaluated these ratios against measured prostate volume in an Indonesian population, and the population is not incidental to the question. The cohort reported here is elderly, lean and predominantly agricultural, a profile far removed from the obese, urban and metabolically loaded populations in which most positive associations have been described. If metabolic loading mediates the association, a lean agrarian cohort is precisely the setting in which it should attenuate. We therefore evaluated whether NLR, MLR and LPR are associated with ultrasonographically measured prostate volume in men with BPH at Dr. Mohammad Hoesin General Hospital, the tertiary surgical referral centre for South Sumatera. Because a null result in a modest sample is uninterpretable unless its precision is quantified, we pre-specified a smallest effect size of interest and analysed the data with equivalence tests and Bayes factors alongside conventional significance testing, so that the study would report not only whether an association was detected but what magnitude of association it can exclude.

2. Methods

Study design and setting

This analytical observational study used a retrospective cross-sectional design and is reported in accordance with the STROBE statement for cross-sectional studies. It was conducted in the urology outpatient service of Dr. Mohammad Hoesin General Hospital, Palembang, the tertiary referral and teaching hospital of the Faculty of Medicine, Universitas Sriwijaya and the principal surgical referral centre for South Sumatera. Data were collected between March and June 2026 from existing medical records. The catchment is predominantly rural and agricultural, and many men reach the service only after years of symptoms or an episode of acute urinary retention, which shapes both the age structure and the range of gland volumes reported below.

Participants and eligibility

Consecutive sampling was used. Men were eligible if they carried a clinical diagnosis of BPH with an available ultrasonographic prostate volume, were older than 40 years, and had a complete blood count and a prostate volume measurement obtained within three months of each other. Exclusion criteria, applied a priori, were a history or diagnosis of prostate cancer; a history of haematological malignancy; acute prostatitis at the time of measurement; use of medication known to alter blood counts, including cytotoxic chemotherapy and immunosuppressive therapy; use of a 5-α-reductase inhibitor before the volume measurement; antibiotic use within the preceding month; and incomplete or invalid laboratory or volume data. Forty-five men met all criteria and all were analysed, with no missing values in the analysed set. The eligibility cascade and the analytical structure are shown in **Figure 1**.

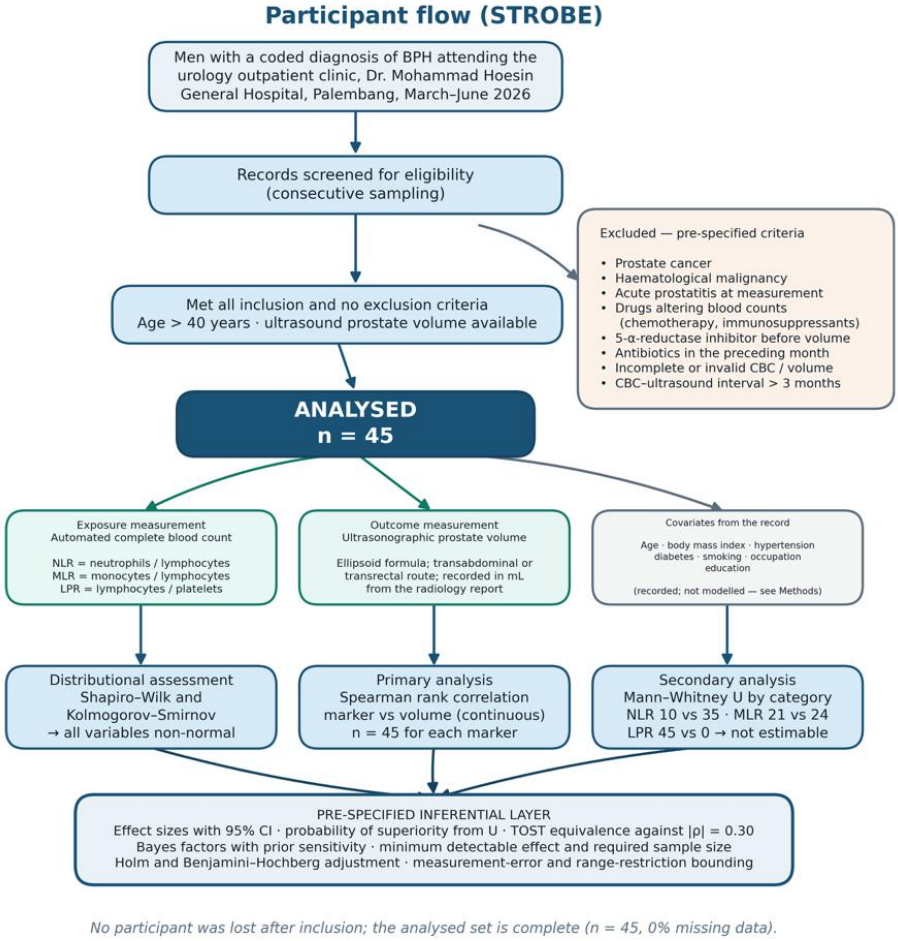

Figure 1. Participant flow, measurement and analysis structure reported according to the STROBE statement. Forty-five of the consecutively screened men met all eligibility criteria and all were analysed, with no missing data.

Variables and measurement

The outcome variable was total prostate volume in millilitres, taken from the radiology report and calculated by the ellipsoid formula from ultrasonographic measurements obtained by the transabdominal or transrectal route according to clinical circumstance. Because both routes were used and the route was not consistently recorded, a route-specific breakdown cannot be given; the ellipsoid method is known to overestimate volume relative to manual planimetry and to carry appreciable inter-rater variability, which is quantified in the sensitivity analysis below.[24,25,26] The exposure variables were three ratios derived from the automated complete blood count: NLR (neutrophils ÷ lymphocytes), MLR (monocytes ÷ lymphocytes) and LPR (lymphocytes ÷ platelets). Each ratio was additionally dichotomised at the threshold used in the source literature — NLR normal 1–2 versus elevated above 2, MLR normal below 0.35 versus elevated at or above 0.35, and LPR decreased below 1.2 versus normal at or above 1.2. Age, body mass index, hypertension, diabetes mellitus, smoking history, occupation and educational attainment were abstracted from the record. Full operational definitions, instruments and units are given in **Table 1**.

Table 1. Operational definitions of the study variables.

Variable	Definition	Instrument	Unit and categories
NLR	Neutrophil-to-lymphocyte ratio from the automated differential count	Automated haematology analyser	Dimensionless; normal 1–2, elevated > 2
MLR	Monocyte-to-lymphocyte ratio from the automated differential count	Automated haematology analyser	Dimensionless; normal < 0.35, elevated ≥ 0.35
LPR	Lymphocyte-to-platelet ratio from the automated count	Automated haematology analyser	Dimensionless; applied cut-off 1.2 (see Results and Figure 3)
Prostate volume	Total glandular volume by the ellipsoid formula, transabdominal or transrectal route	Ultrasonography (radiology report)	mL
Benign prostatic hyperplasia	Clinical diagnosis of BPH with lower urinary tract symptoms and an available prostate volume	Clinical record	Present in all participants
Age	Completed years at the time of data collection	Medical record	Years (> 40 required)
Body mass index	Weight in kilograms divided by height in metres squared	Medical record	kg/m^2; Asian categories < 18.5, 18.5–22.9, ≥ 23
Comorbidity	Recorded history of hypertension, diabetes mellitus or smoking	Medical record	Yes / No
Occupation	Principal reported occupation	Medical record	Farmer / self-employed / labourer / civil servant / not working / other
Education	Highest level of formal education completed	Medical record	Primary / junior secondary / senior secondary / tertiary

Notes: NLR = neutrophil-to-lymphocyte ratio; MLR = monocyte-to-lymphocyte ratio; LPR = lymphocyte-to-platelet ratio; BPH = benign prostatic hyperplasia.

The International Prostate Symptom Score, maximum urinary flow rate, post-void residual volume, serum prostate-specific antigen, transition-zone volume and intravesical prostatic protrusion were sought but were not recorded with sufficient completeness in the retrospective source to permit analysis. Their absence constrains the inferences that can be drawn and is addressed in the Discussion.

Sample size

The sample size was derived from a single-proportion hypothesis-testing formula with $P_0 = 0.678$, $P_a = 0.8678$, $\alpha = 0.05$ and $1 - \beta = 0.80$, giving 39.99 and hence 40 subjects, to which a 10% allowance for incomplete records was added to reach 45. That calculation targets a proportion, whereas the analysis targets a rank correlation: detecting $\rho = 0.30$ at 80% power and $\alpha = 0.05$ requires 85 subjects, so the realised sample is 47% short of the requirement for its own primary estimand. The consequence is stated plainly. This study can exclude a moderate or large association between any of the three ratios and prostate volume; it cannot exclude a small one. Every result below is reported on that basis, and the equivalence framework described next is the response to that limitation.

Statistical analysis

Analyses were performed in IBM SPSS Statistics version 26 and independently reproduced from the reported rank statistics in Python 3.11 with SciPy 1.17. Continuous variables are summarised as mean ± standard deviation and median (range); categorical variables as counts with percentages and Wilson score 95% confidence intervals. Distributional assumptions were assessed with the Shapiro–Wilk and Kolmogorov–Smirnov tests. Because every continuous variable departed from normality, the primary analysis used Spearman rank correlation between each marker and prostate volume, and the secondary analysis used the Mann–Whitney U test to compare prostate volume across the dichotomised marker categories. Two-sided α was 0.05.

Confidence intervals for ρ were obtained by the Fisher z transformation, which for a rank correlation uses the variance-inflated standard error $\sqrt{[(1 + \rho^2/2)/(n - 3)]}$. At the correlations observed here the inflation factor is 1.0008, so intervals are identical to three decimal places whichever form is used. Effect sizes for the Mann–Whitney comparisons are reported as the rank-biserial correlation and as the probability of superiority — the probability that a randomly selected man in the elevated category has the larger prostate — which is numerically equal to the area under the receiver operating characteristic curve for that grouping and is presented with the Hanley–McNeil standard error. This quantity is not a measure of diagnostic accuracy: no reference standard was dichotomised and no threshold was varied. Prostate volumes were recorded to the nearest half millilitre so ties are present; the software applied no tie correction, which renders the standardised statistic conservative, and the exact p-values computed from the untied null distribution are correspondingly approximate.

Equivalence was assessed by two one-sided tests against a pre-specified smallest effect size of interest of $|\rho| = 0.30$, each at $\alpha = 0.05$, with equivalence declared when the 90% confidence interval lies entirely inside the bound.[20] The bound was chosen because a correlation below 0.30 explains under 9% of the variance in prostate volume, which given an observed standard

deviation of 29.49 mL corresponds to less than 9 mL of explained spread — far below the 30, 80 and 100 mL thresholds around which operative technique is selected[5,6] and therefore below any magnitude that could alter a surgical decision. Because a single bound is a strong assumption, the equivalence conclusion is additionally reported across bounds from 0.20 to 0.40. Bayes factors were computed on the Fisher z scale under a uniform prior on $\rho \in (-1, 1)$ and under a prior concentrated on small effects (normal, standard deviation 0.20), and are reported as BF_{01} with the prior sensitivity displayed. Finally we computed the minimum detectable correlation at 80% and 90% power, the sample size required for the primary estimand, and the behaviour of ρ under plausible measurement-error and range-restriction assumptions. Multiplicity across the five hypothesis tests was addressed with the Holm and Benjamini–Hochberg procedures.

Individual-level records were not released for this analysis, only marginal distributions and rank statistics. Multivariable modelling, diagnostic receiver-operating-characteristic analysis for a prostate-volume threshold and survival analysis were therefore not performed, since none can be recovered from marginal data without assuming a joint distribution that was never observed. No operative intervention and no tissue sampling took place within the study, so no complication grading and no histological or immunohistochemical result is reported.

Ethics

This study was granted ethical exemption by the Ethical Committee of Dr Mohammad Hoesin General Hospital, Palembang, Indonesia (Ref. No. Dp.04.03/D.XVII.9.4/ETIK/144/2026, issued 25 May 2026 and valid until 25 May 2027). Because the study was a retrospective review of existing medical records involving no patient contact and no identifiable data leaving the institution, the committee waived the requirement for individual written informed consent. All records were pseudonymised before analysis, and the study was conducted in accordance with the Declaration of Helsinki.

3. Results

Cohort characteristics

Forty-five men were analysed. The mean age was 70.31 ± 7.98 years, with a median of 69.00 years and a range of 56 to 97, and the mean body mass index was 20.89 ± 3.36 kg/m^2 (median 20.30; range 15.8–30.2), placing the cohort at the lower end of the normal range and well below the populations in which most reported inflammatory associations were derived. Mean prostate volume was 58.14 ± 29.49 mL (95% CI 49.28–67.00), with a median of 47.00 mL and a range of 30 to 154 mL. Hypertension was present in 22 men (48.9%; 95% CI 35.0–63.0), diabetes mellitus in 6 (13.3%; 6.3–26.2) and a smoking history in 23 (51.1%; 37.0–65.0). Twenty men (44.4%; 30.9–58.8) were farmers and 21 (46.7%; 32.9–60.9) had completed primary school only, an occupational and educational profile that matches other Indonesian urological series and differs markedly from the cohorts underpinning the international literature.[27] The full characteristics are detailed in **Table 2**.

Table 2. Characteristics of the study population (n = 45).

Characteristic	Mean ± SD	Median (range)	95% CI of the mean
Age, years	70.31 ± 7.98	69.00 (56–97)	67.91 to 72.71
Body mass index, kg/m^2	20.89 ± 3.36	20.30 (15.8–30.2)	19.88 to 21.90
Prostate volume, mL	58.14 ± 29.49	47.00 (30–154)	49.28 to 67.00
NLR	3.79 ± 3.45	2.79 (0.91–22.50)	2.75 to 4.83
MLR	0.403 ± 0.219	0.350 (0.15–1.50)	0.337 to 0.469
LPR	0.0824 ± 0.0553	0.070 (0.01–0.31)	0.0658 to 0.0990
Categorical variable	n (%)	95% CI (Wilson)	—
Hypertension	22 (48.9)	35.0 to 63.0	—
Diabetes mellitus	6 (13.3)	6.3 to 26.2	—
Smoking history	23 (51.1)	37.0 to 65.0	—
Occupation — farmer	20 (44.4)	30.9 to 58.8	—
Occupation — self-employed	10 (22.2)	12.5 to 36.3	—
Occupation — labourer	6 (13.3)	6.3 to 26.2	—
Occupation — civil servant	3 (6.7)	2.3 to 17.9	—
Occupation — not working or retired	3 (6.7)	2.3 to 17.9	—
Occupation — other	3 (6.7)	2.3 to 17.9	—
Education — primary	21 (46.7)	32.9 to 60.9	—
Education — junior secondary	4 (8.9)	3.5 to 20.7	—
Education — senior secondary	15 (33.3)	21.1 to 48.3	—
Education — tertiary	5 (11.1)	4.8 to 23.5	—

Notes: SD = standard deviation; CI = confidence interval. Intervals for proportions are Wilson score intervals. Percentages are of the 45 analysed men and sum to 100% within each category block.

All six continuous variables were right-skewed. Pearson's second coefficient of skewness was +1.13 for prostate volume, +0.87 for NLR, +0.72 for MLR, +0.67 for LPR, +0.53 for body mass index and +0.49 for age; the corresponding distributions are displayed in **Figure 2**, in which the observed medians, means and extremes are marked on method-of-moments log-normal models fitted to the reported means and standard deviations. The Shapiro–Wilk test rejected normality for every variable (W = 0.584 for NLR, 0.734 for MLR, 0.769 for LPR and 0.836 for prostate volume; all $p < 0.001$), as did the Kolmogorov–Smirnov test (D = 0.242, 0.164, 0.229 and 0.173 respectively; all $p \leq 0.004$), confirming that rank-based methods were required throughout. These distributional results are summarised together with the marker categories in **Table 3**.

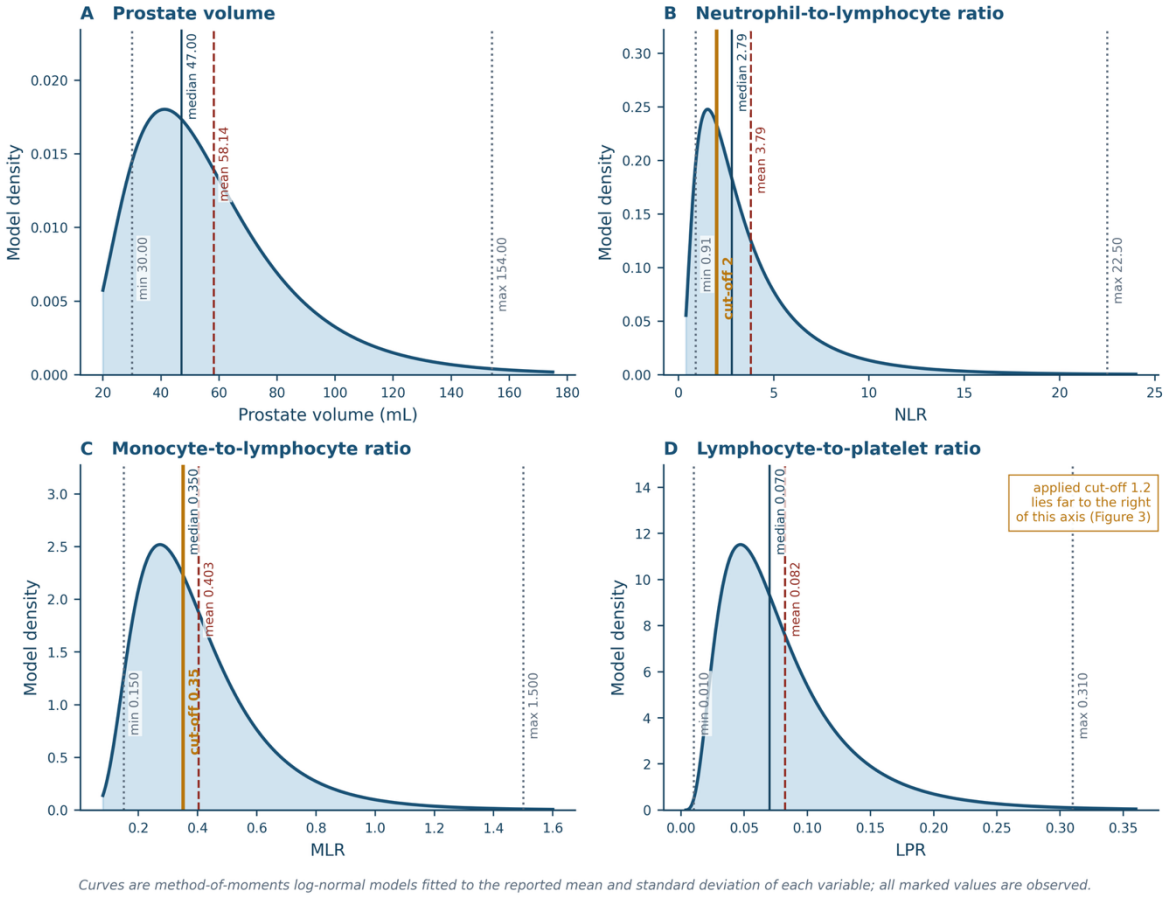

Figure 2. Distribution of the outcome and of the three inflammatory ratios. Curves are method-of-moments log-normal models fitted to the reported means and standard deviations; the observed median, mean and extremes are marked on each panel, and the categorical cut-offs applied to NLR and MLR are indicated. All four variables are right-skewed and departed from normality (Table 3).

Table 3. Distribution of the inflammatory ratios and tests of normality (n = 45).

Marker	Category	n (%)	95% CI (Wilson)	Shapiro–Wilk W	Kolmogorov–Smirnov D	p
NLR	Normal (1–2)	10 (22.2)	12.5 to 36.3	0.584	0.242	< 0.001
	Elevated (> 2)	35 (77.8)	63.7 to 87.5			
MLR	Normal (< 0.35)	21 (46.7)	32.9 to 60.9	0.734	0.164	< 0.001
	Elevated (≥ 0.35)	24 (53.3)	39.1 to 67.1			
LPR	Below cut-off (< 1.2)	45 (100.0)	92.1 to 100.0	0.769	0.229	< 0.001
	At or above cut-off (≥ 1.2)	0 (0.0)	0.0 to 7.9			
Prostate volume	—	45	—	0.836	0.173	< 0.001

Notes: degrees of freedom = 45 for both tests; p-values shown are for the Shapiro–Wilk test, and the Kolmogorov–Smirnov test with Lilliefors correction gave the same conclusion for every variable (p ≤ 0.004). The LPR categorisation is not interpretable because the applied cut-off of 1.2 lies 16.1 standard deviations above the observed maximum of 0.31 (Figure 3).

Prevalence of systemic inflammation

The cohort was distinctly pro-inflammatory, and this is a finding in its own right rather than a preamble to the analysis that follows. As shown in **Table 3**, NLR exceeded the upper reference bound of 2 in 35 of 45 men (77.8%; 95% CI 63.7–87.5), with a mean of 3.79 ± 3.45 (95% CI 2.75–4.83) and a median of 2.79, while MLR reached or exceeded 0.35 in 24 men (53.3%; 39.1–67.1), with a mean of 0.403 ± 0.219. Published reference intervals for these ratios in healthy adults place the upper limit of NLR between roughly 2 and 3 and confirm that the interval widens with age,[28,29] so the observed elevation is unlikely to be an artefact of an inappropriate reference bound. Benchmarking the observed 77.8% against assumed reference prevalences of 15%, 20%, 25%, 30% and 35% yields prevalence ratios of 5.19, 3.89, 3.11, 2.59 and 2.22 respectively, with exact binomial p < 0.001 throughout; these reference prevalences are assumptions imported for context rather than measurements made here. An elevated NLR in this age band is independently associated with all-cause mortality in longitudinal data,[30] so the profile carries prognostic meaning even where it carries no prostatic meaning.

Behaviour of the lymphocyte-to-platelet ratio at the applied threshold

LPR fell below the applied cut-off of 1.2 in all 45 men (100%; 95% CI 92.1–100), leaving the comparison category empty (**Table 3**). Inspection of the observed values shows why. LPR spanned 0.01 to 0.31 with a mean of 0.0824 ± 0.0553, so the 1.2 cut-off lies 16.1 standard deviations above the observed maximum and 20.2 above the mean. Inverting the observed values gives an implied platelet-to-lymphocyte ratio of 14.3 at the median and 3.2 to 100 across the range, an order of magnitude below the values reported for adult reference populations.[29] A lymphocyte-to-platelet ratio computed from absolute counts — lymphocytes 0.8–5.0 × 10^9/L over platelets 100–450 × 10^9/L — can occupy only about 0.002 to 0.05. The observed domain is therefore consistent with a difference of

scale between the ratio as computed and the threshold as imported, and the resulting 100%/0% split is an arithmetic consequence of that mismatch rather than a biological uniformity. The relationship between the attainable domain and the applied threshold is shown in **Figure 3**. The continuous analysis of LPR is unaffected, because Spearman's ρ is invariant under any monotone rescaling of either variable; the correlation reported below therefore stands irrespective of the scale on which the ratio was computed. Only the categorical comparison is uninterpretable, and it is reported as not estimable rather than as a negative result.

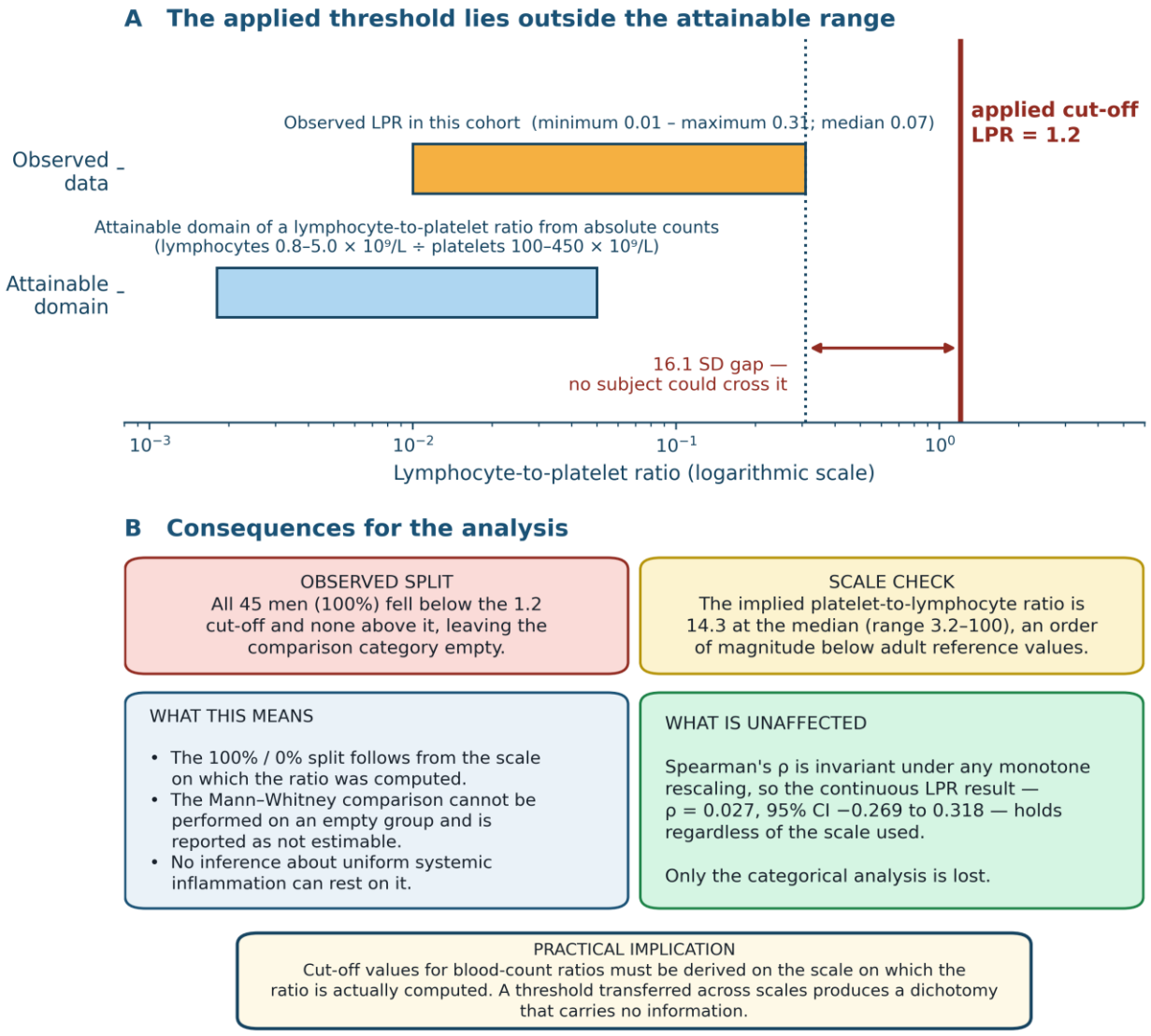

Figure 3. Behaviour of the lymphocyte-to-platelet ratio at the applied threshold. (A) The 1.2 cut-off lies 16.1 standard deviations above the observed maximum and outside the domain attainable by a lymphocyte-to-platelet ratio computed from absolute counts. (B) Consequences for the analysis: the categorical comparison is not estimable, while the scale-invariant rank correlation is unaffected.

Primary analysis: correlation with prostate volume

None of the three markers was associated with prostate volume. As detailed in **Table 4**, Spearman's ρ was 0.055 for NLR (95% CI −0.242 to 0.343; p = 0.721), 0.056 for MLR (95% CI −0.242 to 0.344; p = 0.716) and 0.027 for LPR (95% CI −0.269 to 0.318; p = 0.861). Expressed as explained variance, each marker accounts for less than one third of one percent of the variability in prostate volume — 0.30%, 0.31% and 0.07% respectively — so the point estimates are not merely non-significant but close to zero. Testing against the pre-specified smallest effect size of interest, the two one-sided tests gave p = 0.050 for NLR, p = 0.050 for MLR and p = 0.034 for LPR; read through the TOST-consistent 90% confidence intervals, equivalence to |ρ| < 0.30 is established for NLR (−0.196 to 0.299) and for LPR (−0.223 to 0.274), while for MLR the upper limit reaches 0.300 and the bound is not met. Bayes factors computed on the Fisher z scale under a uniform prior favoured the null by 4.98:1 for NLR, 4.97:1 for MLR and 5.21:1 for LPR. The corresponding intervals are displayed against the equivalence bounds in panel A of **Figure 4**.

Table 4. Primary analysis — rank correlation between each inflammatory ratio and prostate volume, with equivalence testing and Bayes factors (n = 45).

Marker	ρ (95% CI)	p	ρ^2 (%)	90% CI (TOST)	TOST p vs |ρ| = 0.30	Equivalence at 0.30	BF01 uniform / small-effect
NLR	0.055 (−0.242 to 0.343)	0.721	0.30	−0.196 to 0.299	0.050	Established	4.98 / 1.59
MLR	0.056 (−0.242 to 0.344)	0.716	0.31	−0.195 to 0.300	0.050	Not established	4.97 / 1.59
LPR	0.027 (−0.269 to 0.318)	0.861	0.07	−0.223 to 0.274	0.034	Established	5.21 / 1.64

Notes: ρ = Spearman rank correlation; ρ^2 = proportion of variance in prostate volume accounted for by the marker; TOST = two one-sided tests at α = 0.05 each against a pre-specified smallest effect size of interest of |ρ| = 0.30, with equivalence declared when the 90% confidence interval falls entirely inside the bound. BF01 above 1 favours the null hypothesis; the uniform prior is uniform on (−1, 1) and the small-effect prior is normal with standard deviation 0.20. The MLR 90% upper limit is 0.300, so the bound is met at 0.35 but not at 0.30 (Table 6).

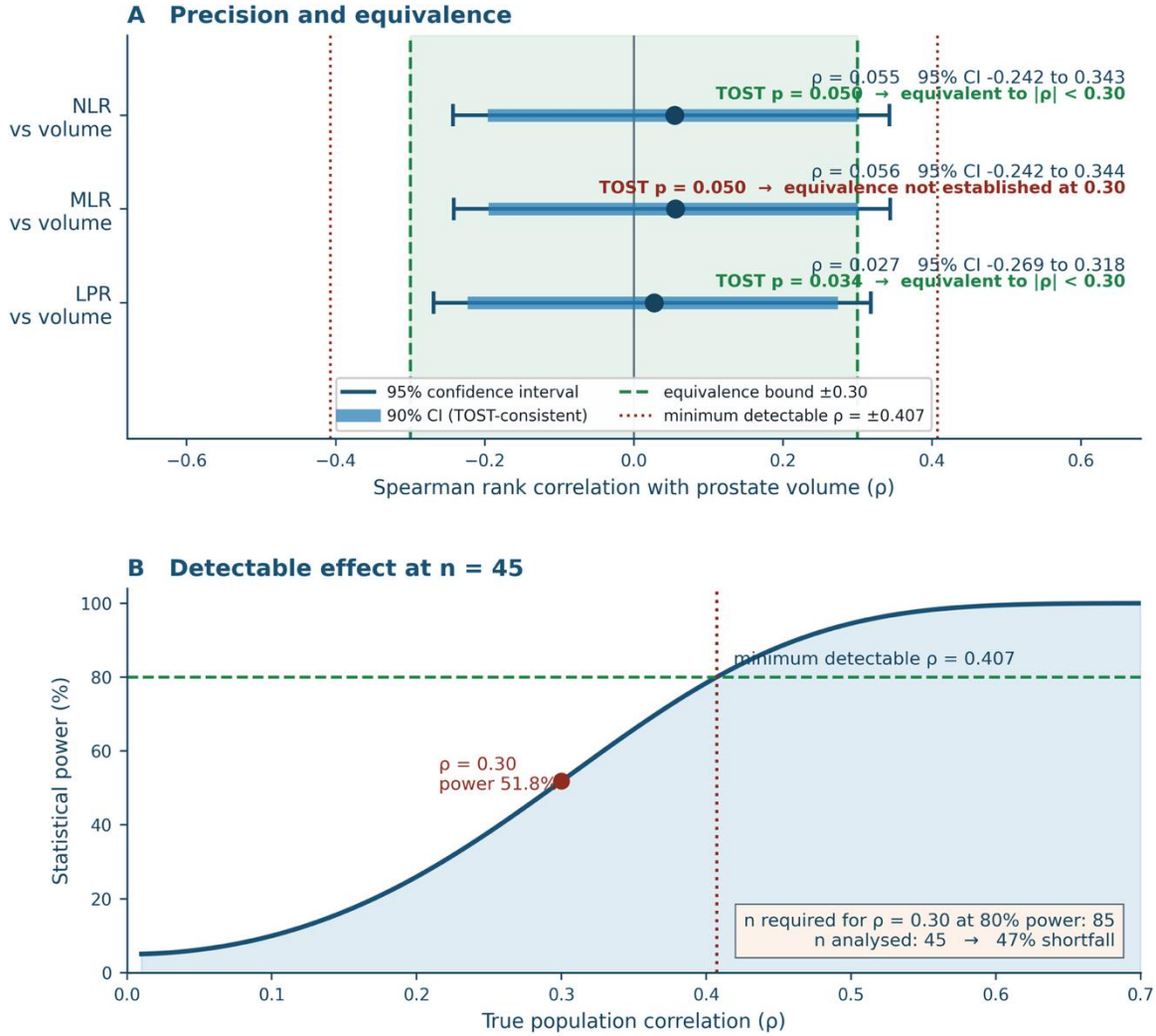

Figure 4. Precision, equivalence and detectable effect. (A) Spearman rank correlations with 95% and 90% confidence intervals against the pre-specified equivalence bounds of ±0.30 and the minimum detectable correlation of ±0.407. (B) Statistical power as a function of the true population correlation at n = 45, showing 51.8% power at ρ = 0.30 and the 85 subjects that would have been required.

The dependence of the Bayes factor on the prior is informative about the design rather than the biology. A uniform prior places most of its mass on correlations that a sample of 45 was well placed to detect, so a factor near 5 chiefly records the absence of large effects. A prior concentrated on small effects places its mass precisely where this sample has little resolving power, and the corresponding factors fall to 1.59, 1.59 and 1.64, which is evidence for neither hypothesis (**Table 4**). The Bayesian and frequentist layers therefore agree: moderate and large associations are excluded, small ones are not addressed.

Secondary analysis: prostate volume across marker categories

Median prostate volume was 41.00 mL (IQR 38.0–53.0) in the 10 men with a normal NLR and 50.50 mL (IQR 37.5–81.6) in the 35 with an elevated NLR. The Mann–Whitney U was 133.0 with Z = −1.147, asymptotic p = 0.252 and exact two-sided p = 0.262; the rank-biserial correlation was 0.240 (95% CI −0.058 to 0.498) and the probability of superiority 0.620 (95% CI 0.431 to 0.809). For MLR, median volume was 45.00 mL (IQR 39.0–70.0) in the 21 men with a normal value and 50.75 mL (IQR 35.0–63.5) in the 24 with an elevated value, giving U = 240.0, Z = −0.273, asymptotic p = 0.785, exact p = 0.796, a rank-biserial correlation of 0.048 (95% CI −0.249 to 0.336) and a probability of superiority of 0.524 (95% CI 0.353 to 0.694). Both intervals include 0.5, and although the direction of the observed difference — a larger median volume in the elevated categories — is consistent between markers, its magnitude is small relative to the interquartile spread. These comparisons are tabulated in **Table 5** and the corresponding distributions are shown in **Figure 5**. The LPR comparison was not estimable for the reason set out above.

Table 5. Secondary analysis — prostate volume compared across inflammatory ratio categories, with effect sizes.

Comparison	n	Median volume, mL (IQR)	U	Z	p (asymptotic)	p (exact)	Rank-biserial r (95% CI)	Probability of superiority (95% CI)
NLR normal (1–2)	10	41.00 (38.0–53.0)	133.0	−1.147	0.252	0.262	0.240 (−0.058 to 0.498)	0.620 (0.431 to 0.809)
NLR elevated (> 2)	35	50.50 (37.5–81.6)						
MLR normal (< 0.35)	21	45.00 (39.0–70.0)	240.0	−0.273	0.785	0.796	0.048 (−0.249 to 0.336)	0.524 (0.353 to 0.694)
MLR elevated (≥ 0.35)	24	50.75 (35.0–63.5)						
LPR below cut-off (< 1.2)	45	47.00 (36.0–68.0)	Not estimable	—	—	—	—	—
LPR at or above cut-off (≥ 1.2)	0	—						

Notes: IQR = interquartile range; U = Mann–Whitney U statistic. The probability of superiority is the probability that a randomly chosen man in the elevated category has the larger prostate; it equals the area under the receiver operating characteristic curve for that grouping and is reported with the Hanley–McNeil standard error, with 0.5 denoting no difference. The LPR comparison is not estimable because the applied cut-off leaves one group empty (Figure 3).

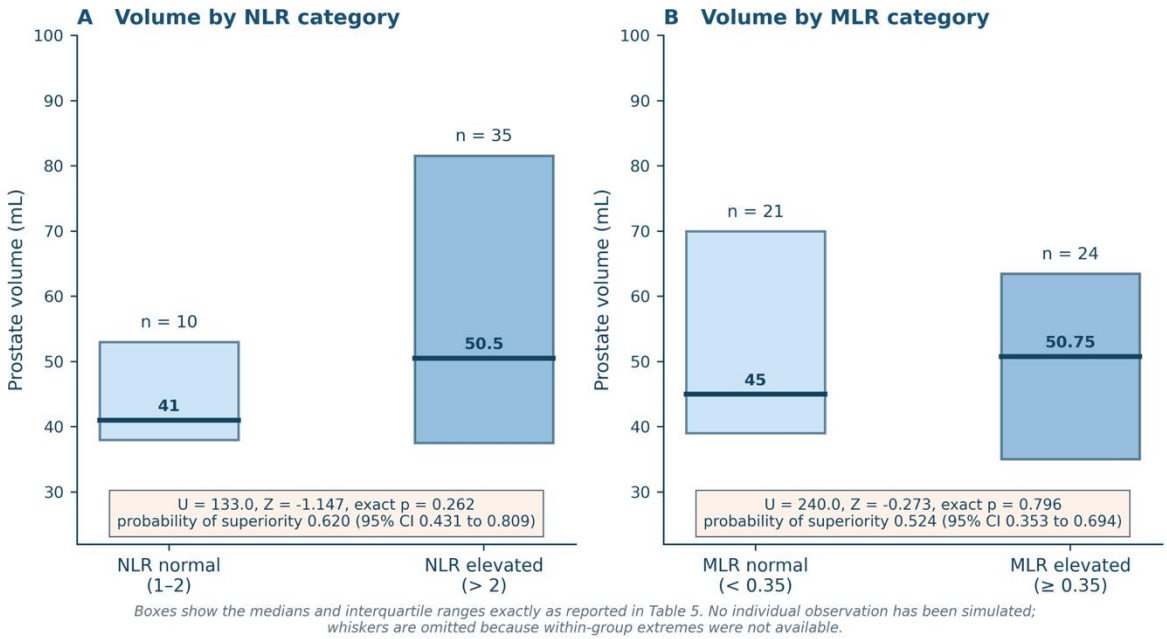

Figure 5. Prostate volume by inflammatory ratio category. Boxes show the medians and interquartile ranges exactly as reported in Table 5; no individual observation has been simulated, and whiskers are omitted because within-group extremes were not available. Neither comparison reached significance and both probability-of-superiority intervals include 0.5.

Sensitivity of the equivalence conclusion to the chosen bound

Because a single smallest effect size of interest is a strong assumption, the equivalence conclusion was recomputed across bounds from 0.20 to 0.40, as detailed in **Table 6**. The pattern is orderly: no marker is equivalent at a bound of 0.20 (TOST p = 0.169, 0.171 and 0.127 for NLR, MLR and LPR) or at 0.25 (p = 0.097, 0.098 and 0.069); NLR and LPR become equivalent at 0.30; MLR joins them at 0.35 (p = 0.022, 0.023 and 0.014); and all three are comfortably equivalent at 0.40 (p = 0.009, 0.009 and 0.005). The defensible summary is therefore that this cohort excludes correlations of 0.35 and above for all three markers, excludes 0.30 and above for two of the three, and is uninformative at 0.25 and below — a limit set by the sample size rather than by the choice of bound.

Table 6. Sensitivity of the equivalence conclusion to the choice of smallest effect size of interest.

Equivalence bound |ρ|	NLR TOST p	NLR verdict	MLR TOST p	MLR verdict	LPR TOST p	LPR verdict
0.20	0.169	Not equivalent	0.171	Not equivalent	0.127	Not equivalent
0.25	0.097	Not equivalent	0.098	Not equivalent	0.069	Not equivalent
0.30 (pre-specified)	0.050	Equivalent	0.050	Not equivalent	0.034	Equivalent
0.35	0.022	Equivalent	0.023	Equivalent	0.014	Equivalent
0.40	0.009	Equivalent	0.009	Equivalent	0.005	Equivalent

Notes: two one-sided tests at α = 0.05 each; equivalence is declared when the 90% confidence interval falls entirely inside the stated bound. Reading down the table, the study excludes |ρ| ≥ 0.35 for all three markers, excludes |ρ| ≥ 0.30 for NLR and LPR, and is uninformative at 0.25 and below.

Precision, detectable effect and robustness

With 45 subjects at α = 0.05 the minimum detectable correlation was ρ = 0.407 at 80% power and ρ = 0.462 at 90% power. Power was 26.0% at ρ = 0.20, 51.8% at ρ = 0.30, 78.4% at ρ = 0.40 and 94.5% at ρ = 0.50, and the sample sizes required at 80% power are 194, 85 and 47 subjects for ρ of 0.20, 0.30 and 0.40 respectively. For the categorical comparisons the position is weaker: with groups of 10 and 35 the smallest detectable probability of superiority is 0.793, equivalent to a standardised difference of 1.16, and with groups of 21 and 24 it is 0.744 (d = 0.93). The power curve and the position of the observed effects are shown in panel B of **Figure 4**, and the full set of precision analyses is collected in **Table 7**.

Table 7. Precision, detectable effect and robustness analyses.

Analysis	Quantity	Value	Interpretation
Minimum detectable effect	ρ at 80% power (n = 45, α = 0.05)	0.407	Only a large correlation was detectable
	ρ at 90% power	0.462	—
Achieved power	at ρ = 0.20	26.0%	Uninformative for small effects
	at ρ = 0.30	51.8%	Coin-flip sensitivity at the smallest effect of interest
	at ρ = 0.40	78.4%	Adequate only for large effects
	at ρ = 0.50	94.5%	—
Required sample size (80% power)	for ρ = 0.20	194	—
	for ρ = 0.30	85	The realised sample is 47% short
	for ρ = 0.40	47	—
Mann–Whitney detectable effect	probability of superiority, 10 vs 35	0.793 (d = 1.16)	Only a very large shift was detectable
	probability of superiority, 21 vs 24	0.744 (d = 0.93)	—
Measurement-error bounding	ρ disattenuated (reliability 0.85–0.95 and 0.70–0.90)	0.059 to 0.071	The null survives plausible measurement error
Range-restriction bounding	ρ corrected (unrestricted SD 35–50 mL)	0.065 to 0.093	The null survives correction for cohort restriction
Multiplicity	smallest raw p across five tests	0.252	—
	Holm-adjusted p (all tests)	1.000	No result approaches significance
	Benjamini–Hochberg q (all tests)	0.861	—

Notes: d = the standardised difference implied by the probability of superiority. Attenuation bounding uses rtrue = robs / √(reliabilityx × reliabilityy); range-restriction bounding uses the Thorndike case II formula with the observed standard deviation of 29.49 mL. Both are illustrative calculations under stated assumptions rather than corrected estimates.

The null was stable under every sensitivity analysis applied, each of which is an illustrative bounding calculation under stated assumptions rather than a corrected estimate. Assuming classical measurement error with reliabilities of 0.85 to 0.95 for ultrasonographic prostate volume[24,25] and 0.70 to 0.90 for a single blood-count-derived ratio, the disattenuated NLR correlation rises only from 0.055 to between 0.059 and 0.071. Correcting for restriction of range by the Thorndike case II formula — the cohort was selected for BPH and contained no gland below 30 mL — under assumed unrestricted standard deviations of 35 to 50 mL yields 0.065 to 0.093. Even the most favourable combination of assumptions leaves the estimate far below the smallest effect size of interest. Across the five hypothesis tests the smallest raw p-value was 0.252, so all Holm-adjusted values were 1.000 and all Benjamini–Hochberg adjusted values 0.861; multiplicity control is reported for completeness and could not have altered any conclusion (**Table 7**).

4. Discussion

In 45 consecutive men with BPH at a tertiary Indonesian referral centre, systemic inflammation was common — three quarters had an elevated NLR and half an elevated MLR — yet none of the three complete blood count-derived ratios was associated with prostate volume. The correlations were not merely non-significant but close to zero, together explaining less than one third of one percent of the variance in gland size, and the data are formally equivalent to no more than a small correlation for NLR and LPR and to no more than a moderate one for all three markers. The clinical instruction that follows is specific: these ratios should not be used to estimate prostatic size or to inform the choice of operative approach.

Total prostate volume may be the wrong outcome

The most useful interpretation of these data is not that inflammation is irrelevant to BPH but that total glandular volume is a poor outcome against which to test a peripheral inflammatory marker. Inflammatory and mechanical signalling in prostatic stroma promotes regional remodelling, and the anatomical consequence that best predicts obstruction is intravesical prostatic protrusion rather than total volume. In a meta-analysis of 18 studies and 4,128 patients, intravesical prostatic protrusion predicted urodynamically defined bladder outlet obstruction and unsuccessful trial without catheter,[31] and a more recent PRISMA-DTA synthesis confirmed its diagnostic performance against a bladder outlet obstruction index above 40.[32] In 369 treatment-naive men assessed with three-dimensional ultrasonography, protrusion volume and IPSS were the only independent predictors of a low maximum flow rate, whereas total prostate volume was not.[33] The molecular counterpart is equally specific: FGF7 is upregulated both in protruding tissue and in stromal cells derived from it, driving a localised proliferative programme confined to the median lobe.[34] Because a median lobe may add only a few millilitres to an ellipsoid volume while dominating a patient's obstruction,[35] a study measuring total volume alone is blind to precisely the compartment in which an inflammatory effect would be expected to appear, and our null is fully compatible with that possibility.

The same argument applies zonally. Transition-zone volume, not total volume, is the compartment that hypertrophies in BPH: magnetic resonance transition-zone volume predicts enucleation weight more accurately than total volume measured by transrectal ultrasound, computed tomography or magnetic resonance imaging,[36] and zonal measurements in 430 men show that transition-zone expansion, rather than proportionate growth of the whole gland, accounts for increasing total volume.[37] Prostate dimensions also behave differently from prostate volume in relation to symptoms, with gland length contributing information that volume alone does not capture.[38] A cross-sectional correlation between a systemic marker and a summary ellipsoid volume is therefore a coarse test of a fine-grained hypothesis.

Comparison with the published literature

Our result is consistent with the better-powered studies in this field and inconsistent chiefly with the smallest. The prospective TCLSIH cohort of 15,783 men established an association between NLR and incident BPH defined dichotomously at 30 mL,[14] and the propensity-matched analysis

of 8,727 men likewise modelled BPH status rather than measured volume.[15] Both are estimands that age drives strongly, and neither addresses whether the marker tracks how large the gland becomes among men who already have the disease — the question asked here. Where symptom severity rather than size has been the outcome, associations are more consistent: complete blood count indices were independently associated with IPSS category in 698 men with BPH/LUTS,[16] NLR was associated with LUTS in 2,709 NHANES participants,[17] and NLR combined with ultrasonographic parameters improved the identification of bladder outlet obstruction in 106 men with BPH studied urodynamically.[39] The apparent conflict with the case-control report of near-perfect discrimination between men with BPH and healthy controls[19] is best read as spectrum bias: symptomatic clinic attenders differ from healthy volunteers in age, comorbidity and intercurrent illness as well as in prostatic status, and a design of that kind cannot isolate a prostatic contribution. The same caution applies to studies in which the ratios are used to separate benign from malignant prostatic disease rather than to grade benign disease itself.[40]

Two further findings sharpen the interpretation. First, the relationship between NLR and lower urinary tract dysfunction is not necessarily monotonic: in 22,343 NHANES participants the association with overactive bladder was U-shaped, with an inflection at a natural logarithm of NLR of 0.65, corresponding to an NLR of approximately 1.9.[18] A monotone rank correlation is the wrong instrument for a U-shaped relationship and would be attenuated toward zero even if a real association existed, which is a further reason to treat our estimate as an upper bound rather than a refutation. Second, the one study that examined MLR against measured prostate volume directly found the correlation only within a diabetic stratum of 671 patients,[21] implying effect modification by metabolic state. Our cohort had a mean body mass index of 20.89 kg/m^2 and only 13.3% diabetes, and metabolic syndrome is a well-established determinant of prostatic growth in prospective data — a hazard ratio of 1.07 for incident BPH across 163,975 men in the UK Biobank, mediated through metabolic biomarkers[22] — as well as in mechanistic and cross-sectional work.[23,41] Sarcopenic obesity carries the highest BPH/LUTS prevalence of any body-composition phenotype.[42] In a lean agrarian population these mediating pathways are largely absent, and the attenuation we observed is the expected consequence rather than a contradiction. Consistent with this, transabdominal prostate volume in men with LUTS correlates with body mass index and waist circumference,[43] both of which are low in this cohort.

Mechanistic account of the dissociation

The dissociation between a raised systemic ratio and an unchanged gland volume is biologically coherent, and the two compartments involved are contrasted in **Figure 6**. In the systemic compartment, ageing and comorbidity raise circulating IL-6, TNF-α and IL-1β, which act through NF-κB and JAK/STAT3 signalling in the haematopoietic niche to bias progenitor output toward the granulocyte–monocyte lineage while suppressing lymphoid output. The peripheral consequence is exactly the phenotype observed here: neutrophils and monocytes relatively increased, lymphocytes relatively decreased, and therefore NLR and MLR elevated and LPR depressed. Reference-interval studies confirm that this drift is a normal correlate of ageing rather than a disease-specific signal,[28,29] and it carries independent prognostic weight for mortality.[30]

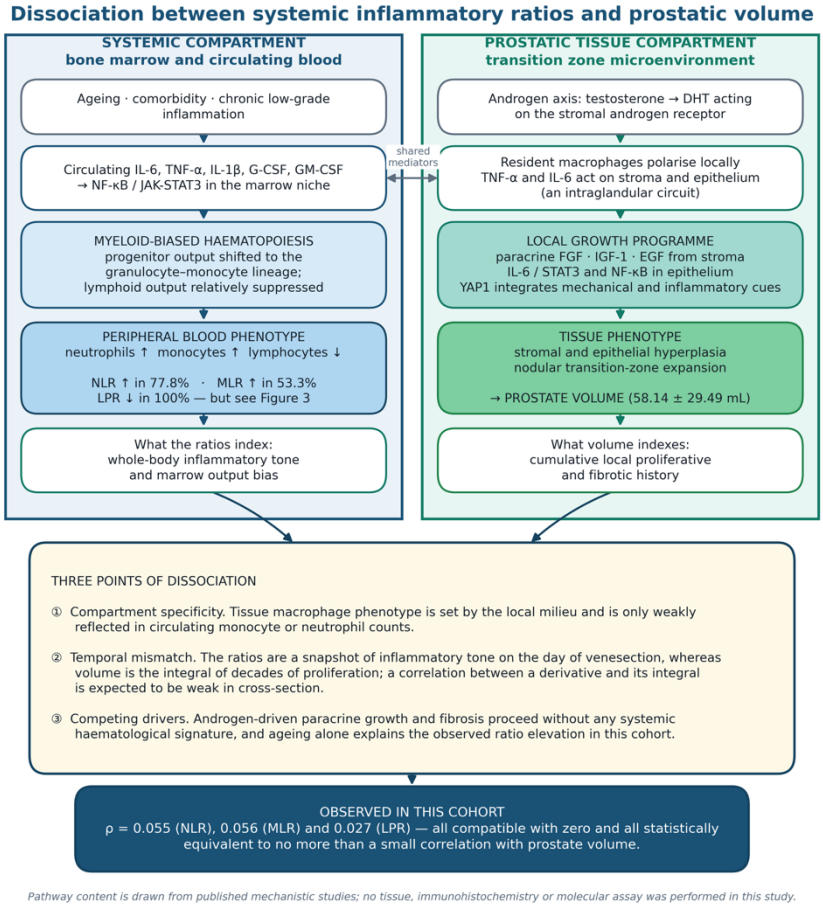

Figure 6. Systemic and prostatic inflammatory compartments and the three points at which they dissociate. The two compartments share mediators but not circuitry, which explains why a peripheral blood count indexes whole-body inflammatory tone rather than glandular size.

In the prostatic compartment the same mediators are present but the circuitry is local. Resident macrophages polarise within the tissue, TNF-α and IL-6 act on stromal fibroblasts and epithelium, and YAP1 integrates inflammatory with mechanical cues to promote survival and fibrosis.[11,8] Androgen-independent stromal proliferation proceeds through growth-factor pathways that leave no systemic haematological signature,[9] and the IL-6/IL-6R axis can be driven by inflammatory foci wholly outside the prostate.[12] Three consequences follow. Compartment specificity means that tissue macrophage phenotype is set locally and is only weakly reflected in circulating counts. Temporal mismatch means that a ratio measured on the day of venesection is a snapshot of inflammatory tone, whereas prostate volume is the accumulated integral of decades of proliferation and fibrosis, so a cross-sectional correlation between a derivative and its integral should be expected to be weak. Competing drivers mean that in a cohort of mean age 70.31 years the observed ratio elevation is adequately explained by ageing alone. Read this way, the finding is not a failure to detect an expected signal but the outcome that the biology predicts.

What inflammation does predict

None of this argues that inflammation is unimportant in BPH. It argues that the outcome against which peripheral markers should be tested is symptom burden and progression rather than gland size. Circulating IL-6 is associated with LUTS severity in men with BPH,[10] plasma IL-6 and TNF-α track prostatic disease activity,[13] and complete blood count indices are associated with IPSS category[16] and with LUTS in population data.[17] Histological prostatic inflammation, assessed in resected tissue from 51 men undergoing transurethral resection, correlated with the persistence of overactive bladder symptoms after surgery rather than with the size of the gland removed,[44] and NLR distinguishes inflammatory bladder conditions from overactive bladder in the absence of any prostatic contribution at all.[45] The constructive reading of our data is therefore a redirection of the question rather than an abandonment of it.

Implications for surgical practice

For the operating surgeon the implication is immediate. Prostate volume determines the selection between transurethral resection, endoscopic enucleation, robot-assisted or open simple prostatectomy and the minimally invasive alternatives,[5,6,7] and this study establishes that a complete blood count contributes nothing to estimating it. Ultrasonographic measurement remains mandatory and cannot be approximated by an inflammatory index; where accuracy matters most, transition-zone volume on cross-sectional imaging predicts enucleation weight better than any total-volume measurement.[36] Two positive implications nonetheless follow. The ratios retain descriptive value: a man presenting with an NLR above 2, an elevated MLR and a depressed LPR has demonstrable systemic inflammatory activation, which is worth knowing in a patient about to undergo anaesthesia and instrumentation even though it says nothing about his prostate. And the prevalence figures are themselves a usable baseline: 77.8% NLR elevation in an unselected elderly surgical population at this institution is a reference point against which future perioperative work in this catchment can be calibrated. The clinical and research pathway that follows from these findings is summarised in **Figure 7**.

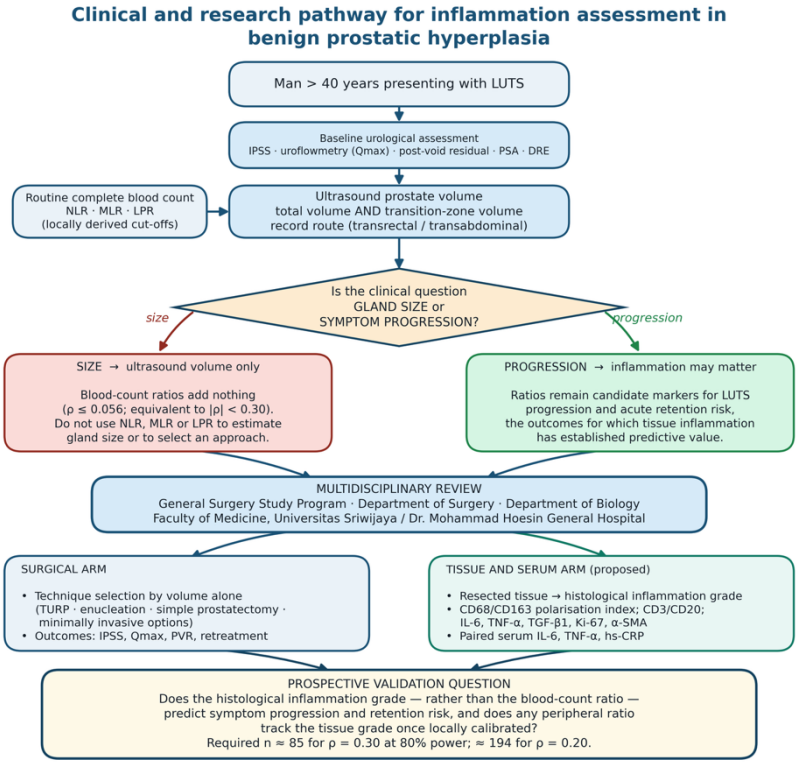

Figure 7. Proposed clinical and research pathway for inflammation assessment in benign prostatic hyperplasia. The decision node separates the question of gland size, for which blood-count ratios have no role, from the question of symptom progression, for which they remain candidate markers, and specifies the prospective validation study that follows.

Strengths and limitations

The principal strengths are the completeness of the analysed set, with 45 of 45 men analysed and no missing values; the rigour of the exclusion criteria, in particular the exclusion of prostate cancer, acute prostatitis, recent antibiotics and 5-α-reductase inhibitors, each of which could independently distort either the blood count or the measured volume; and the pre-specified equivalence framework, which converts a null result into a bounded statement about what the data can exclude.

The limitations are substantial. Sample size is the first: at 45 subjects the study could detect only a large correlation, and

while it excludes a moderate association for NLR and LPR it cannot exclude a small one. Design is the second: a cross-sectional study cannot establish temporality, and the mismatch between a same-day marker and a cumulative anatomical outcome is intrinsic to it. Outcome definition is the third and probably the most consequential: total prostate volume was measured, but transition-zone volume and intravesical prostatic protrusion — the compartments in which an inflammatory effect is most plausible[33,36,34] — were not, and neither were IPSS, maximum flow rate, post-void residual volume or prostate-specific antigen. Measurement is the fourth: volumes were taken from routine radiology reports without standardisation of route, operator or formula, and ellipsoid estimation overestimates volume relative to manual planimetry with meaningful inter-rater variability;[24,25] automated transabdominal methods now offer better reproducibility and would be preferable prospectively.[26] Confounding is the fifth: age, comorbidity and smoking were recorded but could not be adjusted for. Because age raises both prostatic volume and the myeloid-biased ratios,[29,30] it acts as a positive confounder, so unadjusted estimates should be biased away from the null and the observed correlations are plausibly upper bounds on the true adjusted association. Marker scope is the sixth: local prostatic mediators and the composite systemic immune-inflammation index, which has outperformed NLR in several datasets,[18,17] were not measured. Threshold validity is the seventh, as set out for LPR above. Finally, this is a single-centre study in one South Sumatran referral population, and the demographic distinctiveness that gives it novelty also bounds its direct generalisability.

Future directions

The next study is specified rather than gestured at. We propose a prospective cohort at this institution recruiting at least 85 men — the number required to detect $\rho = 0.30$ at 80% power, and preferably 194 to reach $\rho = 0.20$ — in which total volume, transition-zone volume and intravesical prostatic protrusion are measured by a standardised protocol using a single route and, where feasible, automated segmentation;[26,36] IPSS, maximum flow rate and post-void residual volume are recorded; the complete blood count is supplemented by serum IL-6, TNF-α and high-sensitivity C-reactive protein; and, in men proceeding to surgery, resected tissue is graded histologically for inflammation and stained for CD68 and CD163 to yield a macrophage polarisation index, for CD3 and CD20 to characterise the lymphocytic infiltrate, and for IL-6, TNF-α, TGF-β1, Ki-67 and α-smooth-muscle actin. Each marker would be scored by staining intensity on a 0 to 3+ scale combined with the percentage of positive cells as an H-score, with Ki-67 counted in three hot-spot high-power fields and all grading performed blind to the blood counts. The primary endpoint would be the association between the histological inflammation grade and the CD163:CD68 polarisation index; secondary endpoints would be the association of each peripheral ratio with the tissue grade, and the association of the tissue grade with symptom progression and acute urinary retention over 12 to 24 months. Reference intervals for all three ratios should be derived locally within the same protocol rather than imported, which is the direct practical lesson of the threshold behaviour reported here.

5. Conclusion

In 45 men with benign prostatic hyperplasia at Dr. Mohammad Hoesin General Hospital, Palembang, systemic inflammation was highly prevalent — NLR was elevated in 77.8% and MLR in 53.3% — yet neither these markers nor the lymphocyte-to-platelet ratio was associated with ultrasonographic prostate volume. All three correlations lay close to zero, and the data are statistically equivalent to no more than a small association for NLR and LPR and to no more than a moderate one for all three markers. Complete blood count-derived inflammatory ratios should therefore not be used to estimate prostatic size or to select an operative approach, and ultrasonographic measurement remains indispensable. The uniformly low lymphocyte-to-platelet ratio reflected a threshold outside the attainable range of the ratio as computed, a caution that applies wherever such cut-offs are transferred between populations. Future work should test peripheral inflammatory markers against transition-zone volume, intravesical prostatic protrusion and symptom progression rather than against total gland size.

6. Declarations

Ethics Approval and Consent to Participate

This study was granted ethical exemption by the Ethical Committee of Dr Mohammad Hoesin General Hospital, Palembang, Indonesia (Ref. No. DP.04.03/D.XVII.9.4/ETIK/144/2026). Because the study was a retrospective review of existing medical records involving no patient contact and no identifiable data leaving the institution, the committee waived the requirement for individual written informed consent. All records were pseudonymised before analysis, and the study was conducted in accordance with the Declaration of Helsinki.

Consent for Publication

Not applicable. No individually identifiable patient data, images or clinical details are reported.

Availability of Data and Materials

The aggregated data supporting the findings of this study, including all rank statistics from which the reported analyses can be reproduced, are contained within the article and its tables. Individual-level records are held at Dr. Mohammad Hoesin General Hospital and are not publicly available because of institutional data-governance requirements; reasonable requests should be directed to the corresponding author and are subject to institutional and ethics committee approval.

Artificial Intelligence Use Statement

None declared. No AI tool was used to generate or analyse research data. The authors reviewed and approved the final content and take full responsibility for it.

Author Contribution Statement

VMNT conceived the study, collected and curated the data, performed the statistical analysis and drafted the manuscript. FPH supervised the clinical and surgical aspects of the study, contributed to the design and to the interpretation of the urological findings, and critically revised the manuscript. ZM contributed to the study design and the methodological framework, supervised the interpretation of the haematological and inflammatory data, and critically revised the manuscript. All authors read and approved the final manuscript and agree to be accountable for all aspects of the work.

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Conflict of Interest

The authors declare that they have no competing interests.

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