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Discriminating ability of erythrocyte sedimentation rate and derived inflammatory indices for metabolic syndrome among adults in Eastern Uganda: a case-control study

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Abstract

Background

Metabolic syndrome (MetS) is now a major public health challenge driven by chronic low-grade inflammation. Erythrocyte sedimentation rate (ESR) and derived indices may inexpensively identify MetS. This study evaluated the discriminating performance and association of ESR and selected inflammatory indices with MetS among adults in eastern Uganda.

Methods

A hospital based matched sequential case-control study was conducted among 228 adults (114 MetS,114non MetS) in April 2026 in eastern Uganda. MetS was defined by the harmonized Joint Interim Statement (JIS) definition. Derived inflammatory indices including ESR-to-monocyte ratio (EMR) and ESR/WBC ratio were calculated based on the hospital laboratory normal ranges. Receiver operation curve (ROC) analysis and Logistic regression analysis were performed in STATA 14. Statistical significance was considered at $p < 0.05$.

Results

ESR had a moderately high AUC for MetS (Males; AUC = 0.64: cutoff=15mm/hr, Females; AUC = 0.77: cutoff=25mm/hr). Elevated ESR was strongly associated with MetS (Males; cOR 5.01, 95% CI: 1.06–26.12; p = 0.013, females; cOR 13.82 95%CI:4.6-41.12; p < 0.001). After multivariate adjustment, elevated ESR remained independently associated with MetS (Males; aOR 5.4, 95% CI: 1.24–34.63; p = 0.004, Females: aOR 14.1, 95% CI: 1.57–126; p = 0.018). Elevated EMR and ESR/WBC ratio (aOR 3.6, p = 0.046) and (aOR 4.6, p = 0.019) were also independently associated with MetS.

Conclusion

ESR can moderately discriminate Mets individuals from non-MetS individuals in eastern Uganda. Derived ESR based indices are strongly associated with Metabolic syndrome in this population. Further validation of their clinical utility in MetS is recommended.

Background

Metabolic syndrome (MetS) is a major global non-communicable disorder characterized by a cluster of interconnected cardiometabolic abnormalities including central obesity, hypertension, dyslipidemia, insulin resistance, and hyperglycemia[1].The most current definition of Mets by harmonized Joint Interim Statement (JIS) definition between international diabetes federation(IDF) and American Heart Association/National Heart, Lung, and Blood Institute (AHA/NHLBI) is the presence of any 3 of the 5 components; diabetes, hypertension, reduced high density lipoproteins(HDL),high triglycerides and high waist circumference[2].

Metabolic syndrome is now a clinical diagnosis by the World Health Organization (WHO)[3].Although there is widespread inertia in making this diagnosis, its effect on morbidity and mortality risk is more than the risk

associated with the individual MetS components. Metabolic syndrome identifies a specific subgroup of people with common biological processes that gradually predispose them to debilitating clinical outcomes because of its strong association with cardiovascular disease, type 2 diabetes mellitus, stroke, and premature mortality[4].Although the pathways are still unraveling, it is agreed that systemic inflammation is one of the drivers of the metabolic dysfunctions[5 6].Individuals with metabolic syndrome and obesity have increased concentrations of inflammatory mediators in their adipose tissues[7].

Metabolic syndrome was initially more prevalent in the countries of the west but has recently become a global problem[8].Large cohort studies have reported a global MetS prevalence ranging between 12.5% and 31.4%, highlighting considerable regional and demographic variation[9].In Africa, the prevalence of MetS has been rising from 0–1% at the start of the 21st century to 50% by 2020[10].In southwestern Uganda a rural household based cross-sectional survey found the prevalence of metabolic syndrome of 19%[11]. While Muyanja and colleagues found it at 58% in people living with HIV and on stable antiretroviral treatment (ART)[12]. Studies have linked the global rise in metabolic syndrome to western lifestyle particularly of diet and physical inactivity.

Despite the increasing burden of metabolic syndrome in Uganda, there is limited use of inflammatory markers to screen for inflammation among metabolic syndrome patients. ESR is a known inexpensive marker of chronic sub-clinical inflammation[13 14].Considered as a progressive multisystem continuum, it is important to identify such inflammation early enough to manage and reduce the risk of complications[15].Erythrocyte sedimentation rate (ESR) and monocyte count are among the routinely available inflammatory parameters that may reflect the chronic inflammatory processes underlying MetS. While existing research suggests potential association of ESR and monocytes with several metabolic syndrome components[16], the clinical utility of ESR and the ESR- to -monocyte count has not been studied nor clarified among adults with MetS in Uganda.

ESR reflects the rate at which erythrocytes sediment in plasma under the influence of acute-phase reactants such as fibrinogen and immunoglobulins, which promote rouleaux formation and accelerated red blood cell aggregation[17].Compared to C-reactive protein (CRP), ESR rises and normalizes more slowly because it is influenced not only by acute-phase proteins but also by non-acute phase proteins and changes in plasma viscosity. Consequently, ESR may better reflect persistent low-grade inflammatory states characteristic of metabolic syndrome[13].

Studies among patients with rheumatoid arthritis reported significantly higher ESR levels in individuals with concomitant metabolic syndrome compared to those without MetS[18 19]. These findings collectively suggest that ESR may reflect the heightened inflammatory burden associated with metabolic dysregulation.

Several studies have demonstrated significant associations between metabolic syndrome and hematologic abnormalities. In a cohort study among Iranian men, Ahmadzadeh et al. (2018) reported that metabolic syndrome was significantly associated with elevated platelet count, hematocrit, hemoglobin concentration, and total white blood cell count[20] .These findings suggest that chronic inflammatory activation in MetS may influence both erythroid and myeloid hematopoietic pathways.

Platelet indices have also shown potential utility in identifying metabolic syndrome and diabetes-related inflammation. In a comparative cross-sectional study conducted in Ethiopia, Haile et al. (2022) demonstrated significant associations between platelet indices and metabolic syndrome among diabetic patients. Notably,

mean platelet volume (MPV) demonstrated high discriminatory performance for metabolic syndrome with an area under the curve (AUC) of 0.859, suggesting that hematologic indices may possess clinically useful diagnostic value[22].

Early detection of chronic low-grade inflammation is critical in interrupting the progression of metabolic syndrome (MetS) and its downstream complications. This study aimed to investigate the discriminative ability of ESR,ESR -to-monocyte ratio and association of hematologic inflammatory biomarkers with metabolic syndrome and to inform their clinical role in early identification and risk stratification of patients with MetS in a resource limited country of Uganda.

Methods

Design and study setting

A hospital based sequential frequency matched cumulative case control study was conducted at Jinja regional referral hospital (JRRH) in jinja city in eastern Uganda from January 2026 to April 2026. The hospital serves as a referral center for over 10 districts serving a population over 4 million. JRRH serves over 56,000 out-patients per month. The target population was adults above 18 years of age with metabolic syndrome defined as the presence of 3 out of 5 components of metabolic syndrome attending the NCD clinic as cases and controls as normal individuals without the disease components of metabolic syndrome drawn from the outpatient clinics at the hospital. We excluded individuals who did not give informed consent, pregnant women, people with autoimmune disease disorders, rheumatological disorders, people who had cancer and those on chronic immunosuppressive treatment and Controls with two or more of the component diseases of metabolic syndrome or any systemic inflammatory illnesses.

Sample size and sampling procedure

To detect significant level of association of an odds ratio of 2.3, comparing independent proportions among cases and controls with high hematological parameters and indices, Fleiss formula for sample size of a case control study was used to obtain a representative sample size. Cases to control ratio was1:1, with expected exposure in controls at 30% which was estimated from a comparative cross-sectional study of hematologic indices in metabolic syndrome conducted in Ethiopia[22] with 95% confidence interval and a power of 80%. After controlling for possible data loss at 20% we obtained representative sample size of 228(114 cases and 114 controls)

The participants were enrolled by consecutive sampling. Cases were recruited sequentially from the specialized NCD clinic, followed by control recruitment from the general outpatient clinics serving the same hospital catchment area. The multi clinic approach was strategically selected to prevent overlapping shared metabolic risk factors that occur with intra clinic NCD controls. To ensure a shared base, the general outpatient clinic was verified as the primary referral source for the NCD clinic. Temporal bias was minimized by restricting data collection to the same period of 3 months. Frequency matching by age (± 5yrs) and sex was applied during the outpatient parallel consecutive sampling phase. A rigorous screening protocol was implemented to prevent selection bias, controls with signs of acute infections, individuals with autoimmune disease disorders, rheumatological disorders, people who had cancer and those on chronic immunosuppressive treatment and

Controls with two or more of the component diseases of metabolic syndrome or any systemic inflammatory illnesses were excluded.

Instruments and variables

Metabolic syndrome as the primary outcome was defined as presence of any 3 of these 5 components; fasting blood glucose ≥ 5.5mmol/l or a diagnosis of diabetes, high density lipoprotein(HDL) cholesterol < 1.0mmol/l(40mg/dl in men and < 1.3mmol/l(50mg/dl) in women, Triglycerides ≥ 150 mg/dl or the patient taking treatment for elevated triglycerides, measured blood pressure > 130/85 mmhg or the patient taking treatment for hypertension and waist circumference of > 94cm in males and > 80 cm in females for population of the sub Saharan African region. Trained nurses who worked as research assistants collected the data using a pretested and validated questionnaire. The predictor variables of the study were demographic factors, medical factors, Lifestyle factors that were obtained by face-to-face individual interviews and measurement. hematological factors were obtained by laboratory testing. Indices like ESR to white blood cell count ratio(ESR/WBC),monocyte to leucocyte(MLR) ratio, ESR-to-monocyte count ratio (EMR),neutrophil to lymphocyte ratio(NLR),platelet to lymphocyte ratio(PLR),monocyte to lymphocyte ratio(MLR).ESR based indices were calculated based on adjusted ESR values by age. All hematologic parameter normal ranges and indices were calculated based on the Jinja regional referral hospital laboratory reference ranges.

Waist circumference was measured using a stretch-resistant tape with the respondent in standing position. The tape was placed snugly around an exposed abdomen, horizontal to the ground and at the mid-point between the anterior superior ischial spine and the lowest palpable rib at the end of normal inspiration. The hip circumference was measured around the widest points of the buttocks. The measurements were taken according to the WHO report on expert consultation protocol for waist circumference and hip circumference[23].Blood pressure was measured using the automatic blood pressure machine(Omron HEM FL31)according to the protocol described by European society of hypertension for office blood pressure measurement[24].The height and weight were measured using calibrated equipment (SECA 777 series) to the nearest centimeter and kilogram according to standard protocols described by the WHO steps instrument tool, and recalibrated after each measurement[25].Blood samples were obtained according to WHO guidelines on venipunctures by trained research assistants after the respondent has fasted for at least 8 hours preferably in the early morning[26].

Laboratory tests for, lipid profile, complete blood count were performed by the laboratory technician using the chemistry machine blood analyzer (Cobus pure c303 analyzer) and complete blood count (CBC) machine(Sysmex XN 550).Erythrocyte sedimentation rate was performed using the Westergren method within 4 hours of collecting the sample using the disposable ESR pipettes with triple plug tip design (Boenmed ESR Westergren pipettes)

Data analysis

Data was entered into Microsoft access to create a database that was later exported to STATA version 14(College Station, Tx 77845) for analysis. Normality of distribution was tested using the Shapiro wilk, student t test analysis and Manni Whitney u test were performed for the continuous variables while Chi-square test analysis was performed for categorical variables to compare differences in mean and medians between cases and controls with appropriate effect sizes. Using receiver operating curve (ROC) analysis we assessed the

discriminatory performance of ESR, and ESR-to-monocyte count (EMR) for metabolic syndrome. The optimal cutoffs were determined using the Youden index. The area under the curve values (AUC) were determined with the 95% confidence interval at statistical significance of $p < 0.05$ with their sensitivity and specificity. The ROC curves for ESR, and EMR were drawn and graphically compared with each other by sex and presented in the results.

Univariate and bivariate logistic regression analysis was performed to assess the association between hematological parameters and indices with metabolic syndrome. The measures of association, that is, crude and adjusted odds ratios were calculated. A multivariate logistic regression model was used for the analysis after checking for multicollinearity to avoid correlated predictors using variance inflation factor (VIF). The model was adjusted for age, sex alcohol use, smoking, presence of comorbidities, C - reactive protein and creatinine level to determine the parameters that are independently associated with metabolic syndrome.

Results

Table 1
Demographic and anthropometric characteristics of the participants with metabolic syndrome and healthy controls

Age (mean ± SD)	**No -metabolic syndrome(N = 114)** **n (%)**	**Metabolic** **syndrome (N = 114)** **n (%)**	**P value**	**Effect size**
	49 ± 13.59	**57 ± 10.98**	**< 0.001**	**0.59**
Sex			< 0.001	0.24
Male	47(41.23)	22(19.30)		
Female	67(58.77)	92(80.70)		
Residence			0.002	0.23
Rural	41(35.96)	45(39.47)		
City	22(19.30)	5 (4.39)		
Peri urban	51(44.74)	64 (56.14)		
Marriage status			< 0.001	0.30
Married living with a partner	77(67.54)	64(56.14)		
Divorced/separated	15(13.16)	42(36.84)		
Single	22(19.30)	8 (7.02)		
Smoking status			< 0.001	0.28
Current	5(4.39)	0		
Previous/stopped	16(14.04)	2 (1.75)		
Never smoked	93(81.58)	112(98.25)		
Alcohol use			0.225	0.11
Yes	17(14.91)	9(7.89)		
No	80(70.18)	84(73.68)		
Stopped	17(14.91)	21(18.42)		
Family history (first degree)			< 0.001	0.32
Diabetes	16 (14.04)	39(34.21)		
Hypertension	34(29.82)	44(38.60)		

Age (mean ± SD)	**No -metabolic syndrome(N = 114)** **n (%)**	**Metabolic** **syndrome (N = 114)** **n (%)**	**P value**	**Effect size**
	49 ± 13.59	**57 ± 10.98**	**< 0.001**	**0.59**
Cancer	8(7.02)	2 (1.75)		
Allergy/Atopy/asthma	2(1.75)	0		
None	54(47.37)	29(25.44)		
Comorbidities			0.021	0.22
HIV/AIDS	8(7.02)	2(1.75)		
Heart failure	1(0.88)	3 (2.63)		
Asthma/COPD	6(5.26)	2(1.75)		
None	97(85.09)	98(85.96)		
Others	2(1.75)	9(7.89)		
Waist circumference(cm)	79 (11)	104(17)	< 0.001	0.77
Hip circumference(cm)	92(11)	110(13)	< 0.001	0.69
BMI			< 0.001	0.53
Normal	69(60.53)	20(17.54)		
Overweight	40(35.09)	45(39.47)		
Obese	3(2.63)	29(25.44)		
Grossly obese	2(1.75)	20(17.54)		
Systolic BP (mm/hg)	120(13)	146(23)	< 0.001	0.60
Diastolic BP (mm/hg)	75.5(9)	87(15)	< 0.001	0.53

Data is presented as Median (IQR) and Mean ± SD

We contacted 258 eligible participants,123 cases and 125 controls. A total of 9 cases (7.3%) did not meet the inclusion criteria and were excluded leaving 114 cases. And for the controls 11(8.8%) individuals did not meet the inclusion criteria and were excluded leaving a total of 114 controls. We therefore analyzed data for 228 participants. The mean age of individuals with metabolic syndrome was significantly older than those without metabolic syndrome (57 ± 10.98 vs 49 ± 13.59 p = < 0.001 d = 0.59).Most of the participants were female with a higher percentage having metabolic syndrome compared to those without metabolic syndrome(92(80.70%) vs

67(58.77%).Majority of the participants had never smoked in either groups (85.58% of non-MetS and 98.25% of MetS).There was a significant difference in first degree family history of diabetes and hypertension reported by individuals with metabolic syndrome compared to those without metabolic syndrome ($p < 0.001$,$r = 0.32$).The median waist circumference and hip circumference were significantly higher in metabolic syndrome individuals than normal individuals ($p = < 0.001$ $r = 0.77$) and $p = < 0.001$ $r = 0.69$), respectively. Participants with metabolic syndrome had higher levels of BMI and were more obese or grossly obese compared to the normal participants and this difference was statistically significant ($p < 0.001$ $v = 0.53$). Table 1 above summarizes the demographic and anthropometric characteristics of the participants with MetS and those without MetS.

In Fig. 1 above, the distribution of Erythrocyte sedimentation rate (ESR) stratified by metabolic syndrome, sex and age dichotomized at 50 years is presented. The participants with metabolic syndrome had consistently higher ESR values compared to those without metabolic syndrome across the sex and age at 50 years. The difference in median ESR was highest among females above 50 years old where median ESR was elevated above the normal cut off range of 30 mm/hr for this age with an elevated Interquartile range (median = 32, IQR = 17–56). A wider spread of ESR was seen among individuals with MetS and extreme outlier values, were notably higher among females than males. In individuals without metabolic syndrome, the ESR values were not only lower but were clustered tightly. Across the sex and age categories, all participants with metabolic syndrome had a median ESR above the clinically relevant normal cutoffs for age and sex, and this was more commonly seen in older individual.

Table 2

; The AUC values of ESR for discriminating between metabolic syndrome and non-metabolic syndrome categorized by sex, sample derived ESR cut offs and adjusted ESR cut offs.

Variable	MetS n(%)	No MetS n(%)	Se (%)	Spe (%)	Cut-off values (by Youden index)	AUC (95%CI)	DOR (95% CI)	P value
ESR **(mm/hr.)**								
Male Elevated	13(59.09)	14(29.79	59.1	70.21	15	0.64 (0.52–0.77)	3.4 (1.04–11.22)	0.020
Normal	9(40.91)	33(70.21)						
ESR (mm/hr.)								
Female: Elevated	56(60.87)	4(5.97)	60.8	94	25	0.77 (0.74–0.88)	24.5 (7.92–98.5)	< 0.001
Normal	36(39.13)	63(94.03)						
EMR								
Male: Elevated	14(63.64)	14(29.79)	63.6	70.2	25	0.67(0.54–0.79)	4.12(1.25–13.9)	0.007
Normal	8(36.36)	33(70.21)						
Female: Elevated	72(78.26)	23(34.33)	78.3	65.6	42	0.72(0.65–0.79)	6.88(3.21–14.88)	< 0.001
Normal	20(21.74)	44(65.67)						
			Adjusted ESR cut offs (male = age/2, female = age+10/2)					
ESR (mm/hr.)								
Male: Elevated	7(31.82)	4(8.51)	32	91.5	27	0.61(0.50–0.72)	5.02(1.06–26.12)	0.0137
Normal	15(68.18)	43(91.49)						
ESR (mm/hr):								
Female: elevated	43(46.74)	4(5.97)	47	94	46	0.70(0.64–0.76)	13.8(4.5–55.7)	< 0.001
Normal	49(53.26)	63(94.03)						
EMR								

NOTE: Se; Sensitivity, Sp; Specificity, AUC; Area under the curve, ESR; Erythrocyte sedimentation rate, EMR;ESR-to-Monocyte count ratio. DOR; diagnostic odds ratio= (True positives x True negative) / (False positives x False negatives)

Variable	MetS n(%)	No MetS n(%)	Se (%)	Spe (%)	Cut-off values (by Youden index)	AUC (95%CI)	DOR (95% CI)	P value
Male: elevated	14(63.64))	14(29.79)	63.6	70.2	45	0.66(0.54–0.79)	4.15(1.25–13.9)	0076
normal	8(36.36)	33(70.21)						
Female: elevated	47(51.09)	45(48.91)	85.4	56.7	77	0.69(0.63–0.76)	7.7(3.15–20.5)	< 0.001
normal	8(11.94)	59(88.06)						

NOTE: Se; Sensitivity, Sp; Specificity, AUC; Area under the curve, ESR; Erythrocyte sedimentation rate, EMR;ESR-to-Monocyte count ratio. DOR; diagnostic odds ratio= (True positives x True negative) / (False positives x False negatives)

The discriminatory ability of erythrocyte sedimentation rate (ESR) and ESR-to-monocyte ratio (EMR) for differentiating participants with metabolic syndrome (MetS) from controls was assessed using receiver operating characteristic (ROC) analysis stratified by sex and the results are presented in Table 2 and Fig. 2 above.

Among males, ESR demonstrated moderate discriminatory ability with an area under the curve (AUC) of 0.64 (95% CI: 0.52–0.77, p = 0.020) at an optimal cut-off value of 15 mm/hr determined using the Youden index. This cut-off yielded a sensitivity of 59.1% and specificity of 70.2%, with a diagnostic odds ratio (DOR) of 3.4 (95% CI: 1.04–11.22). Similarly, EMR demonstrated moderate discriminatory performance among males with an AUC of 0.67 (95% CI: 0.54–0.79, p = 0.007), sensitivity of 63.6%, specificity of 70.2%, and DOR of 4.12 (95% CI: 1.25–13.9).

Among females, ESR showed good discriminatory ability with an AUC of 0.77 (95% CI: 0.74–0.88, p < 0.001) at a cut-off value of 25 mm/hr. The sensitivity and specificity were 60.8% and 94.0%, respectively, with a high DOR of 24.5 (95% CI: 7.92–98.5). EMR among females also demonstrated acceptable discriminatory performance with an AUC of 0.72 (95% CI: 0.65–0.79, p < 0.001), sensitivity of 78.3%, specificity of 65.6%, and DOR of 6.88 (95% CI: 3.21–14.88).

When age-adjusted ESR cut-offs were applied (male = age/2; female = age+10/2), the discriminatory performance of ESR remained statistically significant though slightly reduced. Among males, adjusted ESR demonstrated an AUC of 0.61 (95% CI: 0.50–0.72, p = 0.0137), sensitivity of 32%, specificity of 91.5%, and DOR of 5.02 (95% CI: 1.06–26.12). In females, adjusted ESR demonstrated an AUC of 0.70 (95% CI: 0.64–0.76, p < 0.001), sensitivity of 47%, specificity of 94%, and DOR of 13.8 (95% CI: 4.5–55.7). Similarly, adjusted EMR maintained moderate discriminatory performance. In males, the AUC was 0.66 (95% CI: 0.54–0.79, p = 0.0076), while among females the AUC was 0.69 (95% CI: 0.63–0.76, p < 0.001).

Overall, ESR and EMR demonstrated moderate to good discriminatory ability for metabolic syndrome, with stronger performance observed among females compared to males.

Table 4
The association of hematologic parameters and indices with presence of metabolic syndrome versus no metabolic syndrome.

Hematologic Parameter	Category	MetS n (%)	No MetS n (%)	cOR (95% CI)	P-value
WBC ×10³/µL					
	4.0–11.0 (Normal)	106(92.98)	101(88.6)	Ref	
	< 4.0 (Leukopenia)	8(7.02)	13(11.4)	0.58(0.202–1.61)	0.252
	> 11.0 (Leukocytosis)	0	0	0	
PLT ×10³/µL	150–450 (Normal)	105(92.11)	102(89.47)	ref	
	< 150 (Thrombocytopenia)	8(7.02)	11(9.65)	0.706(0.27–1.8)	0.474
	> 450 (Thrombocytosis)	1(0.88)	1(0.88)	0.97(0.05–15.7)	0.984
Hb g/dL)					
Male (n = 64)	Normal hemoglobin	17(80.95)	37(86.05)	Ref	
	Anemia	4(19.05)	6(13.95)	1.45(0.26–7.04)	0.60
Female (158)	Normal hemoglobin	84(92.31	63(94.03)	ref	
	Anemia	7(7.69)	4(5.97)	1.31(0.368–4.67)	
Monocytes ×10³/µL	< 0.2 (Low)	5(4.39)	3(2.63)	1.69(0.32–11.16)	0.472
	0.2–1.0 (Normal)	109(95.61)	111(97.37)	ref	
	> 1.0 (Monocytosis)	0	0		
Neutrophils ×10³/µL	< 1.5 (Neutropenia)	9(7.89)	10(8.77)	0.89(0.34–2.2)	0.811
	1.5–7.5 (Normal)	103(90.35)	102(89.47)	Ref	
	> 7.5 (Neutrophilia)	2(1.75)	2(1.75)	0.99(0.13–7.16)	0.992
Lymphocytes ×10³/µL	< 1.0 (Lymphopenia)	0	5(4.39)	0	
	1.0–4.0 (Normal)	110(96.49)	108(94.74)	ref	
	> 4.0 (Lymphocytosis)	4(3.51)	1(0.88)	3.9(0.43–35.7)	0.225
MPV (fL)	< 7.5 (Low)	39(34.21)	44(38.60)	0.844(0.483–1.47)	0.551
	7.5–11.5 (Normal)	63(55.26)	60(52.63)	Ref	
	> 11.5 (High)	12(10.53)	10(8.77)	1.14(0.45–2.8)	0.774
MCV (fL)	< 80 (Microcytic)	26(22.81)	24(21.05)	1.12(0.59–2.1)	0.722

Hematologic Parameter	Category	MetS n (%)	No MetS n (%)	cOR (95% CI)	P-value
	80–100 (Normocytic)	87(76.32)	90(78.95)	Ref	
	> 100 (Macrocytic)	1(0.88)	0	0	
RDW-CV (%)	≤ 14.5 (Normal)	87(76.32)	96(84.21)	Ref	
	> 14.5 (Elevated)	27(23.68)	18(15.79)	1.655(0.81–3.42)	0.13
RDW-SD (fL)	≤ 46 (Normal)	93(81.58)	88(77.19)	Ref	
	> 46 (Elevated)	21(18.42)	26(22.81)	0.76(0.37–1.52)	0.413
MCH (pg)	< 27 (Low)	53(46.49)	39(34.21)	1.66(0.97–2.85)	0.065
	27–33 (Normal)	58(50.88)	71(62.28)	Ref	
	> 33 (High)	3(2.63)	4(3.51)	0.92(0.19–4.26)	0.913
MCHC (g/dL)	< 32 (Low)	66(57.89)	49(42.98)	1.71(1.0-2.9)	0.046
	32–36 (Normal)	48(42.11)	61(53.51)	Ref	
	> 36 (High)	0	4(3.51)	0	
Adjusted ESR (mm/hr)					
Male	< 27(normal)	15(68.18)	43(91.49)	ref	
	≥ 27(elevated)	7(31.82)	4(8.51)	5.01(1.06–26.12)	0.014
female	< 46(normal)	49(53.26)	63(94.03)	Ref	
	≥ 46(elevated)	43(46.74)	4(5.97)	13.82(4.6-41.12)	< 0.001
EMR					
male	< 45(normal)	8(36.36)	33(70.21)	ref	
	≥ 45(elevated)	14(63.64)	14(29.79)	4.13(1.41–12.03)	0.009
Female	< 77(normal)	45(48.91)	59(88.06)	ref	
	≥ 77(elevated)	47(51.09)	8(11.94)	7.70(3.31–17.91)	< 0.001
NLR	< 3.6 (Low inflammatory risk)	111(97.37)	108(94.74)	Ref	
	≥ 3.6 (High inflammatory risk)	3(2.63)	6(5.26)	0.48(0.07–2.3)	0.307
PLR	< 120(Low inflammatory risk)	78(68.42)	56(49.12)	Ref	

Hematologic Parameter	Category	MetS n (%)	No MetS n (%)	cOR (95% CI)	P-value
	≥ 120 (High inflammatory risk)	36(31.58)	58(50.88)	0.44(0.25–0.79)	0.0031
MLR	< 0.24 (Low inflammatory burden)	99(86.84)	77(67.54)	Ref	
	≥ 0.24 (High inflammatory burden)	15(13.16)	37(32.46)	0.32(0.15–0.64)	0.005
ESR/WBC Ratio	< 3.6(normal)	11(50)	41(87.23)	ref	
Male	≥ 3.6(elevated)	11(50)	6(12.77)	6.83(2.06–22.61)	0.002
females	< 6.13(normal)	54(58.70)	64(95.52)	ref	
	> 6.13(elevated)	38(41.30)	3(4.48)	15.01(4.38–51.35)	< 0.001

Table 4 summarizes the results showing the association between hematologic parameters and metabolic syndrome at univariate analysis. Most conventional hematologic parameters including total white blood cell count (WBC), platelet count, hemoglobin level, monocyte count, neutrophil count, lymphocyte count, mean platelet volume, mean corpuscular volume, and RDW-SD were not significantly associated with metabolic syndrome ($p > 0.05$).

However, several inflammatory hematologic indices demonstrated statistically significant associations with metabolic syndrome. Participants with low mean corpuscular hemoglobin concentration (MCHC < 32 g/dL) had higher odds of metabolic syndrome compared to those with normal MCHC levels (cOR 1.71, 95% CI: 1.0–2.9; $p = 0.046$). Elevated adjusted erythrocyte sedimentation rate in males (ESR ≥ 27 mm/hr) was strongly associated with metabolic syndrome, with affected male participants having 5-fold increased odds of metabolic syndrome compared to those with normal ESR values (cOR 5.01, 95% CI: 1.06–26.12; $p = 0.014$).And in the females the odds of metabolic syndrome were approximately fourteen times higher(cOR 13.82,95% CI: 4.6-41.12;$p < 0.001$) with elevated adjusted ESR(≥ 46 mm/hr)

Similarly, male participants with elevated adjusted ESR-to-monocyte ratio (EMR ≥ 35) had significantly increased odds of metabolic syndrome (cOR 4.13, 95% CI: 1.41–12.03; $p = 0.009$),while in females a higher EMR(≥ 77) was also associated with eight times higher odds of metabolic syndrome(cOR 7.70,95% CI:3.31–17.91;$p < 0.001$). A high ESR/WBC ratio in males (≥ 3.6) and females (> 6.13) was also significantly associated with metabolic syndrome (cOR 4.71, 95% CI: 2.5–8.6; $p < 0.001$) and (cOR 15.01 95% CI:4.38–51.35; $p < 0.001$) respectively.

Conversely, elevated platelet-to-lymphocyte ratio (PLR ≥ 120) and elevated monocyte-to-lymphocyte ratio (MLR ≥ 0.24) were associated with reduced odds of metabolic syndrome. Participants with elevated PLR had 56% lower odds of metabolic syndrome compared to those with lower PLR values (cOR 0.44, 95% CI: 0.25–0.79; $p = 0.0031$), while elevated MLR was associated with 68% lower odds of metabolic syndrome (cOR 0.32, 95% CI: 0.15–0.64; $p = 0.005$).

Although low MCH values demonstrated borderline statistical significance (cOR 1.66, 95% CI: 0.97–2.85; p = 0.065), the association did not reach the conventional threshold for statistical significance.

Table 5
Hematologic parameters and indices independently associated with Metabolic syndrome at multivariate analysis.

Hematologic Parameter	Category	No MetS n (%)	MetS n (%)	cOR (95% CI)	P-value	aOR	P-value
WBC ×10³/µL							
	4.0–11.0 (Normal)	101(88.6)	106(92.98)	Ref			
	< 4.0 (Leukopenia)	13(11.4)	8(7.02)	0.58(0.2–1.5)	0.256	0.5(0.084–2.61)	0.388
Lymphocytes ×10³/µL							
	1.0–4.0 (Normal)	108(94.74)	110(96.49)	ref			
	< 1.0 (Lymphopenia)	5(4.39)	0	0			
	> 4.0 (Lymphocytosis)	1(0.88)	4(3.51)	3.9(0.4–35.7)	0.225	3.8(0.05–254)	0.534
RDW-CV (%)							
	≤ 14.5 (Normal)	96(84.21)	87(76.32)	Ref			
	> 14.5 (Elevated)	18(15.79)	27(23.68)	1.65(0.8–3.4)	0.13	0.3(0.06–1.49)	0.142
MCH (pg)							
	< 27 (Low)	39(34.21)	53(46.49)	1.66(0.9–2.9)	0.065	0.7(0.20–3.08)	0.569
	27–33 (Normal)	71(62.28)	58(50.88)	Ref			
	> 36 (High)	4(3.51)	0	0			
ESR (mm/hr)							
Male	Normal	43(91.49)	15(68.18)	ref			
	Elevated	4(8.51)	7(31.82)	5.0(1.1–26.1)	0.014	5.4(1.24–34.63)	0.0.4
Female	Normal	63(94.03)	49(53.26)	ref			
	Elevated	4(5.97)	43(46.74)	13.8(4.5–55.8)	< 0.001	14.1(1.57–126)	0.018
EMR							
Male	< 45(normal)	33(70.21)	8(36.36)	ref			
	≥ 45(elevated)	14(29.79)	14(63.64)	4.1(1.4–12.0)	0.009	5.9(1.41–24.85)*	0.015

Hematologic Parameter	Category	No MetS n (%)	MetS n (%)	cOR (95% CI)	P-value	aOR	P-value
Female	< 77(normal)	59(88.06)	45(48.91)	ref			
	≥ 77(elevated)	8(11.94)	47(51.09)	7.7(3.3–17.9)	< 0.001	9.9(3.32–29.75)	< 0.001
PLR							
	< 120(Low-inflammatory risk)	56(49.12)	78(68.42)	Ref			
	≥ 120 (High-inflammatory risk)	58(50.88)	36(31.58)	0.4(0.3–0.8)	0.0031	1.0(0.28–3.74)	0.960
MLR							
	< 0.24 (Low-inflammatory burden)	77(67.54)	99(86.84)	Ref			
	≥ 0.24 (High-inflammatory burden)	37(32.46)	15(13.16)	0.3(0.2–0.6)	0.005	1.1(0.2–4.9)	0.914
ESR/WBC Ratio							
Male	< 3.6(normal)	41(87.23)	11(50)	ref			
	≥ 3.6(elevated)	6(12.77)	11(50)	6.8(2.1–22.6)	0.002	15.1(2.6–86.2)	0.002
female	< 6.13(normal)	64(95.52)	54(58.70)	ref			
	> 6.13(elevated)	3(4.48)	38(41.30)	15.0(4.4–51.4)	< 0.001	16.8(4.0–70.0)	< 0.001

As shown in Table 5, variables with p-values less than 0.2 at univariate analysis were entered into the multivariate logistic regression model to determine independent predictors of metabolic syndrome. After adjustment for potential confounders; age, sex, CRP, comorbidities, alcohol use, smoking and creatinine levels, elevated ESR remained independently associated with metabolic syndrome in both males and females. Among males, elevated ESR was associated with more than fivefold increased odds of metabolic syndrome (aOR 5.4, 95% CI: 1.24–34.63; p = 0.04). Similarly, females with elevated ESR had approximately 14-fold higher odds of metabolic syndrome compared to females with normal ESR values (aOR 14.09, 95% CI: 1.57–126; p = 0.018).

Elevated EMR (≥ 35) also remained an independent predictor of metabolic syndrome after adjustment for other variables, with affected male participants having 5.9 times higher odds of metabolic syndrome (aOR 5.9, 95% CI: 1.02–13.27; p = 0.046) while affected females had 9.9 times higher odds of metabolic syndrome (aOR 9.9,95% CI:3.32–29.75; p < 0.001). Likewise, a high ESR/WBC ratio (≥ 3.6 in males) independently predicted metabolic syndrome among males, the odds of metabolic syndrome were 15.1 times higher with elevated ESR/WBC ratio (aOR 15.1,95% CI: 2.63–86.18; p = 0.002). And among females, a high ESR/WBC ratio (> 6.13) the odds of

metabolic syndrome were 16.8 times higher than in those with normal ESR/WBC ratio(aOR 16.8,95%CI:4.04–70.02;p < 0.001).

In contrast, associations observed at univariate analysis for PLR, MLR, RDW-CV, MCH, lymphocytosis, and leukopenia lost statistical significance after multivariate adjustment (p > 0.05), suggesting that these parameters were likely confounded by other inflammatory or hematologic variables.

Discussion

In the present study, erythrocyte sedimentation rate (ESR) demonstrated a moderate discriminatory ability for identifying individuals with metabolic syndrome in both sexes. The area under the receiver operating characteristic curve (AUC) for ESR in males was 0.64, with a sensitivity of 59.1% and specificity of 70.21% at an optimal cutoff value of 15 mm/hr determined using the Youden index. Notably, the AUC value for discriminating ability of ESR in males performed less than the established factors; waist circumference (AUC = 0.94) and fasting blood glucose (AUC = 0.93), while in females the ESR AUC for discriminating MetS from controls was 0.77 with a sensitivity of 60.8% and specificity of 94% at an optimum cutoff of 25mm/hr by Youden index.

At the observed sensitivity and specificity, ESR demonstrated a diagnostic odds ratio (DOR) of 3.4 in males, indicating moderate diagnostic performance among males and a diagnostic odds ratio of 24.5 in females indicating a strong diagnostic performance among females. This finding suggests that male individuals with metabolic syndrome were approximately 4 times more likely to have elevated ESR values compared to males without metabolic syndrome and females with metabolic syndrome were 25 times more likely to have elevated ESR values compared to females without metabolic syndrome. These significant diagnostic odds ratios further support the receiver operating characteristic findings.

When compared with adjusted ESR cutoffs, ESR consistently had a discriminatory ability comparable with that from our sample derived cutoffs from the ROC analysis for both males and females (AUC:0.61 vs 0.64) and (AUC:0.70vs 0.77) respectively. These findings reinforce the potential utility of ESR as an inflammatory biomarker associated with metabolic syndrome, suggesting that ESR possesses clinically meaningful discriminatory utility for metabolic syndrome and is higher for female than it is for males.

To the best of our knowledge, there is limited published literature specifically evaluating the discriminatory ability of ESR for metabolic syndrome in the Ugandan setting, and this study may represent one of the first investigations to assess ESR as a potential discriminatory biomarker for metabolic syndrome in this population. Nevertheless, several previous studies have demonstrated significant associations between ESR and metabolic syndrome. Panagiotakos et al. (2010) reported that each unit increase in ESR was associated with a 4% increased likelihood of metabolic syndrome[27].Similarly, Holgersen et al. (2024) found that ESR, as a marker of systemic inflammation, was significantly associated with metabolic syndrome severity among individuals with hidradenitis suppurativa[28].In an observational study involving military pilots, Weber (2018) also demonstrated that participants with metabolic syndrome had significantly higher ESR values compared to healthy individuals[29].

However, when ESR together with other indices were evaluated in overweight adolescents, Podeanu et al (2024) found that ESR did not change much with the number of metabolic syndrome components which may reflect the effect of disease duration of metabolic syndrome risk on ESR[30].

Consistent with most study findings, the present study demonstrated that median ESR values were significantly higher among participants with metabolic syndrome than among those without metabolic syndrome across both sexes and in participants aged below and above 50 years. Importantly, the median ESR values among both male and female participants with metabolic syndrome exceeded the clinically accepted reference ranges for their respective age categories based on the hospital laboratory clinical reference ranges. These findings further support the hypothesis that metabolic syndrome is associated with chronic low-grade systemic inflammation and suggest that ESR may serve as a practical and inexpensive inflammatory marker for identifying individuals at increased cardiometabolic risk.

Elevated erythrocyte sedimentation rate (ESR) values observed among individuals with metabolic syndrome may reflect enhanced aggregation and accelerated sedimentation of red blood cells within the vertically positioned Westergren tube. Erythrocytes sediment more rapidly in the presence of acute-phase reactants and inflammatory proteins such as fibrinogen, serum amyloid A, and abnormal immunoglobulins, which promote rouleaux formation and increase erythrocyte density[31]

Metabolic syndrome is characterized by chronic low-grade systemic inflammation driven by lipotoxicity, insulin resistance, hyperglycemia, and vascular stress secondary to hypertension. Increased circulating free fatty acids and metabolic dysregulation stimulate the persistent release of pro-inflammatory cytokines, including interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and monocyte chemoattractant protein-1 (MCP-1). These inflammatory mediators enhance hepatic production of acute-phase proteins and contribute to endothelial dysfunction and chronic inflammatory activation, which may collectively explain the elevated ESR values observed among individuals with metabolic syndrome in this study.

In the present study, the erythrocyte sedimentation rate-to-monocyte count ratio (EMR) demonstrated moderate to good discriminatory ability for metabolic syndrome in both male and females. The AUC for EMR in males was 0.67 at a cut off of 25 by Youden index with a sensitivity of 63.6% and specificity of 70.2% and a DOR of 4.12.While in females at a cut off of 42 by Youden index ,the AUC was 0.72 with a sensitivity of 78.3% and specificity of 65.6% and DOR of 6.88.When we used the adjusted ESR values to calculate EMR there was no significant difference in AUC and DOR in males and females for discriminating ability of EMR between MetS cases. These findings show that males with metabolic syndrome were 5 times more likely to have elevated EMR compared to controls and the females with MetS were 7 times more likely to have elevated EMR as compared to controls

The ESR-to-monocyte count ratio may provide insight into two complementary inflammatory pathways involved in metabolic syndrome. ESR primarily reflects chronic mediator-driven systemic inflammation through the action of acute-phase reactants and inflammatory proteins, whereas monocyte count reflects components of the innate cellular immune response to inflammation[32]. During chronic inflammatory states such as metabolic syndrome, circulating monocytes may undergo mobilization, redistribution, tissue recruitment, or increased consumption within sites of adipose tissue inflammation and vascular injury, leading to variations in peripheral monocyte counts[33]. In the current study, the mean monocyte count was lower among individuals with metabolic syndrome compared to controls.

The relatively lower monocyte counts observed among participants with metabolic syndrome may be explained by chronic immune activation, accelerated recruitment of monocytes into inflamed adipose tissue, and

sequestration within developing atherosclerotic vascular lesions[34]. Consequently, the elevated discriminatory performance of EMR observed in this study may partly reflect a denominator effect resulting from relative monocytopenia in the setting of chronic low-grade inflammation. Even modest reductions in monocyte count may substantially increase the EMR value when accompanied by elevated ESR levels, thereby amplifying its discriminatory signal for metabolic syndrome.

The findings of this study therefore suggest that EMR may represent a novel composite inflammatory index capable of identifying individuals with metabolic syndrome who exhibit a particularly high inflammatory burden characterized by elevated ESR and relative monocytopenia. Nevertheless, these findings should be interpreted cautiously, as several factors unrelated to metabolic syndrome may influence monocyte counts and were not fully controlled for in the present study. Further validation through large-scale prospective cohort studies is therefore necessary to establish the clinical utility, reproducibility, and prognostic significance of EMR in metabolic syndrome and related cardiometabolic disorders.

The findings demonstrated that ESR, EMR, and ESR/WBC ratio were significantly associated with metabolic syndrome and remained independent predictors after multivariate adjustment. These findings support the growing body of evidence implicating chronic low-grade inflammation in the pathogenesis of metabolic syndrome.

Participants with elevated ESR had markedly increased odds of metabolic syndrome in both univariate and multivariate analyses, with the association remaining particularly strong among females. The stronger association observed among females with elevated ESR may partly reflect sex-related differences in inflammatory response, adiposity distribution, hormonal influences, and baseline ESR physiology. Females generally exhibit slightly higher ESR values than males due to differences in hematocrit and plasma protein composition. Additionally, obesity-related inflammatory activity may be more pronounced in females because of higher subcutaneous adipose tissue burden and adipokine dysregulation.

The current findings are consistent with studies that have demonstrated a relationship between inflammatory hematologic markers and metabolic syndrome. In a study to investigate the effect of different factors on ESR in adults in Northwestern Spain, it was found that ESR median values were higher in metabolic syndrome and even after adjustment for confounders metabolic syndrome and obesity were independently associated with elevated ESR[17].A systematic review and meta-analysis by Elena Raya-Cano and colleagues reported significantly higher leukocyte-related inflammatory markers among individuals with metabolic syndrome, supporting the concept that chronic inflammation contributes substantially to metabolic syndrome pathophysiology[17]. Similarly, a 2023 study reported that inflammatory biomarkers were closely linked with visceral adiposity and metabolic syndrome predictors in older adults[6].

The ESR-to-monocyte ratio (EMR) was also independently associated with metabolic syndrome. This is an important finding because composite inflammatory indices may better capture the complex inflammatory milieu underlying metabolic syndrome than isolated hematologic parameters alone. Monocytes play a central role in atherosclerosis and insulin resistance through migration into adipose tissue and vascular endothelium where they differentiate into macrophages and perpetuate inflammatory signaling. The combination of ESR and monocyte count may therefore provide an integrated measure of chronic systemic inflammation and innate

immune activation. The significant association observed in this study suggests that EMR could serve as a novel and inexpensive biomarker for identifying individuals at increased cardiometabolic risk.

Similarly, the ESR/WBC ratio independently predicted metabolic syndrome. Combining WBC with ESR may have enhanced the discriminatory capacity of inflammatory burden assessment. This finding highlights the potential utility of derived hematologic indices in improving inflammatory risk stratification beyond conventional hematologic parameters. Derived ratios may reduce variability inherent in single biomarkers and better reflect underlying pathophysiological processes. In contrast, no independent association was observed between total white blood cell count and metabolic syndrome after multivariable analysis. This differs from findings reported in a 2023 Chinese cross-sectional study which demonstrated a positive independent association between WBC count and metabolic syndrome[35]. The discrepancy may be explained by differences in study population characteristics, ethnicity, lifestyle factors, sample size, and study design. The Chinese study involved a substantially larger population and may therefore have had greater statistical power to detect weaker associations. Additionally, inflammatory phenotypes of metabolic syndrome may differ across populations due to genetic and environmental differences.

The lymphocyte-based indices, PLR and MLR demonstrated inverse associations with metabolic syndrome at univariate analysis, although these associations lost significance following adjustment in the multivariate model. The loss of statistical significance after adjustment suggests that these markers may be influenced by confounding variables or interactions with stronger inflammatory markers such as ESR. Previous studies evaluating PLR and MLR in metabolic syndrome have reported inconsistent findings, with some studies demonstrating nonsignificant relationships while some studies demonstrating positive associations[36].These discrepancies may arise from differences in study design, ethnic background, sample size, disease severity, and cutoff values used for defining inflammatory indices.

The absence of significant associations between conventional hematologic parameters such as WBC count, platelet count, neutrophil count, lymphocyte count, and MPV with metabolic syndrome may indicate that isolated hematologic parameters are less sensitive in detecting the chronic low-grade inflammatory state associated with metabolic syndrome. Chronic inflammation in metabolic syndrome is often subtle and may not produce overt abnormalities in routine hematologic counts, particularly in relatively stable outpatient populations.

Overall, the findings of this study suggest that ESR and derived inflammatory indices, particularly EMR and ESR/WBC ratio, may represent accessible, inexpensive, and clinically useful biomarkers for identifying individuals with metabolic syndrome in resource-limited settings. However, this observed usefulness may not be generalizable without further validation at the population level.

Limitations of the study

The case-control design may also have overestimated the diagnostic performance of ESR compared to the general population, and because participants were recruited from a hospital setting, the findings may not fully represent the broader community population in Jinja.

Additionally, the nonspecific nature of ESR may have resulted in small numbers of false-positive or false-negative findings, potentially affecting the precision of the diagnostic odds ratios. Although participants with known inflammatory conditions were carefully excluded and major confounding factors such as smoking, alcohol use,

anemia, autoimmune disease, heart failure, and kidney disease were controlled for, residual confounding from unmeasured factors may still have influenced ESR values and the identified cutoff points. Nevertheless, the study sample size was adequate to detect meaningful associations and provided important preliminary evidence regarding the discriminatory role of ESR and EMR in metabolic syndrome

Conclusion

Erythrocyte sedimentation rate can moderately discriminate individuals with metabolic syndrome from those without metabolic syndrome in eastern Uganda. This discriminatory ability is higher among females than for males.

Derived ESR indices are strongly and independently associated with metabolic syndrome in this population. Although ESR is a non-specific inflammatory biomarker, it can have a diagnostic utility in metabolic syndrome as a marker of low-grade chronic inflammation. It is therefore recommended that large population cohort studies be performed for further validation of the clinical utility, the appropriate population cutoff values and diagnostic performance of ESR and its derived indices in metabolic syndrome.

Abbreviations

MetS; Metabolic syndrome

NCD; Non communicable disease

HDL; High Density Lipoprotein

ESR; Erythrocyte sedimentation rate

IDF; International Diabetes Federation

EMR; ESR to monocyte count ratio

AHA; American Heart association

NHLBI; National heart lung and blood institute

TNF; Tumor Necrotic Factor

CRP; c reactive protein

IL-6; Interleukin 6

WHO; World Health Organization

HIV; Human immunodeficiency virus

JRRH; Jinja regional referral hospital

MLR; Monocyte to lymphocyte ratio

PLR; Platelet to lymphocyte ratio

NLR; Neutrophil to lymphocyte ratio

Declarations

We hereby declare that this research report is the result of our original research work and input. No part of this report has ever been published in any journal.

Ethics approval and consent to participate

Ethical approval for this study was sought and obtained from the Research Ethics Committee at Kampala international University. Written informed consent was also obtained from the individual participants and all methods were performed in accordance with the ethical principles of the declaration of Helsinki.

Availability of data and materials

The datasets analyzed during the current study are available on request from the corresponding author at the email; rweyeshera@gmail.com

Competing interests

The authors declare that they have no competing interests.

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Authors Contributions

IPA participated in the study conception and design, data acquisition, analysis and interpretation, manuscript drafting and revising the version for publication.

AB participated in study design, supervision of data curation and report review and editing.

AK participated in study design, supervision of data curation and report review and editing.

HKK participated in the study conception data analysis and interpretation and manuscript drafting.

BBB participated in the study conception design and data acquisition.

AHA participated in report writing, editing and data acquisition.

BMV participated in the study conception, methodology, supervision and revising manuscript for publication.

MN participated in formal data analysis and interpretation.

VO participated in study design, supervision of data curation and report review and editing manuscript for publication.

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Table 3

Table 3 is not available with this version.

Figures

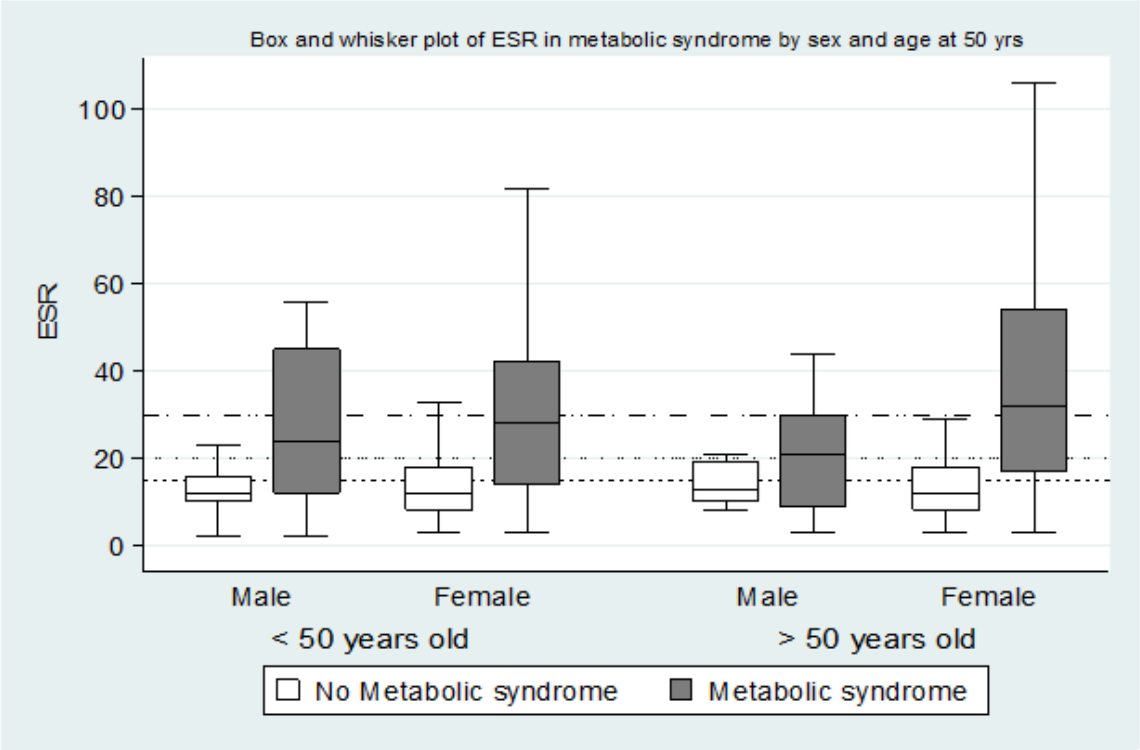


Figure 1

Box and whisker plot showing the dispersion of ESR between metabolic syndrome participants and normal participants showing reference cut-offs lines for ESR by sex and age dichotomized at 50 years.

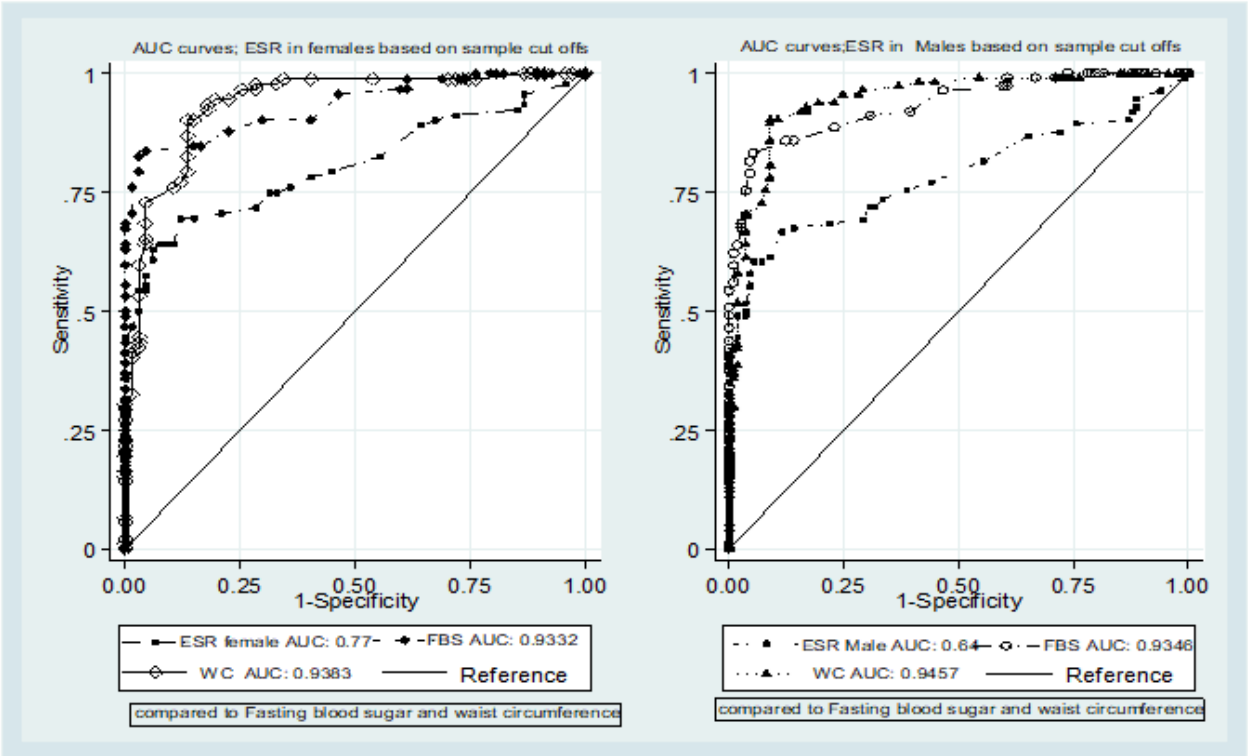


Figure 2

The graphic presentation of discriminating ability of ESR between metabolic syndrome and non-metabolic syndrome based on sample derived ROC analysis cut off values presented by sex category.