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ORIGINAL ARTICLE

Outcome of Adult Acute Myeloid Leukemia Expressing Lymphoid Markers: A Single-Center Experience

Shimaa Abdel-Moneem[1*], Mohammad Al-Akkad[1], Ayman Fathy[1], Alaa Abdel-Moaty[2],Ahmed Embaby[1]
[1]Clinical Hematology Unit, Internal Medicine Department, Faculty of Medicine, Zagazig University, Zagazig,Egypt.
[2] Clinical Pathology Department, Faculty of Medicine, Zagazig University, Zagazig, Egypt.

***Corresponding autho**r:
Shimaa Abdel-Moneem

Email:
Shimaaabdelmoneem123@gmail.com

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ABSTRACT

Background: The aberrant phenotype is a phenomenon in which lymphoid associated and other myeloid lineage markers are expressed in myeloblasts or myeloid- associated markers expressed in lymphoblasts. The development of an aberrant phenotype with varying frequency in acute leukemia has been confirmed, although its prognostic significance remains controversial. So, the aim of this prospective cohort study was to evaluate the outcome of newly diagnosed patients with acute myeloid leukemia expressing lymphoid markers (Ly+ AML).**Patients and Methods:** our prospective cohort study designed to evaluate de novo adult patients with primary Acute Myeloid Leukemia (AML) with the exclusion of acute promyelocytic leukemia admitted to clinical Hematology Unit in Department of Internal Medicine, from October 2016 to the July 2019.**Results:** Patients were classified according to their lymphoid markers expression into two groups Ly+ AML and Ly- AML including; 23 (29.5%) and 55 (70.5%), respectively. Following a median 8.133 months (range, 0.3-26 months) as a period of follow up**,** higher complete response rate was noticed in Ly – AML group in contrast with Ly + AML (P= 0.04). Moreover, Ly+ AML patients group was independently associated with complete response to therapy with OR 0.34 [95%CI; 0.12-0.98], P=0.047 by using the Logistic Regression Multivariate Model. In terms of survival, we did not prove statistically significant difference in 26 months disease-free survival and overall survival as well.**Conclusion:** lymphoid markers expression in AML patients was independently associated with Response to therapy. It has not, however, been identified as an independent survival predictor.
Keywords: AML, Lymphoid markers, BM and Karyotyping.

INTRODUCTION

Acute myeloid leukemia is a malignant neoplasm of immature, bone marrow (BM)-derived myeloid cells exhibiting variable differentiation, AML is a biologically heterogeneous disease group that most often involves the BM and peripheral blood (PB) but may also manifest in extramedullary tissue" [1].Malignant cells display characteristic patterns of surface antigenic expression so immunophenotyping is used to study the antigenic expression of CD markers on leukocytes [2]. These aberrancies provide a valuable manner for properly classifying those myeloid malignancies in compliance with "2008 World Health Organization classification"(WHO), and expression of aberrant antigens is also essential for prognosis[3].Immunophenotyping improves the precision and reproducibility of acute leukemia classification and is also considered useful for detecting AML with lymphoid marker expression[4]. With varying frequency, the aberrant phenotype has been reported in acute leukemias, although its prognostic value remains controversial, aberrant phenotypes have been documented up to 88% in AML[5].As well as lifestyle factors, exposure to environmental agents; including radiation and some chemicals may cause damage to DNA, and associated genetic changes have been associated with increased risk of acute myeloid leukemia[6].One of the most powerful independent prognostic indicators in AML is diagnostic karyotyping, which is used to classify biologically distinct

subsets of disease and has been widely adopted to provide the basis for risk- adapted treatment[7].

AIM OF THE WORK

The aim of the study was to evaluate the outcome of newly diagnosed patients with acute myeloid leukemia expressing lymphoid markers correlated with cytogenetic pattern and clinicobiological features.

METHODS

This prospective cohort study was planned to evaluate 78 de novo adult patients with primary AML excluding acute promyelocytic leukemia (as patients with APL treated with all-*trans* retinoic acid (ATRA) in addition to anthracycline-based protocols). All were 18-years or older with good performance status [8], and were treated at the Clinical Hematology Unit, from October 2016 to July 2019. Written informed consent was obtained from all participants' parents and the study was approved by the research ethical committee of Faculty of Medicine, Zagazig University. The work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.Clinical and laboratory data were obtained such as age, gender, hemogram; total leukocytic count, haemoglobin, platelet count, percentage of circulating and bone marrow blast cells, Cytogenetic analysis, and Immunophenotyping; the patients were diagnosed by "multiparametric flowcytometry using an acute panel of monoclonal antibodies". Immunological characterization was performed on gated blast cells from bone marrow samples by flowcytometry using an exteusive panel of Phycoerythrin (PE) /fluorescein isothiocyanate (FITC), Allophycocyanin (APC) and Peridinin-chlorophyll protein complex (perCP) conjugated monoclonal antibodies, for diagnosis and subtyping of AML using the following markers: CD13, CD33, CD34, HLA-DR, MPO, CD14 and CD64, and cases of myeloid leukemia were considered to be lymphoid-associated antigens (Ly + AML) if they met FAB criteria for diagnosis of AML expressed myeloid surface antigens and when more than 20% of the blastic cells expressed one or more of (CD2, CD3, CD5, CD7, CD19, CD20, CD22) using Becton Dickinson (BD) FACS DIVA and classified into subtypes of AML in accordance with the morphological, immunophenotypic, and cytogenetic features of leukemic blast cells according to French-American-British (FAB) [9] and the WHO 2016 criteria [10].

Treatment plan: Patients received induction chemotherapy which is composed of continuous intravenous infusion (ivi) of cytarabine 100 mg/m2/day for 7 days in a row with ivi of Doxorubicine 25mg/m2/day D1 to D3. Patients who achieved complete remission (CR); received consolidation therapy (post-induction therapy) which included three to four high-dose cytosine arabinoside courses (1.5-2g/m2 per twelve hours on 1, 3, 5 total days, 12 g/m2) [11].

Therapy outcome criteria: Response to induction therapy was assessed. Complete remission was identified as less than 5 percent blasts in BM aspirates with evidence of cell lines maturation and PB counts restoration and no evidence of extramedullary leukemia according to standard criteria [12]. While, early death (ED) was defined as: death within 30 days of chemotherapy initiation [13]. Haematological relapse was reported when more than 5% of blasts in BM aspirate detected or extramedullary leukemia appearance. Regarding Survival; Disease-free survival (DFS) was calculated; starting from the time of CR to the relapse time or death; and overall survival (OS) was calculated from the time of initial diagnosis to the time of death/ last follow-up.

Follow up: After consolidation is completed, the patients were monitored with clinical examination and laboratory investigations including; complete blood count (CBC), with blood smear every 1–3 months for 2 y, then every 3–6 months on ward till the study end. BM aspiration and biopsy were recommended if pathological cytopenias developed to rule-out relapse, as recommended by the National Comprehensive Cancer Network (NCCN) guideline [14]. We censored patients that underwent allogenic bone marrow transplantation (BMT) at the point in time of transplantation.

Statistical analysis: Chi-square testing analyzed the differences in response rates between two groups of patients. Life tables for survival and event-free survival were established using the *Kaplan* and *Meier* method, with variations compared by the log-rank test. Failure to enter remission or early death was considered an incident at zero time. Patients who underwent bone marrow transplantation at the point in time of the procedure were censored.For univariate analysis; the Cox proportional hazards model has been used. Variables which in the univariate analysis were statistically significant included in multivariate analysis. All statistical analyses have been conducted using the Statistical Package for Social Sciences (SPSS 24 Inc. Chicago, IL, USA).P value that was ≤ 0.05, considered statistically significant.

RESULTS

Seventy eight de novo adult patients who had non-M3 AML were classified according to their lymphoid markers expression into two groups Ly+ AML and Ly- AML including; 23 (29.5%), and 55 (70.5%), respectively. As shown in table 1, In Ly+ AML and Ly- AML patients groups, median age of Ly+ AML and Ly- AML patients groups was 41 years versus 43 years. According to the "FAB classification" there was higher frequency of M4 and M2 in both groups, in 80 % of Ly-AML patients and in 81% of Ly +AML patients, cytogenetic analysis was carried out, normal karyotypes were observed in both patient groups, in AML patients expressing lymphoid markers, other clonal abnormalities characteristically associated with AML including t 8; 21), Del 1q, Del Y, Tri 8, Ph + were almost detected. Ly+ AML group significantly associated with lower haemoglobin (Hb) level, P=0.04.High frequency of high total leukocytic count (TLC) was noticed in both patients groups, and no statistically relevant correlation was noticed as regard WBCs count.Moreover, there was no statistically relevant correlation between Ly+ AML and Ly – AML in relation to prognostic factors (age, difference in sex, WBCs count, platelet count, percentage of blast cells, and cellularity of BM, P>0.05.

As shown in Table 2, Forty two(53.8%) patients attained CR, 36 (46.2%) patients did not attain CR after two cycles of induction therapy, and in the end of follow-up 48/78 (61.5%) of patients died. On the follow up; 18/42 of cases that achieved CR relapsed, and 12/42 of cases that achieved CR underwent BMT. While higher CR rates were noticed in Ly – AML group compared to Ly + AML (P= 0.04), no statistically relevant correlation was reported regarding Relapse, Death or HCT. Binary logistic regression was conducted to assess the effects of various variables on the patients' response, in multivariate analysis; only lymphoid expression markers were independently correlated with response to therapy with OR 0.34 [95%CI; 0.12-0.98], P=0.047(Table 3).

Patients were monitored for a median period of 8.133 months (range, 0.3-26 months). The 26 months overall survival rate was 28.60% with, the mean was 12.3±1.2 months (95% CI; 9.9-14.7months); and the median was 10.9±2.3 (95% CI; 6.5-15.3months) while the 26 months disease-free survival rate was 39.3% with 16.5±1.5 months as a mean (95% CI; 13.7-19.4), and 20.0±5.4 months as the median (95% CI; 9.4-30.6) (Table 4). No statistical difference was seen in the Kaplan- Meier analysis either in 26 months OS or 26 months DFS between both groups (p= 0.092, and 0.784, respectively) (Table 4 and Fig 1a, b).

Table (1): Baseline Ly+ AML and Ly- AML clinical patient characteristics:

Parameters		**LYM Markers**		**Total N=78**	**P**
		Negative N=55	Positive N=23		
Age		43(18-63)	41(22-63)	43(18-63)	**0.931**
PS		1±1	1	1±1	**0.7**
Sex	Female	24(43.6%)	13(56.5%)	37(47.4%)	**0.494**
	Male	31(56.4%)	10(43.5%)	41(52.6%)	
TLC×10/L		14.8(1.3-202)	36.1 (3.5-76)	18.3 (1.3-202)	**0.148**
HB g/dl		8.3 (4.9-11)	7.1 (5.6-8.2)	8(4.9-11)	**0.04**
PLT×10/L		30(5-140)	39(12-444)	32(5-444)	
PB blast%		55(10-97)	45(22-80)	52.5(0-98)	**0.566**
BM blast %		74(23-97)	72(28-85)	73(23-97)	**0.776**
FAB (subtype)	M0	2(3.6%)	0(0.0%)	2.6%)	**0.655**
	M1	4(7.3%)	3(13.1%)	7(9%)	
	M2	17(30.9%)	6(26.1%)	23(29.5%)	
	M4	21(38.2%)	11(47.8%)	32(40.8%)	
	M5	11(20.0%)	3(13.1%)	14(18.2%)	
Cytogenetic Risk	Fail	11(20.0%)	5(21.7%)	16(20.5%)	**0.509**
	Fav	2(3.6%)	1(4.3%)	3(3.8%)	
	Intr	36(65.5%)	16(69.6%)	52(66.7%)	
	Ufav	6(10.9%)	1(4.3%)	7(9%)	

PS; performance status: BM: bone marrow; PB: peripheral blood; WBC, white blood cell count; Hb; hemoglobin; PLT: platelets; FAB: French-American-British; BM: bone marrow.[Continuous data are presented as median (range) or n %].

Table (2): Patients clinical outcome No [%] in both groups

		LYM Markers				Total 78		P
		Negative N=55		Positive N=23				
		N	%	N	%	N	%	
Response	CR	34	61.8%	8	34.8%	42	53.8%	0.045
	NR	21	38.2%	15	65.2%	36	46.2%	
Relapse*	N	20	58.8%	4	50.0%	24	57.1%	0.76
	Y	14	41.2%	4	50.0%	18	42.9%	
Death	N	24	43.6%	6	26.1%	30	38.5%	0.230
	Y	31	56.4%	17	73.9%	48	61.5%	
Underwent HCT	N	23	67.6%	7	87.5%	30	71.4%	0.38
	Y	11	32.4%	1	12.5%	12	28.6%	

*Relapse calculated from responders, CR: complete remission, NR: no remission; HCT: hematopoietic stem cell transplantation; Ly+: Lymphoid antigen positive; Ly-AML: Lymphoid antigen negative.

Table 3: Univariate and multivariable logistic regression analyses for Response to therap

Covariates	Univariate			Multivariate		
	OR	95% C.I. for OR	Sig.	OR	95% C.I. for OR	Sig.
Age	1.02	0.99-1.05	0.146			
Sex(F vs M)	0.88	0.36-2.15	0.779			
Risk(Unfav vs Others)	4.73	0.53-42.52	0.166			
FAB M5	Ref					
FAB(M0)	1.33	0.07-25.91	0.849	1.69	0.09-33.46	0.730
FAB(M1)	8.00	0.75-85.31	0.085	7.03	0.63-78.02	0.112
FAB(M2)	0.58	0.15-2.32	0.445	0.54	0.13-2.22	0.390
FAB(M4)	1.33	0.38-4.72	0.656	1.17	0.32-4.32	0.812
Lymphoid Markers(N vs P)	0.33	0.12-0.91	0.032	0.34	0.12-0.98	0.046
PB	1.00	0.98-1.02	0.966			
BM	1.02	0.99-1.04	0.199			
TLC Level N	Ref					
TLC Level(H vs N)	1.63	0.62-4.28	0.318			
TLC Level(L vs N)	0.97	0.23-4.15	0.972			
PLT	1.01	0.99-1.02	0.274			
Hb	0.78	0.53-1.14	0.196			

F: female; M: male; vs: versus; Unfav: unfavorable; FAB: French-American-British; BM: bone marrow; PB: peripheral blood; WBC: white blood cell count; TLC: total leukocytic count; H: high; N: normal; L: low.

Table 4: The overall and Disease-free survival rates in both groups

Groups	Survival rate %	P value	Survival time, months			
			Mean		Median	
			Estimate ± SE	95% CI	Estimate ± SE	95% CI
The 26- months OS%						
Ly- AML	31.00%		13.7±1.5	10.8-16.6	13.3±2.7	8.0-18.6
Ly+ AML	19.60%	0.092	9.0±1.9	5.2-12.8	4.3±1.3	1.7-6.9
Overall	28.60%		12.3±1.2	9.9-14.7	10.9±2.3	6.5-15.3
The 26-months DFS%						
Ly- AML	47.10%	0.784	17.0±1.7	13.6-20.3	20±0	8.0-26.1
Ly+ AML	29.20%		16.5±2.7	11.2-21.9	16.0±5.2	5.9-26.1
Overall	39.30%		16.6±1.5	13.7-19.4	20.0±5.4	9.4-30.6

The 95%CI: 95% confidence interval; SD: standard deviation: Ly+: Lymphoid antigen positive; Ly-AML: Lymphoid antigen negative: OS; overall survival; DFS: disease free survival.

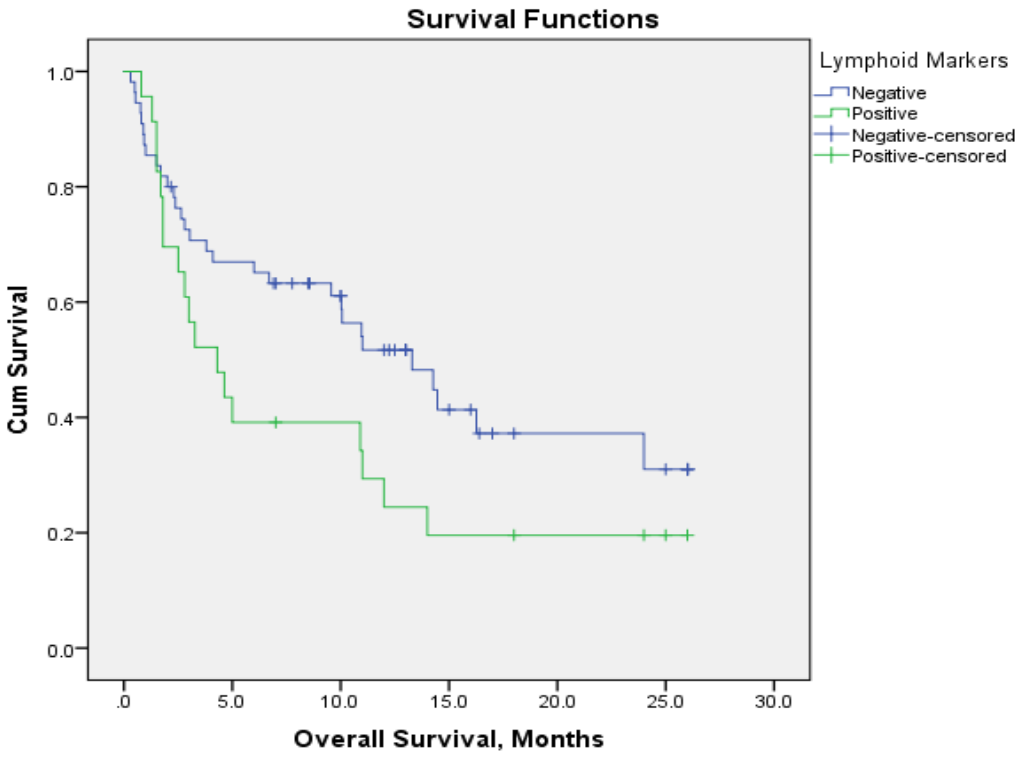

Figure 1: Kaplan– Meier survival curves illustrating overall Survival in months in relation to Lymphoid Markers expression.

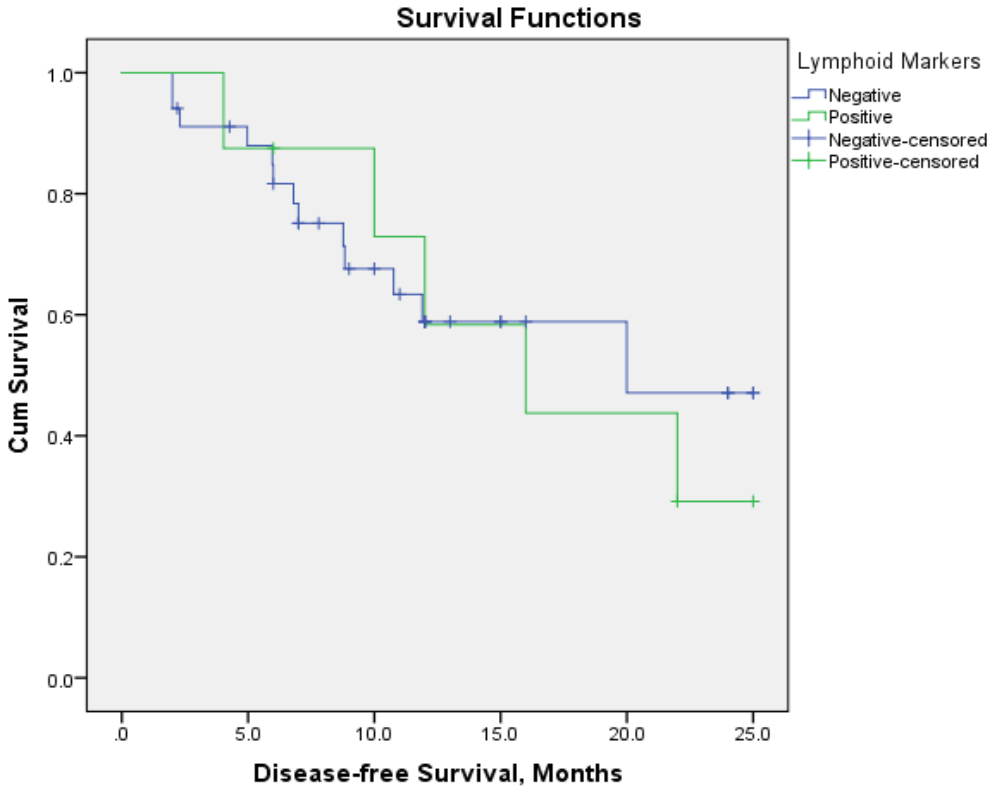

Figure 2: Kaplan– Meier survival curves illustrating disease-free survival rate in relation to Lymphoid Markers expression.

DISCUSSION

Immunophenotyping increases both the precision and reproducibility of classification for acute leukemia and is taken into consideration as a useful method for identifying "AML" with lymphoid marker expression [4]. Some studies showed that Ly +AML patients having poor prognosis, but others reported that those patients having favorable prognosis, while others suggested that lymphoid markers expression in those AML patients has no prognostic benefit [15]. So, in our study, we shed the light on the value of lymphoid markers expression correlated with patients' outcome. In our study, Ly +AML patients group was significantly associated with higher CR rates, but no correlation was reported as a statistically significant regarding FAB subtype or cytogenetic analysis.

From published studies that investigated the prognostic relevance of lymphoid markers expression inpatients; Ossenkoppele and coworkers study, who found that patients with positive lymphoid markers expression (CD7& CD56) had poor response to therapy, and associated with reduced disease-free survival (DFS) and OS [16]. While our study had similar results, they were different in the selected population as cases of M3 were not excluded from their study and also reported that patients with (CD2) as a lymphoid antigen expression associated with inversion (16), and translocation (16&16), patients with (CD19) associated with translocation (8&21), but patients with (CD7) associated with mutated CEBPA.

Also, another study reported that no statistically significant correlation regarding response in Ly+ AML patients compared to Ly- AML patients. This disagreement with our finding because of the inclusion of cases of M3, and in Ly + AML patients, clonal abnormalities including t(15;17), 1 lq, 5q abnormalities, +8, inv(3) and inv/del(16), were nearly exclusively identified. Despite these findings, agreed with our study that both groups of patients had the same survival and event-free survival [17]. Also, study that was done by Ball and coworkers reported that patients with lymphoid antigen expression had higher complete remission rates, and associated with superior OS [18]. This inconsistency with our findings may be attributable to many variables; the FAB morphology of patients with positive lymphoid antigen expression (CD2&CD19) showed that M4 was eight times as frequent, and M3 twice as frequent relative to the CD2and CD19 negative cases, and a higher frequency of these karyotypic anomalies including;(8; 21) (q22; q22), inversion

16(p13q22), t (15; 17)(q22; q12) was significantly reported.

The intrinsic relationship between prognostic factors (age, gender, BM blast hemoglobin level, platelet count, WBCs) and clinical outcome in AML patients correlated with lymphoid antigen expression has been adopted in various laboratory- based researches that reported no correlation as a statistically significant between adverse prognostic factors and aberrant phenotypes [17,19]. We determined this correlation in our study that reported significant variation in hemoglobin level between patients' groups, but no other statistically significant association was reported between other prognostic factors and aberrant phenotypes. In addition, the prognostic value of the initial hemoglobin level correlated with patients' outcome was not identified in the univariate or multivariate analysis. The study that was done by Al-Anizi and coworkers, who evaluated the frequency and existence of aberrant lymphoid phenotypes associated with various "FAB" subtypes, reported that these lymphoid phenotypes might be correlated with different subtypes of leukemia, and T- cell markers are more prevalent than B-cell markers [4]. One of the limitations in our study is that our study aimed to estimate any significant association among aberrant phenotype changing, cytogenetic pattern and clinical outcome, and we did not estimate aberrant expression of certain lymphoid marker correlated with various leukemia subtypes. So broader a prospective study in the future is highly recommended to find if there is correlation among certain lymphoid marker expression, different leukemia FAB subtypes, and specific cytogenetic anomaly with evaluation of their possible potential effect on clinical outcome.

CONCLUSION

This is prospective study that evaluated the outcome of AML patients having lymphoid antigens expression in correlation with cytogenetic patterns and clinicobiological features revealed that lymphoid markers expression appeared as an independent predictor factor for response. However, the lymphoid markers expression was not reported as a prognostic factor for survival. Lastly, before generalization of these results, broader prospective multicenter studies taking into consideration racial and genetic information and other adverse risk factors are needed.

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Availability of data and materials: All analyzed and generated data through this study are incorporated in this published article.

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Competing interests: The authors announce that having no competing interest.

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