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ORIGINAL ARTICLE

Prognostic Value of B Cell Maturation Antigen among Multiple Myeloma Patients

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Abstract

Background: The course of multiple myeloma (MM) varies among cases; some may not show any symptoms for years, while others may experience rapid disease progression despite therapies. MM is a B-cell malignancy of terminally developed plasma cells derived from bone marrow (BM). The quantity of monoclonal immunoglobulins (Ig) produced by these clonal cells is high. The present work aims to investigate B-cell maturation antigen (BCMA) as an early predictor of MM cases outcome. In addition, to determine the relationship of BCMA levels to progression free survival among MM cases. **Subjects and methods:** This prospective cohort investigation included 50 cases diagnosed with multiple myeloma and underwent novel chemotherapy as bortezomib and/or immunomodulators. All cases were subjected to full history, clinical and laboratory assessment and fundus examination were done. Radiological studies include skeletal surveys. Special investigations included B-cell Maturation antigen using ELISA. **Results:** BCMA was higher among patients who showed complete response compared to those who showed partial response with slight remarkable variance (p=0.046). There was a notable difference between response regarding anemia and hypercalcemia (p=0.01). Meanwhile, BCMA yielded significance (p=0.045) in predicting complete response level at cutoff level >2245 in predicting complete response with sensitivity of 75% and specificity of 83.3%. **Conclusion:** BCMA yielded significance in predicting complete response level at cutoff level >2245 in predicting complete response with sensitivity of 75% and specificity of 83.3%. It could aid in improved risk classification and more customized clinical care, improving therapeutic outcomes and elevating the life expectancy of MM patients.

Keywords: B Cell Maturation Antigen, Prognostic Value, Multiple Myeloma.

INTRODUCTION

Multiple myeloma (MM) is a completely differentiated B-cell cancer that causes an increase of monoclonal plasma cells in the bone marrow (BM). There remains no recognized effective therapy readily available and cases' median survival is 5 years [1]. Protease inhibitors (PI), immunomodulatory medicines, and antibody-based treatments have greatly improved the management of MM. The discovery and production of these novel medications have led to better outcomes, especially overall survival (OS) [2].

Despite improved therapy choices and higher OS rates, MM is still a highly aggressive and incurable condition. With a growing range of therapeutic alternatives, a more rapid and accurate technique of identifying progressive disease (PD) is essential to ensure that cases remain on treatment, which is still efficient. Recently accessible tests to track cases with MM include determining monoclonal paraprotein (M-protein) and serum-free light chain (SFLC) concentrations by a combination of protein electrophoresis, SFLC assay, and immunofixation [3]. BCMA is a member of the tumor necrosis

factor receptor family, is expressed on plasma cell surfaces and increased in MM cases' serum. Currently, sBCMA has been demonstrated to anticipate cases outcomes and quickly detect alterations in cases' clinical condition [4].

Furthermore, sBCMA values relate to the number of plasma cells in BM biopsies from MM cases, as well as their clinical condition. Furthermore, people with active MM had greater sBCMA concentrations than those with smoldering MM, whereas persons with MGUS had the lowest amounts [5]. In retrospective investigations including cases that received a wide range of treatments, sBCMA reliably measures variations in condition state among cases with MM and recognizes alterations in condition status more fast than either SFLC or M-protein. The biomarker's serum half-life of 24-36 hours enables faster identification of clinical alterations in MM cases compared to conventional assays. This should enable clinicians to assess the efficiency of medications and make clinical choices to stop unsuccessful medications and switch to new treatments more fast [6].

Several studies were performed to assess if B-cell maturation antigen could be an early predictor of MM cases outcome, so this study designed to investigate BCMA as an early predictor of outcome of cases with MM. In addition, to determine the relationship of sBCMA concentrations to progression free survival among MM cases among Zagazig University Hospitals.

METHODS

This prospective cohort study included 50 MM patients recruited from Hematology Unit of Internal Medicine Department Zagazig University hospitals, from February 2023 to January 2024. The goal and scope of the investigation, as well as the risk-benefit evaluation, were described to the individuals before their admission to this investigation. Verbal and written informed consent were collected from all individuals after an explanation of the procedure and medical research. The research was conducted under the World Medical Association's Code of Ethics (Helsinki Declaration) for human research. This study was carried out after the approval of the Institutional Review Board (IRB#10389/12-2-2023).

Inclusion criteria:

Patients diagnosed with multiple myeloma aging> 18 years and underwent novel chemotherapy as bortezomib and/or immunomodulators were included in this investigation.

Exclusion criteria:

Patients with precursor lesion (MGUS), patients with smoldering MM (asymptomatic myeloma), patients had other causes of anemia, renal impairment, and bone lesions, patients who were treated with other lines of treatment other than proteasome inhibitors or immunomodulators, pregnant women, and patient refuses to give consent and lack of cooperation.

All cases were subjected to complete history taking, clinical and laboratory examination, and radiological studies including skeletal surveys: which consisted of a lateral radiograph of the skull, anteroposterior (AP) and lateral views of the spine, and AP views of the humeri, ribs, pelvis, and femora. Inclusion of at least these bones is important for both diagnosis and staging".

Laboratory examination:

Each individual had 10 ml of peripheral fasting venous blood drawn under strict aseptic circumstances. The laboratory investigations include hemoglobin, kidney function tests, serum albumin, LDH, β2-microglobin, Electrolytes, BM aspiration and biopsy, protein electrophoresis, and immune fixation.

B-cell Maturation antigen:

The assay was carried out by B-cell maturation antigen (BCMA) ELISA Kit, Catalog No. SG-16330. SinoGeneClon Biotech Co., Ltd the procedure was done according to the manufacturer's instruction. Normal lab reference of BCMA is range 18.78 -180.39 ng/mL.

Treatment:

All patients were treated with VCD (Velcade 1.3 mg/m2 day 1, 4, 8, 11; Dexamethasone 40 mg/week, and Cyclophosphamide 300 mg/m2/week). Or VRD (Velcade 1.3mg/m2 on days 1, 4, 8, and 11, Dexamethasone 40mg/week, and Lenalidomide 25mg/day). Or VDT (Velcade 1.3mg/m2 on days 1, 4, 8, and 11, Dexamethasone 40mg/week, and Thalidomide 100mg/day). Or CRD (cyclophosphamide 300mg/m2/week, dexamethasone 40mg/week, and lenalidomide 25mg/day). Or CDT (thalidomide 100mg/day, cyclophosphamide 300mg/m2/week, and dexamethasone 40mg/week).

Outcome measures:

Response to induction treatment was evaluated after one course cycle of chemotherapy (duration of induction chemotherapy = 4 months). Evaluation serum BCM antigen percentage before treatment detection. it's impact on response to treatment. Disease free survival was measured

from the time of response to treatment to the time of relapse or death and OS from the time of initial diagnosis to the time of death.

Follow Up:

Follow up with the patients after receiving treatment (4 months of induction chemotherapy) then every 2 months till the end of study.

Statistical Analysis:

All data were analyzed using SPSS 24.0 for Windows (SPSS Inc., Chicago, Illinois, USA). The Shapiro Walk test was done to see if the data reflected a normal distribution. Qualitative data was presented as frequencies and relative percentages. Qualitative variables were analyzed utilizing the chi-square test ($\chi 2$) and Fisher exact test, as indicated. Non-parametric data were displayed as median and range, while parametric data were presented as mean ± SD (standard deviation). The variation between quantitative variables in two groups was evaluated employing the independent t-test for parametric variables and the Mann-Whitney test for non-parametric variables. The one-way ANOVA test was developed for comparing two or more dependent groups with normally distributed variables. The Kruskal-Wallis test was used for variables that did not follow a regular distribution. The Spearman's correlation test was used to correlate variables. Event-free survival was calculated using the Kaplan-Meier method, and survival curves were compared employing the log-rank test. The receiver operating characteristic (ROC) curve was developed to help with the determination of threshold levels for test results and comparing various testing processes. The values for the area under the ROC curve (AUC) are as follows: 0.90-1 = excellent, 0.80-0.90 = good, 0.70-0.80 = acceptable, 0.60-0.70 = poor, and 0.50-0.6 = fail. The best cutoff point was determined to be the point of highest accuracy. All statistical comparisons were two-tailed and significant. P-values < 0.05 imply significance.

RESULTS

The mean age of the patients was 53.63 ± 10.56 years. 60% of the patients were females. Regarding comorbidities, 70% of the patients had comorbidities, the most prevalent of them was hypertension that was found in 30% of the patients while hypertension & DM were found in 20% of patients. Moreover, 10% of patients had ischemic heart disease, 6% were hypothyroidism, and 4% were asthmatic. Regarding presenting symptoms, most of the patients (46%) complained from bone ache, 24% suffered from fatigue, and 10% suffered from bone ache and generalized swelling. The laboratory data were summarized in (Table 1).

Regarding clinical presentation, 24% had abnormal cytogenetics and high-risk FISH, 70% had lytic lesions, 16% had EMD at diagnosis, 30% were anemic, 16% suffered from hypercalcemia, 20% had renal insufficiency. Concerning staging, 46% had stage III, 30% had stage I, and 24% had stage II. Regarding M protein types, 80% were IgG, and 20% were IgA. 56% of cases were treated with VRD and 44% of cases were treated with VCD. Regarding response, 80% of the patients revealed complete response, and 20% of cases revealed partial response (Table 2).

There was a remarkable direct association between BCMA and platelets only ($p<0.05$) (Table 3).

Table (4) represented that there was no significant relation between BCMA and different parameters and clinical characteristics.

There was a substantial variance between CR and non-CR cases regarding age, sex, hemoglobin, and stage. Moreover, BCMA was higher among patients who showed complete response compared to those who showed partial response with slight remarkable variance. There was a notable difference between response regarding anemia and hypercalcemia (Table 5).

There was no significant association between B cell maturation antigen (BCMA) and age, gender, comorbidities, Hb, PLT, Serum. albumin, g/dl, creatinine, Bencejonesprotein.in urine, Elevated lactate dehydrogenase, Serumbeta.2microglobulin, mg/l, calcium, types of M.protein , Bone marrow plasma cells (Table 6). BCMA yielded significance level at cutoff level >2245 in predicting complete response with sensitivity of 75% and specificity of 83.3% (Table 7).

Mean OS time was 9.65 days (95% confidence interval 9.35 – 9.96) and PFS time was 5.66 (95% confidence interval 5.348 -5.964) among all patients. We found that patients with high levels of BCMA had slightly shorter OS and PFS time than those with lower levels of BCMA, there was no significance difference (Fig. 1).

Table (1): Demographic, clinical characteristics, and laboratory data among the studied patients.

	Patients (n=50)	
	Mean ± SD	**Range**
Age (years)	53.63 ± 10.56	27 - 72
Gender	N	%
Female	30	60
Male	20	40
Comorbidities		
Hypertension	15	30%
Hypertension & DM	10	20%
IHD	5	10%
Hypothyroidism	3	6%
Asthma	2	4%
Presenting symptoms		
Bone ache	23	46%
Fatigue	12	24%
Bone ache & generalized swelling	5	10%
Lower limb weakness	3	6%
Hemorrhage and pathological fracture	3	6%
Shortness of breath	2	4%
Muscle pain	2	4%
Laboratory data	Mean ± SD	
Hemoglobin (g/dl)	10.17 ± 1.41	
Platelets ($x10^3$/L)	246.33 ± 84.53	
Albumin (g/dl)	3.47 ± 0.226	
Creatinine (mg/dl)	1.41 ± 0.749	
Serum β2-microglobulin (mg/l)	4.25 ± 1.01	
Calcium (mg/dl)	10.15 ± 1.19	
Bence jones protein in urine	32 (64%)	
Elevated LDH	10 (20%)	
Bone marrow plasma cells	40.37 ± 20.45	
BCMA (pg/ml)	2723.5 ± 1371.44 (1570.2 - 8203.8)	

Table (2): Clinical characteristics, treatment, and response to treatment among the studied patients.

	Patients (n=50)	
	N	**%**
Abnormal cytogenetics		
Yes	12	24%
No	38	76%
High risk FISH		
Yes	12	24%
No	38	76%
Lytic lesions		
Yes	35	70%
No	15	30%
EMD at diagnosis		
Yes	8	16%
No	42	84%
Anemia (hemoglobin ≥2 g/dl to ≤10 g/dl)		

	Patients (n=50)	
	N	N
Yes	15	30%
No	35	70%
Hypercalcemia (Ca ≥11.5 mg/dl)		
Yes	8	16%
No	42	84%
Renal insufficiency (serum creatinine ≥2 mg/dl)		
Yes	10	20%
No	40	80%
Stage		
I	15	30%
II	12	24%
III	23	46%
M protein types		
IgG	40	80%
IgA	10	20%
Treatment		
VRD	38	56%
VCD	22	44%
Response to treatment		
Complete response	40	80%
Partial response	10	20%

Table (3): Correlation between BCMA and other parameters**.**

	BCMA	
	R	P
Hemoglobin	.211	.264
Platelets	**.474**	**.008**
Albumin	-.025	.894
Creatinine	-.153	.420
β2-microglobulin	-.267	.161
Calcium	-.125	.520
BM plasma cells	-.178	.347

Table (4): Relation between BCMA levels and clinical characteristics and investigated parameters among the studied patients.

	BCMA (pg/ml)	
	Mean ± SD	P-value
Gender		
Female	3011.26 ± 1595.8	.168
Male	2291.9 ± 827.1	
Comorbidities		
Present	2943.5 ± 1559.4	.287
Absent	2210.2 ± 561.2	
Stage		
I	2859.0 ± 899.1	.277
II	2858.4 ± 1264.9	
III	2568.96 ± 1706.4	
Abnormal cytogenetics		
Yes	2920.8 ± 1030.7	.364
No	2663.5 ± 1474.2	
High risk FISH		

	BCMA (pg/ml)	
	Mean ± SD	**Mean ± SD**
Yes	3136.4 ± 2337.97	.998
No	2597.8 ± 958.01	
Lytic lesions		
Yes	2582.2 ± 1492.2	.103
No	3053.2 ± 1037.7	
EMD at diagnosis		
Yes	2054.8 ± 511.32	.172
No	2857.2 ± 1455.1	
Types of M protein		
IgG	2862.9 ± 1492.5	.324
IgA	2166.1 ± 443.1	
Elevated LDH		
Yes	23417.3 ± 2423.3	.452
No	2550.1 ± 968.4	
Anemia (hemoglobin ≥2 g/dl to ≤10 g/dl)		
Yes	2845.8 ± 2086.21	.441
No	2671.1 ± 988.3	
Hypercalcemia (Ca ≥11.5 mg/dl)		
Yes	2031.01 ± 570.5	.066
No	2862.0 ± 1448.6	
Renal insufficiency (serum creatinine ≥2 mg/dl)		
Yes	2238.9 ± 668.2	.287
No	2844.65 ± 1482.5	

Table (5): Patient characteristics distribution and clinical presentation among the studied patients according to response.

		Complete response (n=40)	**Partial response** (n=10)	**P**
Age (years) Mean ± Collected_Paper_for_amateurs[1] SD		51.58 ± 10.72	61.83 ± 4.12	.031
Gender	Female	27 (67.5%)	3 (30%)	.030
	Male	13 (32.5%)	7 (70%)	
Comorbidities		27 (67.5%)	8 (80%)	.441
Treatment	VRD	21 (52.5%)	7 (70%)	.319
	VCD	19 (47.5%)	3 (30%)	
Hemoglobin (g/dl) Mean ± SD		10.6 ± 0.991	8.43 ± 1.6	<0.001
Platelets ($x10^3$/L) Mean ± SD		259.17 ± 72.57	195.0 ± 115.19	.146
Albumin (g/dl) Mean ± SD		3.51 ± 0.228	3.32 ± 0.147	.062
Creatinine (mg/dl) Mean ± SD		1.45 ± 0.784	1.28 ± 0.637	.735
β2-microglobulin (mg/l) Mean ± SD		4.18 ± 0.998	4.6 ± 1.11	.407
Calcium (mg/dl) Mean ± SD		10.14 ± 1.22	10.2 ± 1.15	.923
Bone marrow plasma cells Mean ± SD		40.54 ± 20.87	39.67 ± 20.53	.856

		Complete response (n=40)	Partial response (n=10)	P
BCMA (pg/ml) Mean ± SD		2894.3 ± 1298.5	2040.28 ± 403.51	0.046
Bence jones protein in urine		25 (62.5%)	7 (70%)	.659
Elevated LDH		8 (20%)	2 (20%)	1
Types of protein	IgG	32 (80%)	8 (80%)	1
	IgA	8 (20%)	2 (20%)	
Stage	I	13 (32.5%)	2 (20%)	**.038**
	II	12 (30%)	0	
	III	15 (37.5%)	8 (80%)	
Abnormal cytogenetics		10 (25%)	2 (20%)	.741
High risk FISH		9 (22.5%)	3 (30%)	.619
Lytic lesions		27 (67.5%)	8 (80%)	.441
EMD at diagnosis		6 (15%)	2 (20%)	.70
Anemia		7 (17.5%)	8 (80%)	**0.01**
Hypercalcemia		3 (7.5%)	5 (50%)	**0.01**
Renal insufficiency		8 (20%)	2 (20%)	1

Table (6): Regression analysis between B cell maturation antigen and different parameters.

	Unstandardized Coefficients		**Standardized Coefficients**	**t**	**Sig.**
	UPB cell maturation antigen (BCMA)DRS				
	B	**Std. Error**	**Beta**		
Age	-28.318	41.687	-.211	-.679	.507
Gender	-609.180	712.632	-.216	-.855	.406
comorbidities	1115.021	973.970	.377	1.145	.270
Hb	-85.434	328.607	-.077	-.260	.798
PLT	4.953	6.493	.296	.763	.457
Serum.albumin, g/dl	1407.018	1618.938	.232	.869	.398
creatinine	296.020	591.242	.162	.501	.624
inurine Bencejonesprotein.	757.916	873.888	.263	.867	.399
dehydrogenase Elevatedlactate.	-266.090	1187.343	-.079	-.224	.826
Serumbeta.2microglobulin, mg/l	186.619	452.806	.135	.412	.686
Calcium	119.978	361.633	.103	.332	.745
M.protein of Types	-580.907	887.810	-.160	-.654	.523
Bone marrow plasma cells	-20.107	15.780	-.300	-1.274	.222

Table (7): BCMA as a predictor of treatment response among MM patients.

AUC	**S.E.**	**Sig.**	**95% Confidence Interval**	
.715	.108	.045	.522 - .864	
Cutoff	**Sensitivity**	**Specificity**	**PPV**	**NPV**
> 2245	75%	83.3%	92.9	35.3

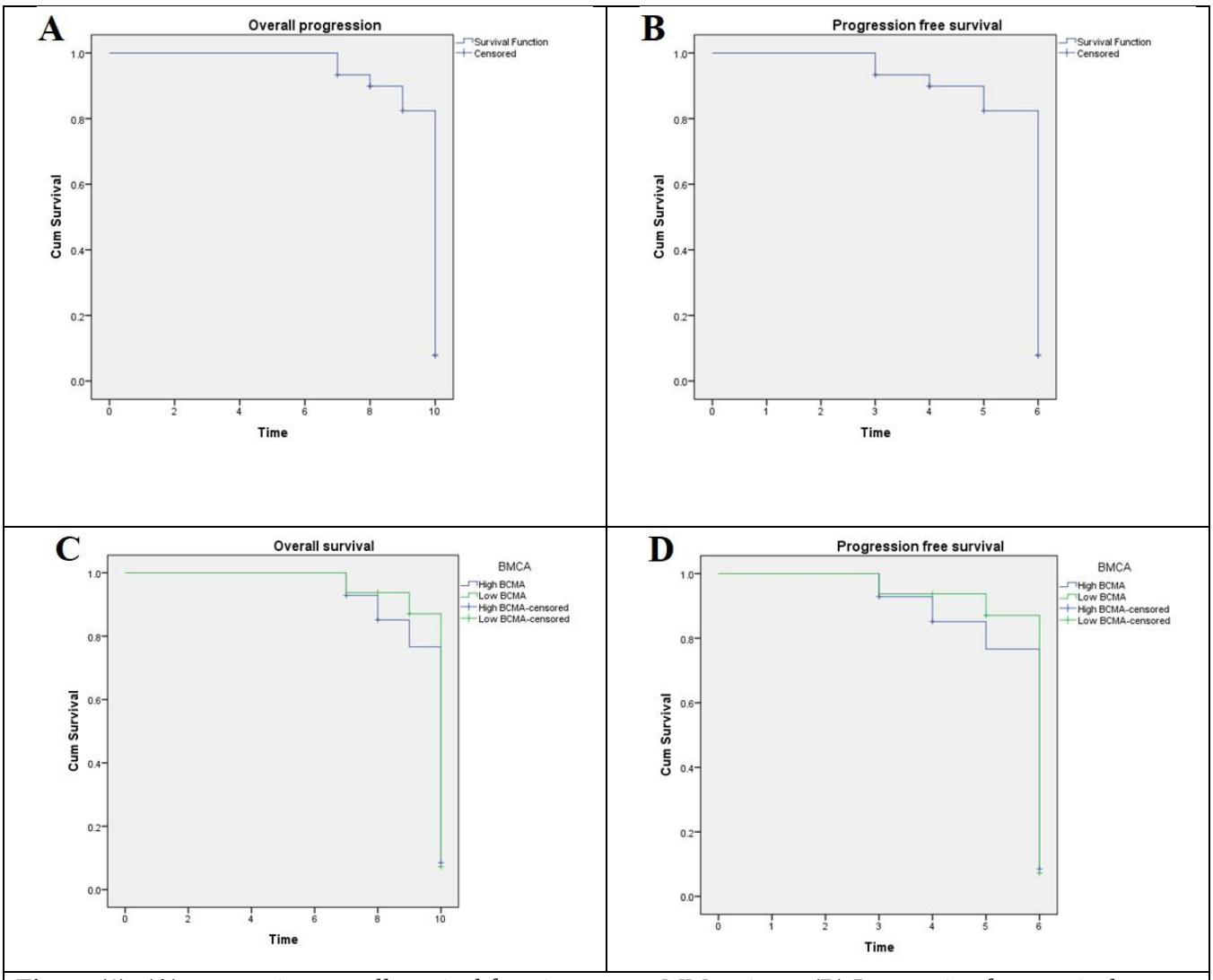

Figure (1): (A) progression overall survival function among MM patients. (B) Progression free survival among MM patients. (C) Overall survival among MM patients according to BCMA levels. (D) Progression free survival among MM patients according to BCMA levels.

DISCUSSION

MM is a malignant hematological disease characterized by the proliferation of monoclonal plasma cells. Numerous clinical outcomes, such as increased calcium levels, renal failure, anemia, and bone tumors, can result from this condition. The median OSS of MM cases has increased dramatically because to a number of innovative therapeutic medications. Nonetheless, there is still a significant chance of relapse, and there is a wide range of outcomes for MM [7,8]. Additionally, immune-based methods have been added to the list of MM treatment options. However, approaches for assessing the illness status of MM cases have not kept pace with this evolving profile. As a result, creating more efficient and reliable approaches for assessing and monitoring these cases has become increasingly critical [9]. BCMA has been linked to B-cell cancers. BCMA is lost from plasma cell membranes through γ-secretase cleavage, leading to a soluble form (sBCMA). This is critical in regulating B-cell growth and transformation into plasma cells. [10].

BCMA represents an impressive new target for MM treatments. Various types of BCMA-targeting medications, such as bispecific antibody complexes, ADCs, and CAR T-cell treatments, have demonstrated anti-myeloma activity in RRMM cases and could contribute to tackling a key unmet demand for therapeutics in MM cases. Despite the absence of trials in progress using BCMA-targeted treatment options for the management of newly diagnosed MM, these treatments may also provide promise for these cases, as demonstrated by the high ORR, high MRD negativity rates, and durable responses reported to date with select BCMA-targeted medications [6]. The primary goal of the research

was to assess the application of BCMA as a novel prognostic indicator in MM.

In the present study, we observed elevated BCMA levels above normal levels. In agreement Fadilah *et al*., [7] that BCMA was remarkably elevated in MM cases. This result was similar to Ghermezi *et al*., [1] findings that levels of BCMA were elevated in MM cases comparted to normal participants. Sanchez *et al*. [11] and Lee *et al*. 2016 were in line with these findings. Meanwhile, we found no association between Stage, LDH, abnormal cytogenetics, lytic lesions, anemia, hypercalcemia, and renal insufficiency. In consistency, Fadilah *et al*., [7] who reported that the relationship between lytic bone disease or ISS and BCMA was unremarkablet. This study findings were in accordance with by Sanchez *et al*. [11], Lee *et al*. [12] and Ghermezi *et al*. [1] results that BCMA is independent of MM bone disease. We found that there was a remarkable direct association between BCMA and platelets only ($p<0.05$). However, there was no significant relation between BCMA and different parameters and clinical characteristics. Similarly, Fadilah *et al*., [7] investigated the relationship between the analyzed factors and several laboratory variables. There was unremarkable relationship between BCMA and B2M, calcium, creatinine, or Hb. Concerning BCMA, these findings were in line with Ghermezi *et al*. [1], Lee *et al*. [12] and Sanchez *et al*. [11]. However, Fadilah *et al*., [7] found remarkable positive association between BCMA and plasma cells levels and pre and treatment. they performed association between BM findings and BCMA in non-secretory disease MM cases. Utilizing protein electrophoresis for M-protein evaluation is the best way for MM cases monitoring [13]. However, we found no substantial correlation between M-protein and BCMA.

In contrast, Fadilah *et al*., [7] and Ghermezi *et al*. [1] found a remarkable positive relationship between BCMA and M-protein concentration pre- and post-treatment. Furthermore, investigations have found that BCMA has a less half-life (24-36 hours) than IgA (7 days) and IgG (21 days).

In the present study, regarding response, 80% of the patients represented complete response, and 20% of the cases represented partial response. We documented that BCMA was significantly associated with response to treatment. In agreement, Fadilah *et al*., [7] and Sanchez *et al*. [11] documented a remarkable association between response to treatment and BCMA. They found that individuals with partial or complete remission (n = 80) had reduced BCMA concentrations than cases with progressing disease (n = 79) (4.06 vs. 19.76 ng/mL). Moreover, Jew *et al*. [13] stated that all 27 cases who attained CR had normalized BCMA levels after therapy. Nevertheless, 86% of cases who obtained SD or PD did not have normal BCMA levels after therapy. They figured out that BCMA normalization following therapy predicts a stronger overall response in rats.

The present study results as regards prediction of therapeutic response in MM, we found that BCMA yielded significance level at cutoff level >2245 in predicting complete response with sensitivity of 75% and specificity of 83.3%. Sanchez *et al*. [14] reported that BCMA could determine when cases fail to respond to their present treatment, permitting them to be switched to another treatment faster. Also, Ghermezi *et al*. [1] suggested that BCMA can assess the effectiveness of therapy more quickly.

Last but not least, we found that patients with high levels of BCMA had slightly shorter OS and PFS time than those with lower levels of BCMA, there was no significance difference. In consistency, Fadilah *et al*. [7] reported that cases with elevated BCMA concentrations had a substantially reduced PFS time than cases with reduced BCMA concentrations. In accordance to these results, Sanchez *et al*. [11] and Ghermezi *et al*. [1] indicated that increased BCMA was associated with a lower PFS and OS. They demonstrated that BCMA is an independent prognostic marker. Depending on Sanchez *et al*. [11] studies, Ghermezi *et al*. [1] reported that higher-than-median sBCMA concentrations predicted shorter PFS and OS ($p<0.05$). Another report by Sanchez *et al*. [14] stated that sBCMA values are negatively linked with uninvolved polyclonal antibodies generated in MM cases, indicating a possible sBCMA-mediated mechanism for immunological insufficiency in these cases.

Lee *et al*. [12] reported that sBCMA levels in 42 cases specimens varied from 3.8-1 062 ng/mL ($p < 0.0001$ vs. normal controls). There was no variance between NDMM cases and those with relapsed conditions. Contrary to our findings, there was no correlation between sBCMA levels and therapy survival or response.

Points of strength

This study sought to achieve its desired goal. It had strict inclusion, and exclusion criteria. We performed all the required laboratory tests for all included patients. BCMA-targeted therapies have demonstrated promising and exciting clinical results in heavily pretreated patients with RRMM.

Limitations

This study was prospective cohort study included 50 MM patients recruited from Hematology Unit of Internal Medicine Department Zagazig University hospitals, from July 2023 to January 2024. Small sample size. The included patients are needed and longer period for follow up. It was conducted in single center.

Author contribution: All authors contributed to the study. MHZ was responsible for selecting the subject, AAG, RKS, AFA were accountable for laboratory revisions and analysis, AMAB was responsible for data collection, statistical analysis, and initial writing, and TMG was responsible for collecting the data of the studied cases and all shared for the formulation of the study design, editing, revision, and preparation of the final manuscript.

Declaration of interest:

The authors report no conflicts of interest.

Funding information:

This study was not supported by any source of finding.

CONCLUSION

BCMA yielded significance in predicting complete response level at cutoff level >2245 in predicting complete response with sensitivity of 75% and specificity of 83.3%. It could aid in improved risk classification and more customized clinical care, improving therapeutic outcomes and elevating the life expectancy of MM patients.

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