RESEARCH ARTICLE

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Association between Vitamin D Deficiency and Angiographic Severity in Patients with Coronary Artery Disease

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Abstract

Background and Aim: This study explored the correlation between vitamin D status and the severity of coronary artery disease (CAD), as well as left ventricular function in patients with acute coronary syndrome (ACS).

Materials and Methods: This prospective observational study included 102 patients diagnosed with ACS admitted to an Indian tertiary care facility from January 2021 to December 2021. Upon admission, the researchers collected baseline data of the patients and measured serum vitamin D levels. CAD severity was evaluated using Gensini and SYNTAX scores, and left ventricular ejection fraction (LVEF) was measured using echocardiography.

Results: The study cohort had a median age of 56.5 years, with males comprising 62.7% of the total patient population. Anterior wall myocardial infarction was the most common presentation (59.8%), and 32.4% of patients had double vessel disease (DVD). The median vitamin D level was 18.3 ng/mL (interquartile range 12.7-26.8). Patients with vitamin D deficiency (≤20 ng/mL) exhibited significantly higher Gensini (46.5-94) and SYNTAX (7.5-38) scores than those with optimal levels (>30 ng/mL) ($P < 0.001$). A notable inverse correlation was found between vitamin D levels and both Gensini (r=-0.572, $P < 0.001$) and SYNTAX (r=-0.787, $P < 0.001$) scores. Reduced vitamin D levels were linked to decreased LVEF ($P = 0.018$) and a higher incidence of multivessel disease, particularly DVD ($P = 0.009$).

Conclusion: This study revealed a significant negative correlation between low vitamin D status, CAD severity, and left ventricular dysfunction in patients with ACS. These results suggest that low vitamin D status indicates vitamin D deficiency and plays a crucial role in the occurrence and progression of coronary atherosclerosis.

Keywords: Acute coronary syndrome, coronary artery disease, coronary angiography, ventricular dysfunction, left, vitamin D

INTRODUCTION

Acute coronary syndrome (ACS) accounts for a large proportion of morbidity and mortality worldwide. Among them, atherosclerosis is a significant cause of myocardial infarction leading to majority cases of mortality.[1,2] Hypertension, dyslipidemia, smoking, diabetes, and a history of cardiovascular disease are some well-established risk factors of ACS.[3]

Researchers have discovered new inflammatory markers like vitamin d, which is a fat-soluble vitamin playing a vital role in numerous physiological processes apart from its importance calcium homeostasis.[4] Several mechanisms that contribute to progression of cardiovascular diseases include its anti-inflammatory, anti-thrombotic, and anti-hypertensive properties, as well as its ability to modulate endothelial function and vascular calcification.[5]

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Hence, early identification and appropriate management of vitamin D deficiency in ACS patients may have the potential to improve clinical outcomes and prevent complications. However, the results have been inconsistent, with some studies failing to establish a significant relationship between vitamin D levels and clinical outcomes in patients with ACS. This study aimed to investigate the association between vitamin D levels and the extent of coronary artery disease (CAD) and left ventricular dysfunction in patients with ACS.

METHODS

Study Design and Population

This prospective observational study was performed at a tertiary care center in India between January 2021 and December 2021. All patients admitted to intensive cardiac care unit with a diagnosis of ACS, including unstable angina, ST segment elevation myocardial infarction, and non-ST segment elevation myocardial infarction, were included in the study. Patients with chronic kidney disease, known parathyroid hormone disorders, or who were taking calcium or vitamin D supplements were excluded from the study. After obtaining written informed consent, 102 eligible patients were enrolled in the study. The study adhered to the principles of the Declaration of Helsinki and was approved by the Institutional Ethics Committee M.K.C.G. Medical Collage (number: 1005, date: 18.12.2020).

Data Collection

For each patient, baseline clinical data, including age, sex, weight, blood pressure, and risk factors. ACS diagnosis was confirmed using clinical history, electrocardiogram findings, and elevated cardiac enzyme levels. We calculated the estimated glomerular filtration rate (eGFR) using the Chronic Kidney Disease Epidemiology Collaboration and measured the left ventricular ejection fraction (LVEF) via echocardiography during the initial hospitalization.

Laboratory and Angiographic Assessments

Upon admission, venous blood samples were collected to measure serum vitamin D levels. All patients underwent coronary angiography during their hospital stay. CAD assessment was performed with the help of two methods: Gensini score (GS) and SYNTAX score. The GS was calculated using a standardized algorithm that considered the degree of luminal narrowing and the significance of the affected coronary artery segments. Figure 1 outlines the step-by-step calculation process.[6] The SYNTAX scoring system is a detailed

STEP 1 Calculation of the severity score for each lesion ≥ 25% and adjustment for total occlusions or 99% obstructive lesions receiving collaterals

Degree of stenosis (%)	Receiving collaterals	Adjustment for collaterals	Severity Score
1-25	-	0	1
26-50	-	0	2
51-75	-	0	4
76-90	-	0	8
91-99	no	0	16
99	yes	-8	8
100	no	0	32
100	yes, and normal source vessel	-16	32-16=16
100	yes, and 25% stenosis source vessel	-12	32-12=20
100	yes, and 50% stenosis source vessel	-8	32-8=24
100	yes, and 75% stenosis source vessel	-4	32-4=28
100	yes, and 90% stenosis source vessel	-2	32-2=30
100	yes, and 99% stenosis source vessel	-1	32-1=31

STEP 2 A multiplying factor is applied to each lesion score based upon its location in the coronary tree

Segment	Right Dominance	Left Dominance
RCA proximal	1	1
RCA mid	1	1
RCA distal	1	1
PDA	1	1
PLB	0.5	0.5
Left Main	5	5
LAD proximal	2.5	2.5
LAD mid	1.5	1.5
LAD apical	1	1
1st Diagonal	1	1
2nd Diagonal	0.5	0.5
LCx proximal	2.5	3.5
LCx mid	1	2
LCx distal	1	2
Obtuse Marginal	1	1

STEP 3 Sum of all the lesion severity scores

Figure 1: Procedure for calculating the Gensini score; (A) step 1: Lesion severity assessment- for lesion ≥25% and total occlusions or 99% obstructive lesions receiving collaterals. (B) step 2: Apply a multiplication factor to each lesion score, the factor varies based on the lesions position in coronary system (C) step 3: Total of all the lesion severity scores

method for evaluating CAD. This numerical assessment reflects both the intricacy and extent of lesions in the coronary arteries. This score was determined using a computer program with sequential, interactive questions, as shown in Figure 2, which illustrates the algorithm's 12 main components.[7]

Definition

Vitamin D status is typically calculated by assessing serum 25(OH)D levels, with concentrations below 30 nmol/L generally considered deficient, 30-50 nmol/L insufficient, and above 50 nmol/L adequate, according to guidelines set by major health organizations such as The European Food Safety Authority, Endocrine Society, Institute of Medicine, and the Scientific Advisory Committee on Nutrition.[8]

Statistical Analysis

IBM® SPSS® Statistics software was used for data analysis. Continuous variables are presented as mean ± standard deviation, whereas categorical variables are expressed as frequencies and percentages. The Kolmogorov-Smirnov test was used to examine the relationships between vitamin D levels and various clinical characteristics, angiographic findings (Gensini and SYNTAX scores), and left ventricular dysfunction. We considered *P*-value <0.05 as statistically significant.

RESULTS

Our study involved 102 patients comprising 62.7% of male. Table 1 presents an overview of the initial demographic and clinical data of the study participants. Common risk factors were diabetes (53.9%), hypertension (40.2%), dyslipidemia (32.4%), smoking (31.4%), arrhythmia (16.6%), and thrombosis (15.7%). The median vitamin D concentration was 18.3 ng/mL (IQR 12.7-26.8). Kidney function, as measured by the eGFR, had a median of 90 mL/min/1.72 m^2 (IQR 80.8-99.3). The median ejection fraction was 48% (IQR 42-50.5). The Gensini and SYNTAX scores, which were used to assess CAD, had median values of 64 (IQR 42-82) and 20 (IQR 9.8-24.1) respectively.

Vitamin D status varied among participants; 35.4% were deficient (11-20 ng/mL), 33.3 % had suboptimal levels (21-30 ng/mL), 18.6 % were severely deficient (≤10 ng/mL), and only 12.7% maintained optimal levels (>30 ng/mL).

The correlation between patient vitamin D concentrations and various demographic and clinical factors are presented in Table 2. Vitamin D concentrations were correlated with the extent of CAD. Patients with single vessel disease had significantly higher vitamin D levels ($P = 0.047$), whereas those with double vessel disease (DVD) had lower vitamin D levels ($P = 0.009$). Although not statistically significant, there was a trend toward decreased vitamin D intake in patients with TVD ($P = 0.188$) and left main coronary artery involvement ($P = 0.660$).

A significant association emerged between vitamin D and LVEF. An LVEF of 54% (IQR 48-60 %) was observed in patients with vitamin D levels >30 ng/mL which was considerably higher than those with levels ≤20 ng/mL (median 46-48%, $P = 0.018$).

Table 3 illustrates that patients with the lowest vitamin D levels (≤10 ng/mL) had a substantially higher median GS of 88 (IQR

1. Dominance
2. Number of lesions
3. Segments involved per lesion

Lesion Characteristics

4. Total occlusion
   i. Number of segments involved
   ii. Age of the total occlusion (>3 months)
   iii. Blunt Stump
   iv. Bridging collaterals
   v. First segment beyond the occlusion visible by antegrade or retrograde filling
   vi. Side branch involvement
5. Trifurcation
   i. Number of segments diseased
6. Bifurcation
   i. Type
   ii. Angulation between the distal main vessel and the side branch <70°
7. Aorto-ostial lesion
8. Severe tortuosity
9. Length >20mm
10. Heavy calcification
11. Thrombus
12. Diffuse disease/small vessels

Segment No		Right dominance	Left dominance
1	RCA proximal	1	0
2	RCA mid	1	0
3	RCA distal	1	0
4	Posterior descending artery	1	n.a.
16	Posterolateral branch from RCA	0.5	n.a.
16a	Posterolateral branch from RCA	0.5	n.a.
16b	Posterolateral branch from RCA	0.5	n.a.
16c	Posterolateral branch from RCA	0.5	n.a.
5	Left Main	5	6
6	LAD proximal	3.5	3.5
7	LAD mid	2.5	2.5
8	LAD apical	1	1
9	First diagonal	1	1
9a	First diagonal[a]	1	1
10	Second diagonal	0.5	0.5
10a	Second diagonal[a]	0.5	0.5
11	Proximal circumflex artery	1.5	2.5
12	Intermediate/ anterolateral artery	1	1
12a	Obtuse marginal[a]	1	1
12b	Obtuse marginal[b]	1	1
13	Distal circumflex artery	0.5	1.5
14	Left posterolateral	0.5	1
14a	Left posterolateral[a]	0.5	1
14b	Left posterolateral[b]	0.5	1
15	Posterior descending	n.a.	1

Diameter reduction*	
- Total occlusion	x5
- Significant lesion (50-99%)	x2
Total occlusion (TO)	
- Age >3months or unknown	+1
- Blunt stump	+1
- Bridging	+1
- First segment visible beyond TO	+1/ per non-visible segment
- Side branch (SB) - Yes, SB <1.5mm**	+1
- Yes, both SB < & ≥ 1.5mm	+1
Trifurcations	
- 1 diseased segment	+3
- 2 diseased segments	+4
- 3 diseased segments	+5
- 4 diseased segments	+6
Bifurcations	
- Type A, B, C	+1
- Type D, E, F, G	+2
- Angulation <70°	+1
Aorto ostial stenosis	+1
Severe tortuosity	+2
Length > 20mm	+1
Heavy calcification	+2
Thrombus	+1
"Diffuse disease"/small vessels	+1/ per segment number

x: multiplication
+: addition

* In the SYNTAX algorithm there is no question for % luminal diameter reduction. The lesions are considered as significant (50-99% luminal diameter reduction) or occlusive.

** If all the side branches are 1.5mm in diameter, no points are added since the lesion is considered as a bifurcation and it will be scored as such.

Figure 2: (A) SYNTAX score algorithm and (B) Scoring system of the SYNTAX score characteristics points

68-92) compared with those with >30 ng/mL, who had a score of just 16 (IQR 6-32) ($P < 0.001$). A similar trend was observed for SYNTAX scores, ranging from a median of 36 (IQR 30-38) in the lowest vitamin D level to those in the highest 5 (IQR 3-11) ($P < 0.001$).

Table 1: Baseline clinical characteristics

Characteristics	n=102 patients
Male, n (%)	64 (62.7)
Risk factors	
Diabetes, n (%)	55 (53.9)
Hypertension, n (%)	41 (40.2)
Dyslipidaemia, n (%)	33 (32.4)
Smoking, n (%)	32 (31.4)
Arrhythmia, n (%)	17 (16.6)
Thrombosis, n (%)	16 (15.7)
Type of MI	
AWMI, n (%)	61 (59.8)
IWMI, n (%)	17 (16.7)
IWMI + PWMI, n (%)	10 (9.8)
IWMI+ RVMI, n (%)	8 (7.8)
IWMI + PWMI + RVMI, n (%)	6 (5.9)
KILLIP classification	
Class I, n (%)	67 (65.7)
Class II, n (%)	25 (24.5)
Class III, n (%)	0 (0)
Class IV, n (%)	10 (9.8)
NYHA classification	
Class 1, n (%)	65 (63.7)
Class 2, n (%)	26 (25.5)
Class 3, n (%)	11 (10.8)
Coronary artery involvement	
Normal coronary, n (%)	9 (8.8)
SVD, n (%)	32 (31.4)
DVD, n (%)	33 (32.4)
TVD, n (%)	27 (26.5)
LMCA, n (%)	4 (3.9)
Vitamin D, ng/mL [(median (IQR)]	18.3 (12.7-26.8)
eGFR, mL/min/1.73 m^2 [median (IQR)]	90 (80.8-99.3)
LVEF, % [median (IQR)]	48 (42-50.5)
Gensini scores [median (IQR)]	64 (42-82)
Syntax scores [median (IQR)]	20 (9.8-24.1)

Data are expressed as n (%) and median (IQR).

AWMI: Anterior wall myocardial infarction, DVD: Double vessel disease, eGFR: Estimated glomerular filtration rate, IWMI: Inferior wall myocardial infarction, LMCA: Left main coronary artery, LVEF: Left ventricular ejection fraction, MI: Myocardial infarction, NYHA: New York Heart Association, PWMI: Posterior wall myocardial infarction, RVMI: Right ventricular myocardial infarction, SVD: Single vessel disease, TVD: Triple vessel disease

As shown in Table 4, there was a significant negative correlation between vitamin D levels and both Gensini (r=-0.572, $P < 0.001$), and SYNTAX scores (r=-0.787, $P < 0.001$), indicating that lower vitamin D levels were associated with more severe CAD.

DISCUSSION

Vitamin D deficiency is a pervasive health concern affecting diverse demographic groups. While its role was primarily known for calcium regulation, recent research has highlighted the potential significance of vitamin D in CAD. There are studies that suggest links between vitamin D levels and acute myocardial infarction risk and outcomes associated with high rates of illness and death, emphasizing the importance of vitamin D supplementation beyond bone metabolism.[9] Our findings support this connection, demonstrating that low vitamin D status is associated with a higher probability of CAD, its angiographic severity, and a greater incidence of vitamin D insufficiency and deficiency among patients, which is consistent with previous studies. In a prior study, 83% of the patients had vitamin D level <30 ng/mL[10]. This finding was observed to be similar in our study, in which 87.3% of the patients had vitamin D levels <30 ng/mL. However, Syal et al.[11] in their study revealed that a substantial majority of patients (93%) had vitamin D concentrations below 30 ng/mL, which is considered insufficient, and only a small fraction (7%) of patients demonstrated adequate vitamin D levels. Notably, only 12.7% of patients in our study had sufficient vitamin D levels (> 30 ng/mL). Rahman et al.[12] observed that most of the male patients exhibited vitamin D concentrations below 30 ng/mL, which demonstrates gender differences in vitamin D levels. However, this finding is in contrast with other research that suggests women typically have lower vitamin D levels. The potential causes postulated were variations in adipose tissue composition, insufficient nutritional composition, reproductive events, and onset of menopause.[13,14] In our study, no difference in vitamin D levels was noted among both genders ($P = 0.425$). The present study revealed a significant relationship between decreased vitamin D levels and reduced LVEF, which is a marker of left ventricular dysfunction. These results align with earlier research that has established a correlation between vitamin D insufficiency and a heightened susceptibility to heart failure as well as adverse cardiac remodeling.[15,16]

The correlation between vitamin D insufficiency and increased susceptibility to cardiovascular complications have been elucidated. Multiple extensive observational studies, both cross-sectional and longitudinal, have shown a relationship with diminished vitamin D levels and a higher incidence of cardiovascular events.[10,17] Furthermore, insufficient vitamin D levels have been identified as a significant biological indicator for elevated rates of various cardiovascular disease risk factors, including hypertension, obesity, diabetes, and

Table 2: Classification of vitamin D levels according to clinical characteristics

Variables	Vitamin D level (ng/mL)					*P*-value
		≤10 (n=19)	11 to 20 (n=36)	21 to 30 (n=34)	>30 (n=13)	
Age, n (%)	<40 years	3 (27.3)	3 (27.3)	2 (18.2)	3 (27.3)	0.266
	40 to 60 years	12 (21.8)	22 (40.0)	16 (29.1)	5 (9.1)	
	>60 years	4 (11.1)	11 (30.6)	16 (44.4)	5 (13.9)	
BMI, median (IQR)	kg/m^2	28.3 (25.7-31.2)	27.7 (26.3-30.1)	28.8 (27-30.8)	28 (26.6-32.2)	0.519
Gender, n (%)	Male	12 (18.8)	26 (40.6)	18 (28.1)	8 (12.5)	0.425
	Female	7 (18.4)	10 (26.3)	16 (42.1)	5 (13.2)	
Diabetes, n (%)	Yes	8 (14.5)	22 (40.0)	19 (34.5)	6 (10.9)	0.535
	No	11 (23.4)	14 (29.8)	15 (31.9)	7 (14.9)	
Hypertension, n (%)	Yes	5 (12.2)	17 (41.5)	14 (34.1)	5 (12.2)	0.514
	No	14 (23.0)	19 (31.1)	20 (32.8)	8 (13.1)	
Dyslipidaemia, n (%)	Yes	6 (18.2)	13 (39.4)	11 (33.3)	3 (9.1)	0.892
	No	13 (18.8)	23 (33.3)	23 (33.3)	10 (14.5)	
Smoking, n (%)	Yes	6 (18.8)	13 (40.6)	8 (25.0)	5 (15.6)	0.648
	No	13 (18.6)	23 (32.9)	26 (37.1)	8 (11.4)	
Thrombosis, n (%)	Yes	1 (6.3)	5 (31.3)	7 (43.8)	3 (18.8)	0.42
	No	18 (20.9)	31 (36.0)	27 (31.4)	10 (11.6)	
KILLIP class, n (%)	I	15 (22.4)	24 (35.8)	20 (29.9)	8 (11.9)	0.88
	II	3 (12.0)	8 (32.0)	10 (40.0)	4 (16.0)	
	III	0 (0)	0 (0)	0 (0)	0 (0)	
	IV	1 (10.0)	4 (40.0)	4 (40.0)	1 (10.0)	
NYHA class, n (%)	1	10 (15.4)	23 (35.4)	22 (33.8)	10 (15.4)	0.302
	2	6 (23.1)	8 (30.8)	11 (42.3)	1 (3.8)	
	3	3 (27.3)	5 (45.5)	1 (9.1)	2 (18.2)	
SVD, n (%)	Yes	3 (9.4)	8 (25.0)	14 (43.8)	7 (21.9)	0.047
	No	16 (22.9)	28 (40.0)	20 (28.6)	6 (8.6)	
DVD, n (%)	Yes	9 (27.3)	15 (45.5)	9 (27.3)	0 (0)	0.009
	No	10 (14.5)	21 (30.4)	25 (36.2)	13 (18.8)	
TVD, n (%)	Yes	7 (25.9)	12 (44.4)	7 (25.9)	1 (3.7)	0.188
	No	12 (16.0)	24 (32.0)	27 (36.0)	12 (16.0)	
LMCA, n (%)	Yes	0 (0)	2 (50.0)	1 (25.0)	1 (25.0)	0.66
	No	19 (19.4)	34 (34.7)	33 (33.7)	12 (12.2)	
100% cut, n (%)	Yes	2 (16.7)	2 (16.7)	7 (58.3)	1 (8.3)	0.271
	No	17 (18.9)	34 (37.8)	27 (30.0)	12 (13.3)	

Data are expressed as n (%) and median (IQR). *P*-value <0.05 was considered statistically significant. DVD: Double vessel disease, LMCA: Left main coronary artery, NYHA: New York heart association, SVD: Single vessel disease, TVD: Triple vessel disease

metabolic syndrome. Moreover, inadequate vitamin D concentrations are correlated with determinants of disease progression, including inflammatory responses. The expanding compendium of empirical evidence indicates that vitamin D status may be integral to the maintenance of cardiovascular health.[18] In the present investigation, the correlation between diverse risk factors and vitamin D levels was evaluated, and no statistically significant differences were observed. However, Hussein et al.[19] reported that diabetes and dyslipidemia were significantly correlated with vitamin D levels. Our study found a higher prevalence of multivessel disease, particularly DVD, among patients with lower vitamin D levels. This observation is consistent with the findings of Rahman et al.[12], who documented a noteworthy association between vitamin D

Table 3: Vitamin D levels in relation to GFR, ejection fraction, and angiographic scores

Variables	Vitamin D level (ng/mL)				*P*-value
	≤10	11 to 20	21 to 30	>30	
eGFR, mL/min/1.73 m² [median (IQR)]	90 (85-94)	88 (80-97.8)	86 (79-99.3)	99 (90-108)	0.088
LVEF, % [median (IQR)]	48 (42-50)	46 (42-48)	48 (44-50)	54 (48-60)	0.018
Gensini scores [median (IQR)]	88 (68-92)	71 (60-82)	58 (46.5-72.5)	16 (6-32)	<0.001
Syntax scores [median (IQR)]	36 (30-38)	23 (20.4-24.5)	11.5 (7.5-18)	5 (3-11)	<0.001

Data are expressed as n (%) and median (IQR). *P*-value <0.05 was considered statistically significant.

eGFR: Estimated glomerular filtration rate, LVEF: Left ventricular ejection fraction

Table 4: Correlation between vitamin D level and coronary artery disease severity scores

Severity scores	Correlation coefficient	*P*-value
Gensini score	-0.572	<0.001
Syntax score	-0.787	<0.001

P-value <0.05 was considered statistically significant

levels and the severity of CAD involvement. A previous study showed a significant inverse relationship between vitamin D levels and GS noted (r=-0.430, $P < 0.001$).[19] Comparable findings were likewise noted in the present investigation. The augmented SYNTAX score, which serves as a metric for evaluating the intricacy of CAD, has been demonstrated to function as an independent prognostic factor for significant adverse cardiac events in individuals diagnosed with ACS.[2] The current investigation revealed that vitamin D levels exhibited an inverse correlation with the severity and complexity of CAD, as quantified by the SYNTAX score, which is consistent with the findings observed in our research. Earlier research conducted by Seker et al.[18] demonstrated a comparable negative correlation between vitamin D concentrations and SYNTAX scores, with statistical significance (r=-0.549, $P < 0.001$). Although the exact processes linking low vitamin D levels to the extent of CAD remain incompletely understood, they likely encompass multiple biological pathways. Vitamin D possesses several beneficial properties that may influence cardiovascular health. These strategies include reducing inflammation, preventing blood clots, lowering blood pressure, and regulating the function of blood vessel linings. Vitamin D also plays a role in controlling calcium deposits in blood vessels. These various effects could potentially impact the formation and advancement of atherosclerosis, which might result in more significant and widespread damage to the coronary arteries.[18]

Study Limitations

Our study had a single center design and a compatibility-limited sample size, which may have constrained the external validity of the results. Moreover, the study failed to consider potential confounding factors, including dietary habits and sun exposure, which could influence vitamin D levels. The absence of a control group within the same age group, comparable risk factors, and comprehensive patient data represents a significant methodological limitation of our study. Consequently, further extensive investigations are warranted to confirm these results and explore possible therapeutic implications.

CONCLUSION

The present study highlights a significant negative correlation between vitamin D status and the severity of CAD, as evidenced by the Gensini and SYNTAX scores, in patients with ACS. Furthermore, reduced vitamin D levels were associated with decreased LVEF and a higher prevalence of multivessel disease, particularly DVD.

Ethics

Ethics Committee Approval: The study adhered to the principles of the Declaration of Helsinki and was approved by the Institutional Ethics Committee M.K.C.G. Medical Collage (number: 1005, date: 18.12.2020).

Informed Consent: Informed consent was obtained from patients.

Footnotes

Authorship Contributions

Surgical and Medical Practices: K.K.S., H.G., Concept: K.K.S., H.G., Design: K.K.S., Data Collection or Processing: H.G., Analysis or Interpretation: K.K.S., H.G., Literature Search: K.K.S., H.G., Writing: K.K.S., H.G.

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