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REVIEW ARTICLE

Culprit Lesion Morphology on Optical Coherence Tomography in ST-elevation Myocardial Infarction vs Non ST-elevation Myocardial Infarction − A Systematic Review of 7526 Patients

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Abstract

Background: **Patients with STEMI are postulated to have different culprit lesion morphology compared to NSTEMI. The use of OCT in ACS can help delineate lesion morphology. The aim of this systematic review was to analyze the available data on culprit plaque morphology in ACS patients.**

Methods: **The available literature was systematically screened for studies on culprit lesion morphology in ACS patients. Data was extracted from the selected studies and analyzed for baseline characteristics as well as culprit lesion morphology on OCT. Lesion characteristics between STEMI and NSTEMI groups were compared.**

Results: **A total of 32 studies were selected for the final analysis. The average age of the study population was 62.4 years. Majority of patients (66.6%) had STEMI on presentation. NSTEMI patients had a higher prevalence of diabetes compared to STEMI. Both STEMI and NSTEMI patients had similar prevalence of thin-cap fibroatheroma (44.9%). The mean fibrous cap thickness was 84.2 μm in the study. STEMI patients had higher prevalence of lipid plaques, macrophages and luminal thrombus as compared to NSTEMI patients. Plaque rupture was the predominant culprit lesion morphology in both STEMI and NSTEMI groups, with higher prevalence in STEMI patients. Plaque erosion was also more common in STEMI patients (34.4% vs 13.2%).**

Conclusion: **Plaque rupture is the predominat culprit lesion morphology in both STEMI and NSTEMI patients, despite having differences in baseline characteristics. Use of OCT to determine plaque morphology in ACS patients can help guide management strategy in select cases. [PROSPERO CRD42021249742].**

Keywords: Optical coherence tomography, Acute coronary syndrome, Plaque erosion, Plaque rupture

1. Introduction

Acute coronary syndrome (ACS) has been conventionally linked with plaque rupture and thrombosis. However, recent studies have elucidated distinct mechanisms for ACS events − plaque rupture, plaque erosion and superficial calcified nodules [1]. Coronary angiography does not provide information regarding culprit lesion morphology, which varies amongst patients. Identification of lesion morphology is important as it helps us to individualise therapy for patients rather than adopting a "stent all" approach, as well as analysing their natural history at the same time [2]. Intravascular imaging plays an important role in helping us achieve this objective.

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Optical coherence tomography (OCT) has lent a cutting edge to the field of intravascular imaging with its excellent resolution and sensitivity. Its superior sensitivity over other intravascular imaging modalities has been proven in various studies [3]. One of the most important characteristics of OCT is its ability to determine the plaque morphology and delineate its components. The high resolution of OCT enables the measurement of fibrous cap thickness, thus identifying vulnerable plaques. Delineation of plaque morphology becomes pertinent as it determines the clincial presentation and course in patients presenting with ACS. Plaque rupture was associated with more myocardial damage and larger infarct size when compared to lesions with intact fibrous cap [4]. OCT imaging has aptly shown that culprit plaque morphology varies amongst patients with ACS [5]. Detection of culprit plaque morphology helps in prognostication of the patients.

Conventionally, patients presenting with ACS are classified as STEMI and NSTEMI/UA depending on clinical features. This classification system does not provide adequate information regarding the etiology of ACS. In-vivo imaging studies have been performed in ACS patients for determination of culprit lesion morphology. However, there is a gap in literature regarding definitive etiology amongst patients presenting with STEMI and NSTEMI. It is imperative to have a pooled analysis of available data on the current topic. This will pave the way for future research in the same area and help in clinical management. This systematic review was performed with the aim of identifying the most common culprit lesion morphology in ACS along with notable differences in plaque morphology between STEMI and NSTEMI/UA patients.

2. Materials and methods

2.1. Search strategy

The search strategy was developed according to the guidelines laid down by Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA). The study was registered with PROSPERO (CRD42021249742). We performed an extensive electronic search of four databases: PubMed, Embase, Google Scholar, and World Health Organization library, using the keywords: "optical coherence tomography" or "acute coronary syndrome "or "myocardial infarction "or "plaque rupture" or "plaque erosion" on January 23,2021. The reference lists of the initially selected studies were screened for additional sources. All duplicate studies in the search were removed.

Abbreviations

ACS	Acute coronary syndrome
CAD	Coronary artery disease
LAD	Left anterior descending artery
NIH	National institute of health
NSTEMI	Non ST elevation myocardial infarction
OCT	Optical coherence tomography
PRISMA	Preferred reporting items for systematic reviews and meta-analysis
QCA	Quantitative coronary angiography
RCA	Right coronary artery
STEMI	ST elevation myocardial infarction
TCFA	Thin cap fibroatheroma

2.2. Study selection

The inclusion criteria for the research selected in this review were those studies which provided OCT data on culprit lesion morphology in patients presenting with ACS. Additional criteria were that the research was published in English, the entire data was fully extractable and was performed on humans. Studies which had a sample size of more than 10 were included. Observational studies or randomized control trials (RCT) were included in the current review. Case reports, pictorial reviews, systematic reviews,meta-analysis, and review articles were not included. AM and VO were responsible for initial selection of studies for the review. The selected studies were independently reviewed by HS, SG and BS, based on the above-mentioned inclusion criteria. Disagreements were resolved by consensus and if needed, in consultation with a senior reviewer (AKV). The quality of the selected studies was assessed on the basis of Newcastle Ottawa scale by two independent reviewers (AM and VO) [6]. We aimed to conduct a narrative synthesis and review of the findings. Ethical committee approval was waived for this study as it did not include recruitment of any patients.

2.3. Data extraction

The data from the selected studies was extracted by AM and VO using a Microsoft Excel datasheet. Data extracted was grouped under multiple headings like year and country of publication, sample size, demographics, clinical characteristics, angiographic characteristics, and OCT findings. Various subfields were used to classify the angiographic data and OCT data in the form of vessels involved, lesion length, minimum lesion diameter, plaque cap thickness, lipid arc extent, thrombus and cap stability. Any discrepancies found were resolved with

consensus amongst the reviewers. All analysis of the data was done using SPSS version 25 (IBM, Armonk, NY). We primarily aimed to perform a narrative synthesis of the findings (synthesis without meta-analysis). PRISMA guidelines were followed during analysis and reporting of this study.

2.4. OCT data analysis

OCT data was analysed (BL,AKV) according to the standard definitions used in clinical practice. Culprit lesion morphology was defined as plaque rupture, plaque erosion, and calcific nodule. Plaque rupture was defined as a discontinuity in the fibrous cap of the plaque along with a cavity formation. It is usually associated with a lipid rich plaque. Plaque erosion was defined as the presence of an intact fibrous cap with/without the presence of intracoronary thrombus. In the absence of luminal thrombus, mild luminal irregularities can point to the diagnosis of probable plaque erosion. Eruptive calcific nodule was defined as calcium deposits projecting into the vessel lumen, disrupting the fibrous cap. Thin cap fibroatheroma was defined as fibrous cap thickness of <65 μm [7].

2.5. Patient and public involvement

As this is a systematic review of previously published studies, there was no direct public/patient involvement in the present study.

3. Results

3.1. Study selection

A total of 3275 studies were screened for this review. After eligibility assessment, 32 studies were selected for the final pooled analysis (Fig. 1). Amongst the selected studies, 11 were from Japan [8–18], 6 were from China [19][[20–24], 5 were from Italy [25–29], 4 were from USA [30–33] whereas there was one study each from Canada [34], Egypt [35] and France [36]. The remaining 3 studies were multicenter studies, which involved multiple institutions in different countries [37–39] (Supplementary Table 1). Methodologic quality, as assessed by Newcastle Ottawa scale by two reviewers (AM and VO), was fair for most of the studies (Supplementary Table 2).

3.2. Study population characteristics

The average age of the study population was 62.7 years and females comprised 23% of the study group. The baseline characteristics of the study patients are described in Table 1. Majority of patients had risk factors in the form of diabetes, hypertension, and dyslipidemia. STEMI was the most common presentation (66.6%) in this cohort. Average LDL levels in the study group were 114.4 ± 9.3 mg/dl. LAD was the most common culprit vessel (50.8%) followed by RCA (36.2%), as noted on coronary angiography (Table 2). Majority of the lesions were situated in the mid segment of vessels. Average diameter stenosis was 79.6 ± 12.7%. About two-thirds of patients had complex angiographic lesions (AHA/ACC type B2/C).

3.3. OCT analysis

Pooled OCT characteristics of the culprit lesions are enumerated in Table 3. Average fibrous cap thickness was 84.2 ± 23.4 μm. Thin-cap fibroatheroma was noted in 44.9% patients whereas lipid rich plaques were noted in 71.6%. Average lipid arc noted on OCT was 228°. OCT detected luminal thrombus in 75% of the study population. Patients in the study group had a higher proportion of white thrombus in comparison to red thrombus. Macrophages were seen in two-thirds of the study population whereas microchannels and microcalcifications were noted in about 35% of the study cohort. Average area stenosis on OCT was 73.2 ± 5.8%. The most common plaque morphology in culprit lesions was plaque rupture (54.2%). On the other hand, plaque erosion was noted in 35% of the patients.

3.4. Subgroup analysis (STEMI vs NSTEMI)

A subgroup analysis was performed between STEMI and NSTEMI patients regarding baseline characteristics and OCT plaque morphology. Both STEMI and NSTEMI patients had a mean age of 62 years. The proportion of females was similar in both the groups (25% vs 27.9%, $p = 0.36$) (Table 4). Patients with NSTEMI had a higher proportion of diabetes and tobacco intake compared to STEMI group ($p < 0.05$). LAD was the most common vessel involved in both STEMI and NSTEMI patients. However, proportion of LAD involvement was significantly higher in NSTEMI group (65.8% vs 51.3%, respectively) whereas RCA involvement was higher in STEMI group (43.1% vs 25.2%, respectively).

OCT analysis showed that there was no significant difference in minimum fibrous cap thickness amongst STEMI and NSTEMI patients. Both STEMI and NSTEMI patients had similar incidence of

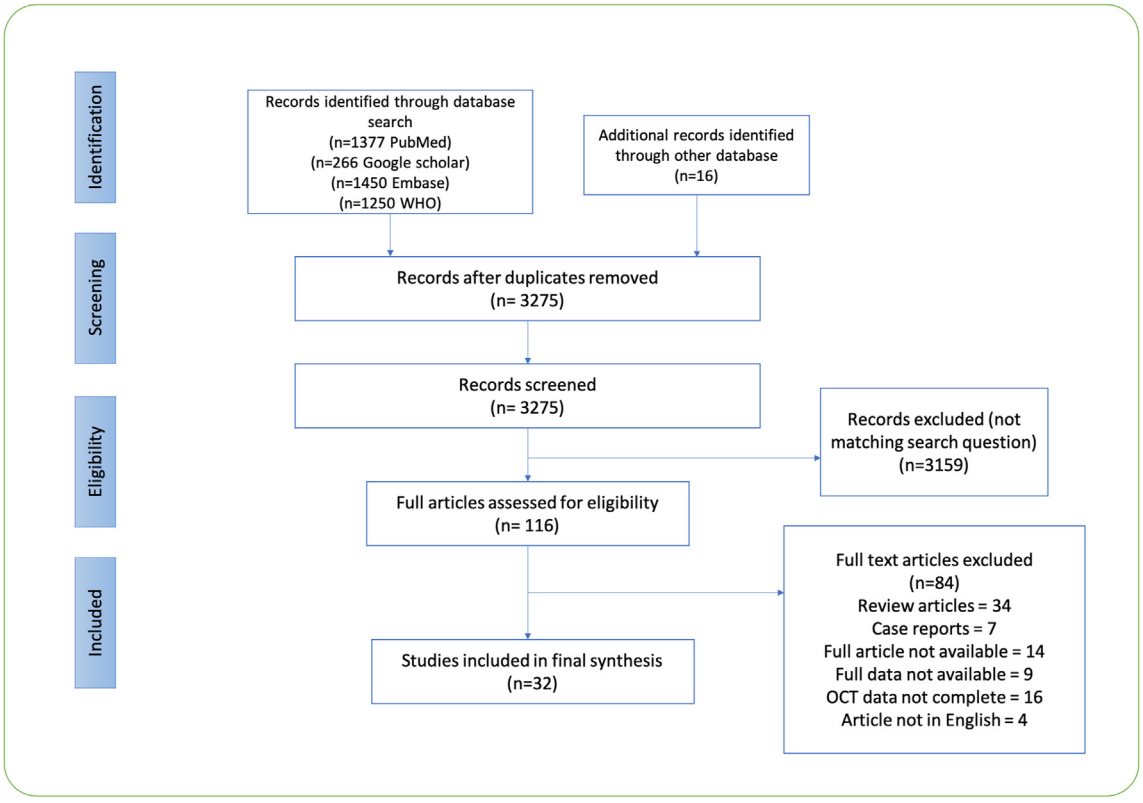

Fig. 1. PRISMA flowchart for selected studies.

TCFA (48.1% vs 50.6%, respectively; p = 0.411). OCT revealed higher prevalence of lipid plaques in STEMI patients as compared to NSTEMI patients (73.4% vs 56.6%, respectively), signifying presence of high-risk substrate in this group. Expectedly, the thrombus burden was significantly higher in STEMI patients (91.1% vs 32.5%, respectively; p < 0.001). However, no predilection for red or white thrombus was noted in either groups. Macrophages were more commonly seen in STEMI patients (70.6% vs 49.4%, respectively; p < 0.001), indicating presence of intra-plaque inflammation. Plaque rupture was

Table 1. Baseline characteristics of study population.

Baseline characteristics	Number of studies (n = 32)	N = 7526
Age in years	32	62.4 ± 4.65
Female gender	32	1738/7526 (23.1)
Diabetes	31	2302/7490 (30.7)
Hypertension	31	4585/7490 (61.2)
Dyslipidaemia	29	4648/7369 (63.1)
Smoker	28	3589/6449 (55.6)
Previous MI	11	287/4066 (7)
Previous PCI	12	409/4428 (9.2)
Clinical presentation		
Unstable angina	09	373/1971 (18.9)
NSTEMI	18	2144/4581 (46.8)
STEMI	26	4777/7162 (66.6)
LDL in mg/dl	18	114.4 ± 9.3
LV ejection fraction	10	54.5 ± 5.3

All values are depicted as percentages except age, LDL and ejection fraction which are demonstrated as mean(SD).LDL – low density lipoprotein, MI – myocardial infarction, NSTEMI – Non ST elevation myocardial infarction, STEMI – ST elevation myocardial infarction, PCI – percutaneous coronary intervention.

Table 2. Angiographic characteristics of study population.

Angiographic characteristics	Number of studies (n = 32)	N = 7526
Culprit vessel		
LAD	28	3300/6494 (50.8)
LCX	28	938/6494 (14.4)
RCA	28	2353/6494 (36.2)
Lesion location		
Proximal	2	329/867 (37.9)
Mid	2	388/867 (44.7)
Distal	2	150/867 (17.3)
Lesion length, mm	13	14.1 ± 2.37
MLD, mm	16	.55 ± .37
RVD, mm	16	2.95 ± .26
Diameter stenosis, %	18	79.6 ± 12.7
Complex lesion (AHA type B/C)	8	1926/3274 (58.5)

Values are depicted as percentages or mean (SD) [lesion length, MLD, RVD, Diameter stenosis]. MLD – minimum lumen diameter, RVD – reference vessel diameter.

Table 3. OCT characteristics of study population. TCFA – thin cap fibroatheroma, MLA – minimum lumen area, RVA – reference vessel area.

OCT characteristics	Number of studies (n = 32)	N = 7526
Plaque cap thickness, μm	26	84.2 ± 23.4
Lipid arc, degrees	20	228 ± 70.8
Lipid core length, mm	12	11.1 ± 3.4
TCFA	28	3119/6939 (44.9)
Lipid rich plaques	27	4534/6333 (71.6)
Thrombus	27	3963/5264 (75.2)
Red thrombus	11	553/1730 (31.9)
White thrombus	9	488/1322 (36.9)
Macrophages	14	3375/5184 (65.1)
Microcalcification	14	1712/4684 (36.5)
Microchannels	12	1059/3057 (34.6)
MLA, mm^2	19	1.76 ± .5
RVA, mm^2	7	6.57 ± .95
Area stenosis, %	6	73.2 ± 5.8
Plaque rupture	32	4079/7526 (54.2)
Plaque erosion	23	2038/5867 (34.7)

the most common lesion morphology noted in both STEMI and NSTEMI patients. STEMi patients had a higher incidence of plaque rupture as compared to NSTEMI patients (61.3% vs 29.3%, respectively). The incidence of plaque erosion was overall low in the study cohort. However, erosions were more commonly noted in STEMI patients (34.4% vs 13.2%, respectively).

4. Discussion

To our knowledge, this is the largest systematic review of coronary culprit lesion analysis using OCT in patients presenting with acute coronary syndrome. This systematic review included 32 studies, comprising a total of 7526 patients. Females comprised around one-fourth of the study population and STEMI was the most common clinical presentation. NSTEMI patients had a higher prevalence of diabetes as compared to their STEMI counterparts. On OCT analysis, both groups had similar plaque cap thickness and prevalence of TCFA. However, the prevalence of lipid plaques, thrombus, and macrophages were higher in STEMI group. Plaque rupture was the most common lesion morphology noted in both groups.

The main aim of the study was to analyse the difference in lesion characteristics between STEMI and NSTEMI patients using in-vivo OCT analysis. NSTEMI patients are commonly noted to have higher prevalence of multivessel disease and associated with a higher mortality as compared to STEMI patients [40]. Consequently, NSTEMI patients are noted to have higher comorbidities in the form of chronic kidney disease (CKD) and inflammatory disorders like rheumatoid arthritis in addition to conventional coronary artery disease risk factors. Diabetes is one of the most important CAD risk factors which promotes accelerated atherosclerosis [41]. The presence of diabetes predisposes to the development of multivessel disease and these patients commonly present with NSTEMI. Smoking predisposes to a prothrombotic milleu and poses a high risk for ACS events. Smokers are more likely to present with STEMI [42]. The prevalance of diabetes was higher in NSTEMI patients in our review as well, indicating presence of diffuse atherosclerosis in this group.

Table 4. Comparison of baseline and lesion characteristics between STEMI and NSTEMI patients. Values are depicted as mean ± SD or percentages.

	STEMI (n = 2867)	NSTEMI (n = 286)	p-value
Age in years	62.8 ± 4.4	62.3 ± 5.3	.427
Females	718/2867 (25)	80/286 (27.9)	.36
Diabetes	825/2867 (28.7)	99/250 (39.6)	.0003
Hypertension	1625/2867 (56.6)	153/250 (61.2)	.166
Dyslipidemia	1962/2867 (68.4)	105/156 (67.3)	.768
Smoking	1405/2867 (49)	155/250 (62)	.00008
Culprit vessel			<.0001
LAD	1005/1958 (51.3)	141/214 (65.8)	
LCX	230/1958 (11.7)	19/214 (8.8)	
RCA	843/1958 (43.1)	54/214 (25.2)	
OCT characteristics			
Minimum fibrous cap thickness, μm	84.8 ± 24.6	79.5 ± 21.9	.390
TCFA	1258/2613 (48.1)	145/286 (50.6)	.411
Lipid arc, in degrees	255.2 ± 82.1	161.25 ± 51.9	.17
Lipid plaques	1860/2531 (73.4)	162/286 (56.6)	<.0001
Thrombus	1683/1846 (91.1)	93/286 (32.5)	<.001
Macrophages	1618/2291 (70.6)	88/178 (49.4)	<.0001
Plaque rupture	1758/2867 (61.3)	84/286 (29.3)	.007
Plaque erosion	872/2531 (34.4)[a]	22/166 (13.2)[a]	

[a] 2 studies did not provide OCT data regarding plaque erosion in patients with STEMI and NSTEMI.

OCT provides the unique ability of analysing plaque morphology in vivo as well as determine plaque cap thickness. This helps us to determine vulnerable plaques which can lead to acute events in the future. STEMI patients are known to have higher degree of plaque inflammation and vulnerable plaques. Vulnerable plaques are identified on OCT as those having a high lipid burden and thin fibrous cap (TCFA). TCFA are usually more commonly seen in patients with STEMI as compared to NSTEMI(16). Patients with TCFA are prone to develop plaque rupture as these plaques are associated with positive remodeling and higher inflammatory markers [43,44]. In our current analysis, NSTEMI patients were noted to have similar prevalence of TCFA as compared to STEMI patients. However, lipid plaques were more commonly seen in STEMI group, signifying presence of higher degree of atherosclerosis in these patients. The similar prevalence of TCFA amongst NSTEMI and STEMI patients in the current review signifies that NSTEMI patients also have a inflammatory milleu similar to STEMI patients which predisposes to development of vulnerable plaques. This can be attributed to presence of associated risk factors like chronic kidney disease and systemic inflammatory disorders in NSTEMI patients. Consequently, identification of vulnerable plaques using in-vivo imaging becomes important in both subset of patients.

The development of acute coronary syndrome is linked to heightened plaque inflammation, predisposing to formation of intracoronary thrombus. OCT has the unique ability of identifying luminal thrombus and differentiating between red and white thrombus. Disruption of fibrous cap leads to exposure of thrombogenic materials, causing activation of coagulation system and complete vessel closure. Hence, plaque rupture is usually associated with red thrombus and is seen frequently in STEMI patients. Presence of intra-plaque macrophages on OCT is a marker of inflammation and predisposes to fibrous cap disruption [45]. Hence, these findings are more commonly noted in patients with STEMI. Plaque erosion, on the other hand, is mainly caused due to endothelial denudation which can cause platelet activation. Presence of intact fibrous cap denotes lower degree of plaque inflammation and reduced risk of adverse events in future.

While we classify patients according to their clinical presentation, it is important to note that culprit lesion morphology varies widely amongst patients. The culprit lesion morphology is determined by the presence of risk factors and the state of inflammation. Thus, NSTEMI patients with significant risk factor burden are equally prone to develop plaque rupture compared to STEMI patients. This explains the higher incidence of plaque rupture in both STEMI and NSTEMI cohort. Similarly, identification of plaque erosion in ACS patients is also important because it helps in determining long term prognosis. Niccoli et al. showed that patients with plaque rupture had a higher incidence of adverse events at a follow up period of 3 years as compared to those with intact fibrous cap [27]. The overall low incidence of plaque erosion in the current study could be attributed to the fact that it is difficult to diagnose on OCT. Therefore, majority of imaging studies in ACS patients predominantly report about plaque rupture only, leaving out plaque erosion.

Recent studies have made further advances in understanding mechanisms and outcomes of plaque erosion. As these patients have low risk plaque morphology denoted by an intact fibrous cap, it was explored whether such patients could be treated with only anti-platelet therapy. The EROSION study showed that ACS patients with plaque erosion, who were managed with anti-platelets, had reduction in thrombus volume and the majority remained free from major adverse cardiovascular events at the end of one year [46]. Use of OCT-guidance during primary PCI in STEMI patients was associated with lower rates of stent implantation in EROSION III study. Majority of patients with plaque erosion did not undergo stent implantation and had similar event rates at 1 year as compared to the stented cohort [47]. These findings underline the importance of OCT imaging in ACS patients to identify the subgroup who may not require stenting. This should further encourage the use of OCT in ACS patients.

The current review has its limitations. Firstly, OCT data regarding plaque erosion was not available in all studies. This prevents us from better understanding of this subset of patients. Second, individual data on various subsets of patients was not available in the majority of studies. This precluded us from doing a detailed analysis of each subset of patients. Third, the time from symptom onset to OCT analysis was not mentioned in any of the studies. This may lead to bias in the results obtained.

5. Conclusion

Plaque rupture is the predominant culprit lesion noted in both STEMI and NSTEMI patients, despite differences in baseline characteristics. Identification of culprit lesion morphology using OCT can guide management in ACS patients and avoid stenting when deemed not necessary.

Author contributions

AM, VO. Literature review: AM, VO. Acquisition of data: AM, VO. Analysis and interpretation of data: AM, VO, HS, BS. Research investigation and analysis: AM, VO, HS, BS. Data collection: AM, VO. Drafting of manuscript: AM, VO, BS, SG. Revising and editing the manuscript critically for important intellectual contents: HS, BS, SG, AKV. Data preparation and presentation: BS, SG,AKV.

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Nil.

Supplementary table.

Supplementary Table 1. Selected studies for analysis.

SL No.	Year of study	Name of Author	Country	Total patients	Age	Female Sex
1.	2018	Dai et al. [19]	China	773	57.7	215
2.	2013	Jia H et al. [31]	USA	126	59.2	26
3.	2016	Bogale et al. [34]	Canada	36	60.5	13
4.	2019	Yamamoto et al. [38]	Multi-centre	1241	64	262
5.	2015	Higuma et al. [9]	Japan	111	70	22
6.	2019	Sheng Z et al. [21]	China	279	57.9	50
7.	2018	Iannaccone et al. [39]	Multi-centre	209	60.1	40
8.	2017	Chandran et al. [32]	USA	40	62.5	14
9.	2018	ElFaramawy A et al. [35]	Egypt	27	48	3
10.	2015	Refaat et al. [26]	Italy	107	64	36
11.	2015	Niccoli et al. [27]	Italy	139	64.3	37
12.	2016	Dai et al. [22]	China	206	58.2	46
13.	2020	Yonetsu et al. [10]	Japan	409	66.2	90
14.	2017	Sun et al. [23]	China	211	57.9	49
15.	2017	Satogami et al. [11]	Japan	103	64.5	24
16.	2018	Kobayashi et al. [12]	Japan	333	67.3	59
17.	2016	Kobayashi et al. [13]	Japan	120	64	22
18.	2019	Wang et al. [24]	China	94	55.8	23
19.	2019	Roule et al. [36]	France	27	59.9	3
20.	2017	Sakaguchi et al. [14]	Japan	84	68	27
21.	2017	Yonetsu et al. [15]	Japan	442	65.4	87
22.	2018	Sugiyama et al. [33]	USA	322	59.8	76
23.	2011	Ino et al. [16]	Japan	89	64.5	18
24.	2019	Hoshino et al. [17]	Japan	510	67.3	107
25.	2013	Kato et al. [18]	Japan	75	67.9	11
26.	2015	Wang et al. [28]	Italy	62	66	14
27.	2020	Zhou et al. [20]	China	277	57.6	126
28.	2020	Araki et al. [37]	Multi-centre	648	65.5	127
29.	2016	Niccoli et al. [29]	Italy	51	65	22
30.	2013	Ozaki et al. [8]	Japan	261	66.5	62
31.	2007	Chia et al. [30]	USA	42	58	9
32.	2013	Niccoli et al. [25]	Italy	72	65	17

Supplementary Table 2. Newcastle Ottawa scale for rating of quality of selected studies in the systematic review.

Study	Year	Number of stars			
		Selection[a]	Comparability[b]	Exposure[c]	Overall
Dai et al. [19]	2018	3	1	2	6/9
Jia H et al. [31]	2013	3	2	2	7/9
Bogale et al. [34]	2016	2	1	3	6/9
Yamamoto et al. [38]	2019	3	2	3	8/9
Higuma et al. [9]	2015	3	2	2	7/9
Sheng Z et al. [21]	2019	2	2	2	6/9
Iannaccone et al. [39]	2018	4	2	2	8/9
Chandran et al. [32]	2017	4	1	2	7/9
ElFaramawy A et al. [35]	2018	3	1	2	6/9
Refaat et al. [26]	2015	3	2	2	7/9
Niccoli et al. [27]	2015	3	2	2	7/9
Dai et al. [22]	2016	2	2	2	6/9
Yonetsu et al. [10]	2020	3	2	2	7/9
Sun et al. [23]	2017	3	1	3	7/9
Satogami et al. [11]	2017	4	1	2	7/9
Kobayashi et al. [12]	2018	3	2	2	7/9
Kobayashi et al. [13]	2016	4	1	3	8/9
Wang et al. [24]	2019	3	2	2	7/9
Roule et al. [36]	2019	3	2	3	8/9
Sakaguchi et al. [14]	2017	3	1	2	6/9
Yonetsu et al. [15]	2017	3	2	3	8/9
Sugiyama et al. [33]	2018	3	2	2	7/9
Ino et al. [16]	2011	3	1	2	6/9
Hoshino et al. [17]	2019	3	1	3	7/9
Kato et al. [18]	2013	2	1	2	5/9
Wang et al. [28]	2015	3	1	2	6/9
Zhou et al. [20]	2020	3	1	2	6/9
Araki et al. [37]	2020	3	2	2	7/9
Niccoli et al. [29]	2016	4	2	2	8/9
Ozaki et al. [8]	2013	3	1	3	7/9
Chia et al. [30]	2007	3	1	2	6/9
Niccoli et al. [25]	2013	3	2	2	7/9

[a] Maximum 4 stars.
[b] Maximum 2 stars.
[c] Maximum 3 stars.

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