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Original Article

Impact of Smoking Status on Risk Factors, Treatment and Clinical Outcomes in Acute Coronary Syndrome in Taiwan

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SUMMARY

Background: Smoking is a major public health problem and is a well-established risk factor for acute coronary syndrome (ACS). However, the patient characteristics and outcomes for smokers with ACS remained unclear in Taiwan.
Methods: ACS patients with known smoking status were analyzed from a Taiwan nationwide registry. We compared the current smokers and non-current smokers in terms of baseline demographics, clinical presentation, risk factors, medical treatment, and one-year outcomes.
Results: Of the 3128 patients, 1313 (42%) were current smokers. At baseline, current smokers were younger (56.9 ± 12.6 vs. 67.5 ± 12.4 years, $p < 0.0001$), more obese (BMI: 25.9 ± 3.9 vs. 25.1 ± 3.8 kg/m^2, $p < 0.0001$), and male predominant (95.4% vs. 65.2%, $p < 0.0001$), but had a lower prevalence of hypertension (52.4% vs. 72.2%, $p < 0.0001$), diabetes (27.7% vs. 41.9%, $p < 0.0001$), and dyslipidemia (37.0% vs. 40.8%, $p = 0.03$). Additionally, the younger the age the lower the prevalence of hypertension and diabetes, but the higher prevalence to be current smokers (all $p < 0.0001$). During hospitalization, antiplatelets, angiotensin converting enzyme inhibitors/angiotensin receptor blockers, and statins were more prescribed in smokers (all $p < 0.05$). Over the course of a one year follow-up, cumulative all-cause mortality, recurrent myocardial infarction (re-infarction), and stroke (5.5% vs. 10.1%, $p < 0.0001$) were lower in current smokers.
Conclusion: Smoking is associated with nearly 10 years earlier development of ACS in Taiwan. Younger ACS group had lower percentages of traditional cardiovascular risk factors but a higher percentage to be current smokers. Although smokers had better one-year outcomes, it may be attributed to younger age, fewer comorbidity, and more aggressive medical treatment.

1. Introduction

Smoking is a major health problem worldwide. On one hand, smoking is a well-established risk factor for cardiovascular disease.[1–3] On the other hand, several studies all over the world showed a lower mortality rate of smokers following acute coronary syndrome (ACS), called "smoker's paradox", which is largely attributable to the younger age and having fewer other cardiovascular risk factors of ACS smokers compared with nonsmokers.[4–6]

Although adolescent smoking is reducing due to Taiwan government's effort by implementing the Tobacco Hazards Prevention Act (THPA) in 1997, the Health and Welfare Surcharge (HAWS) in 2002, and the Tobacco Hazards Prevention Act Amendment (THPAA) in 2009,[7,8] there were still 13.0% smokers in Taiwan in 2018.[9] Apart from other traditional ACS risk factors, smoking is a strong controllable risk factor that needs more efforts to modify. However, there was little information about how smoking influences ACS in Taiwan.

The main purpose of this study was to document the baseline demographics, clinical presentation, risk factors, medical treatment, and outcomes between current smokers and non-current smokers in Taiwan using the data from The Taiwan ACS Full Spectrum registry.[10]

2. Materials and methods

2.1. Study design

The Taiwan ACS Full Spectrum registry, conducted by Taiwan Society of Cardiology, is a prospective, nationwide, multicenter study to assess real-world clinical practices and outcomes for patients with ACS in Taiwan. The study period started from the time of admission and continued for one year post-discharge. The study applied an observational design and recruited patients ≥ 20 years of age between October 2008 and January 2010 who presented with symptoms of ACS, and were admitted within 24 hours at any of the 39 participating hospitals (20 medical centers and 19 regional hospitals) in Taiwan. Each hospital enrolled 50–200 consecutive eligible patients.[10]

In brief, ACS was defined by a wide range of symptoms including ST elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI) and unstable angina (UA) based on electrocardiogram findings and cardiac enzyme change. Baseline characters,

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including age, gender, body mass index (BMI), blood pressure, serum creatinine, lipid profile, left ventricular ejection fraction, were obtained during index hospitalization. Smoking status and past medical history was determined by previous medical records and/or self-reporting. Hospital therapies were medication prescribed at discharge.

A total of 3183 patients were enrolled. We excluded patients without any record of smoking status, patients lost to follow-ups after discharge, patients whose data were incorrect or not sufficiently clear, and patients who were misdiagnosed on admission if their discharge diagnosis excluded ACS. Overall, 3128 (98.3%) patients were analyzed in this study.

All 3128 patients were divided into 2 groups, current smokers and non-current smokers, the latter group included non plus former smokers. Former smokers were defined as previous smokers that had quit smoking cigarettes > 1 month prior to admission. We compared the 2 groups in term of baseline demographics, clinical presentation, risk factors, medication, and outcomes over the following 12 months.

The primary endpoint was a composite outcome that included all-cause mortality, recurrent myocardial infarction (re-infarction), and stroke in 1 year. The secondary endpoint consisted of the all-cause mortality, cardiovascular death, re-infarction, stroke, and re-vascularization in 1 year.

The study was carried out in accordance with the local regulatory guidelines and international guidelines for good epidemiological practices. This study was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Review Board of Mackay Memorial Hospital. Patient-related records were hided and de-identified prior to analysis.

2.2. Statistical analysis

Continuous variables are presented as means ± standard deviation (SD); categorical variables are presented as absolute numbers and percentages. The independent t-test, two-sample t-test, and One-way ANOVA was used for comparison of continuous variables. For categorical variables, we applied Pearson's chi-square test. A p value of less than 0.05 was considered significant for all factors. The one-year follow-up event analysis is presented with Kaplan-Meier survival curves and log rank test. All analyses were conducted using SPSS software, version 20.0 (SPSS Inc., Chicago, IL, USA).

3. Results

3.1. Clinical characteristics

Table 1 lists the baseline characteristics of all 3128 patients based on smoking status. The current smokers (n = 1313) were younger than the non-current smokers (56.9 ± 12.6 vs. 67.5 ± 12.4 years; p < 0.0001). Males were by far predominant in the current smokers and higher in portion compared to non-current smokers (95.4% vs. 65.2%, p < 0.0001). The current smokers, compared to the non-current smokers, had fewer cardiovascular risk factors and co-morbidities, including hypertension, diabetes mellitus, dyslipidemia, and history of stroke, heart failure, coronary artery disease (CAD), and peripheral artery disease (PAD; see Table 1).

Regarding the medication during hospitalization (Table 2), higher proportions of the current smokers took aspirin, P2Y12 inhibitors, ACEI/ARB, and statin therapy (all p < 0.05). The use of beta blockers and nitrates was similar between the two groups.

3.2. Distribution of risk factors and comorbidities according to age

We further separated the patients into different groups ac-

Table 1
Baseline characteristics of all patients, current smokers and non-current smokers in Taiwan full spectrum ACS registry.

	All (N = 3128)	Current smoker (N = 1313)	Non-current smoker (N = 1815)	p value
Age (years)	63.0 ± 13.5	56.9 ± 12.6	67.5 ± 12.4	< 0.0001
Male (%)	2436 (77.9)	1253 (95.4)	1183 (65.2)	< 0.0001
BMI (kg/m^2)	25.4 ± 3.9	25.9 ± 3.9	25.1 ± 3.8	< 0.0001
SBP (mmHg)	139.2 ± 32.7	136.8 ± 32.4	141.0 ± 32.8	0.0004
DBP (mmHg)	81.5 ± 20.8	82.6 ± 20.7	80.8 ± 20.9	0.0159
Heart rate (bpm)	82.2 ± 22.4	79.8 ± 21.0	83.9 ± 23.3	< 0.0001
Height (cm)	163.9 ± 7.9	167.0 ± 6.4	161.7 ± 8.1	< 0.0001
Weight (kg)	68.4 ± 12.9	72.1 ± 12.6	65.7 ± 12.4	< 0.0001
Waist circumference (cm)	90.4 ± 9.5	90.5 ± 9.3	90.3 ± 9.8	0.7248
Serum creatinine (mg/dl)	1.7 ± 2.2	1.4 ± 1.5	1.9 ± 2.5	< 0.0001
Total cholesterol	178.8 ± 46.3	185.9 ± 46.9	173.4 ± 45.2	< 0.0001
LDL-C	112.6 ± 38.0	118.7 ± 38.7	107.8 ± 36.8	< 0.0001
HDL-C	39.6 ± 23.6	37.6 ± 13.5	41.2 ± 29.1	< 0.0001
Triglycerides	142.7 ± 107.1	157.1 ± 120.8	131.6 ± 93.8	< 0.0001
LV ejection fraction	54.4 ± 13.0	54.2 ± 12.4	54.5 ± 13.5	0.6630
ESRD (%)	123 (3.9)	23 (1.8)	100 (5.5)	< 0.0001
Hypertension (%)	1979 (63.9)	679 (52.4)	1300 (72.2)	< 0.0001
DM (%)	1118 (36)	361 (27.7)	757 (41.9)	< 0.0001
Dyslipidemia (%)	1215 (39.2)	479 (37.0)	736 (40.8)	0.0308
Prior stroke (%)	277 (8.9)	75 (5.7)	202 (11.1)	< 0.0001
Previous heart failure (%)	171 (5.5)	46 (3.5)	125 (6.9)	< 0.0001
Prior CAD (%)	771 (24.6)	232 (17.7)	539 (29.7)	< 0.0001
Prior PAD (%)	69 (2.2)	17 (1.3)	52 (2.9)	0.0032
STEMI (%)	1641 (52.5)	832 (63.4)	809 (44.6)	< 0.0001
NSTEMI (%)	1056 (33.8)	360 (27.4)	696 (38.3)	< 0.0001
UA (%)	392 (12.5)	115 (8.8)	277 (15.3)	< 0.0001

BMI, body mass index; bpm, beats per minute; LDL-C, low density lipoprotein-cholesterol; HDL-C, high density lipoprotein-cholesterol; LV, left ventricular; ESRD, end-stage renal disease; DM, diabetes mellitus; CAD, coronary artery disease; PAD, peripheral artery disease; STEMI, ST elevation myocardial infarction; NSTEMI, non-ST elevation myocardial infarction; UA, unstable angina; Data are mean ± SD, unless mentioned else.

cording to age (< 35, 35–44, 45–54, 55–64, and ≥ 65 years) (Supplementary Table 1), and found the higher the age, the higher the prevalence of traditional atherosclerotic risk factors but the lower the prevalence of current smokers (Figure 1). In addition, non-STEMI and unstable angina were more prevalent as the age increased (Supplementary Table 1).

3.3. Hospital stay and one-year survival

Current smokers had shorter hospital stay compared to non-current smokers (6.8 ± 6.0 vs. 9.0 ± 9.3 days, p < 0.0001). The clinical events within 1 year post the indexed myocardial infarction, including all-cause mortality, re-infarction, and stroke, were analyzed (Table 3). Overall, 244 (8.1%) patients suffered from all-cause mortality, re-infarction, and stroke. Comparing the 1 year outcome of current smokers and non-current smokers, the current smokers had significantly lower incidents of a composite endpoint of all-cause mortality, stroke, and re-infarction (5.5% vs. 10.1%, p < 0.0001), as well as all-cause mortality (2.1% vs. 6.2%, p < 0.0001) and cardiovascular death (0.3% vs. 2.2%, p < 0.0001), but not stroke and re-infarction (Figure 2).

4. Discussion

Smoking is one major risk factor for atherosclerotic cardiovascular disease (ASCVD).[1–3] Consistent with this, in our analysis, in

Table 2
Hospital therapies of all patients, current smokers and non-current smokers in Taiwan full spectrum ACS registry.

	All (N = 3128)	Current smoker (N = 1313)	Non-current smoker (N = 1815)	p value
Aspirin	2877 (92.0)	1245 (94.8)	1632 (89.9)	< 0.0001
P2Y12-inhibitor	2947 (94.2)	1265 (96.3)	1682 (92.7)	< 0.0001
Beta blocker	1434 (45.8)	624 (47.5)	810 (44.6)	0.1086
ACEI/ARB	1848 (59.1)	805 (61.3)	1043 (57.5)	0.0309
Nitrate	2098 (67.1)	863 (65.7)	1235 (68.0)	0.1736
Statin	1540 (48.4)	700 (53.3)	818 (45.1)	< 0.0001
Other lipid-lowering agent	80 (2.6)	38 (2.9)	42 (2.3)	0.3105
CCB	344 (11.0)	93 (7.1)	251 (13.8)	< 0.0001
Digoxin	64 (2.0)	13 (1.0)	51 (2.8)	0.0004
Diuretics	642 (20.5)	194 (14.8)	448 (24.7)	< 0.0001

ACEI, angiotensin converting enzyme inhibitors; ARB, angiotensin II receptor blockers; CCB, calcium channel blockers; Data are mean (%)

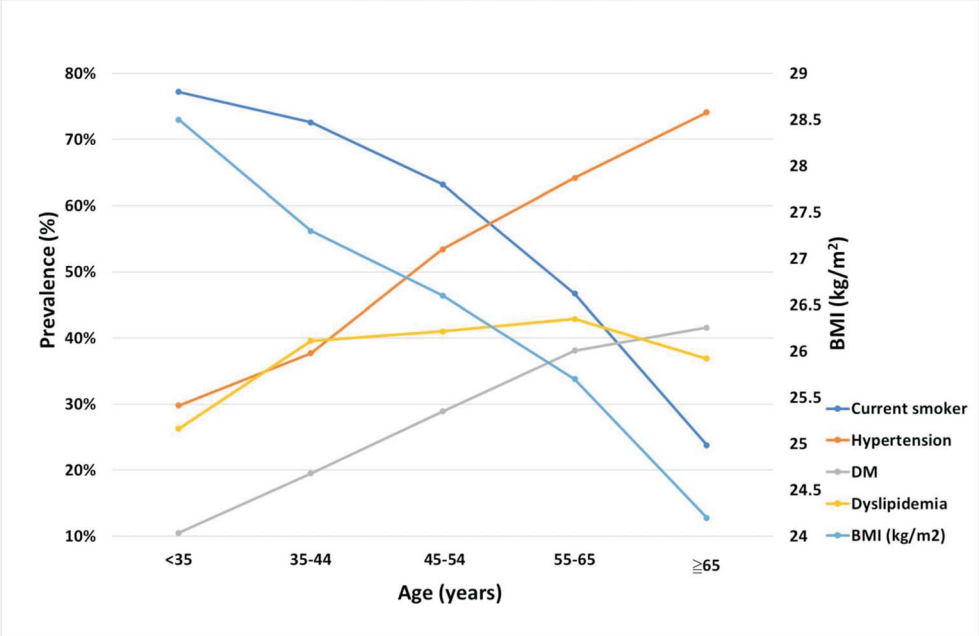

Figure 1. Comparison of prevalence of current smoker, hypertension, diabetes (DM), dyslipidemia and body mass index (BMI) between different age groups.

Table 3
One year outcome of all patients, current smokers and non-current smokers in Taiwan full spectrum ACS registry.

	All (N = 2998)	Current smoker (N = 1283)	Non-current smoker (N = 1715)	p value
All-cause mortality, Re-MI, stroke	244 (8.1)	70 (5.5)	174 (10.1)	< 0.0001
All-cause mortality	133 (4.4)	27 (2.1)	106 (6.2)	< 0.0001
Cardiovascular death	42 (1.4)	4 (0.3)	38 (2.2)	< 0.0001
Re-hospitalization	1126 (37.6)	460 (35.9)	666 (38.8)	0.1033
Stroke	36 (1.2)	11 (0.9)	25 (1.5)	0.1856
Re-MI	97 (3.2)	38 (3.0)	59 (3.4)	0.5299
Recurrent ischemia requiring revascularization	178 (5.9)	87 (6.8)	91 (5.3)	0.1068

Abbreviations are as in Table 1. Re-MI, recurrent myocardial infarction. Data are mean (%).

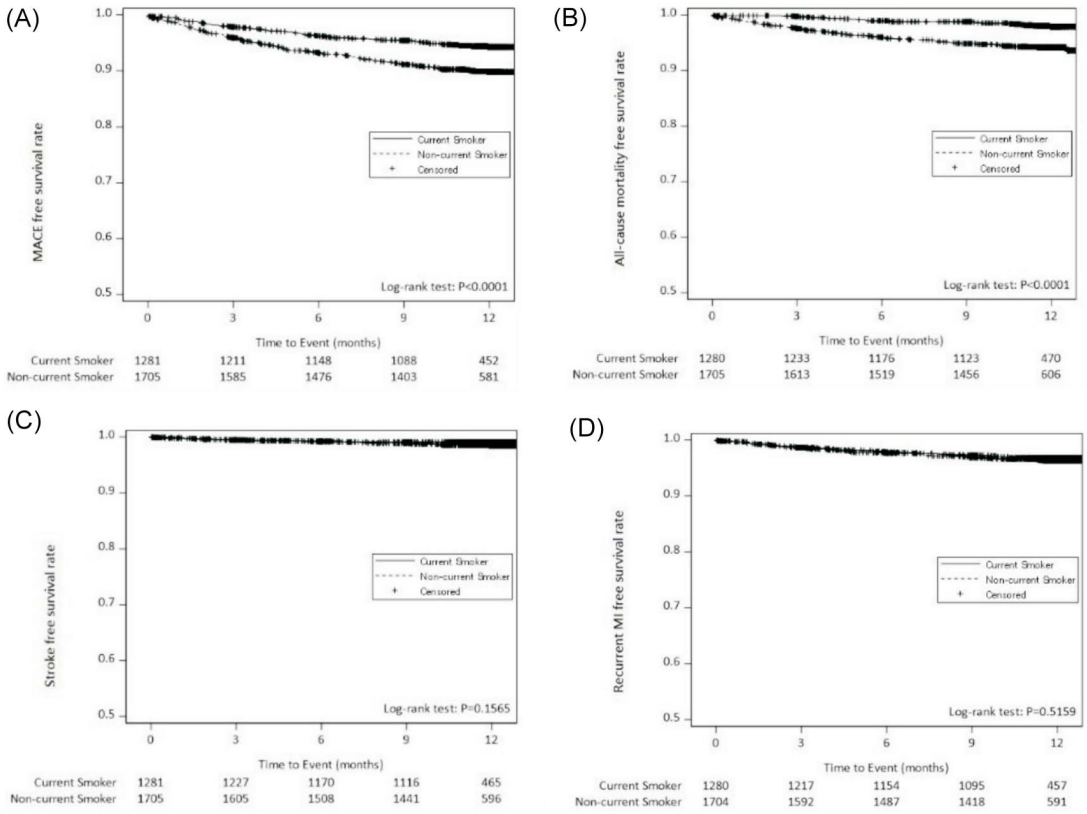

Figure 2. Kaplan-Meier curves of one year outcomes of primary cardiovascular events, consisting of all-cause mortality, stroke, and recurrent myocardial infarction (A), all-cause mortality (B), stroke (C), and recurrent myocardial infarction (D).

Taiwan, current-smokers develop ACS around 10 years earlier than non-current smokers. The estimated prevalence of current smokers among ACS patients was around 20–30% in a previous report.[11] However, in our Taiwanese ACS Full Spectrum registry, the prevalence of current smokers was 42%, much higher than others all over the world. It implies that smoking plays a tremendous role in Taiwan ACS patients.

Like other studies,[4,12] among these Taiwanese ACS patients, current smokers were younger and had fewer traditional cardiovascular risk factors, such as hypertension, diabetes mellitus, dyslipidemia, and a history of cardiovascular events including stroke, heart failure, CAD, and PAD than non-current smokers. This distribution provides a hint toward the harmful effects of smoking. When we further grouped patients according to age (< 35, 35–44, 45–54, 55–64, ≥ 65 years), this phenomenon are more prominent. This finding is consistent with a previous study in Taiwan that smoking is a much stronger risk factor in the younger population.[13]

Regarding the medical treatment of the ACS patients, we observed an age-dependent phenomenon. Although there was a higher prevalence of traditional cardiovascular risk factors in the non-current-smoker group, the guideline-directed medication achievement rate was much lower than the current smoker group. A possible explanation is that there is a relatively higher percentage of medication-related adverse effects in the aged people who are more in the non-smoker group.

Several previous studies have already reported that smokers had favorable outcomes of AMI,[4–6] acute ischemic stroke,[14] acute heart failure,[15] and cardiac arrest,[16] which was called “smoker’s paradox”. These phenomena were largely attributed to a cumulative effect of younger age,[5] fewer comorbidities, lesser extent of CAD, and more aggressive treatment in smokers. Alternatively, pathophysiological differences between smokers and nonsmokers with acute MI have also been postulated as a basis for this paradox, including a greater thrombus burden in smokers, leading to greater ef?cacy of thrombolytic therapy[17–19] and greater responsiveness to antiplatelet therapies.[20–23] Whether a true biochemical basis exists for the smoker’s paradox remains inconclusive.

In the present study, we also found such a phenomenon. The current smokers had a significantly shorter hospital stay and lower incidence of primary composite endpoint, especially all-cause mortality and cardiovascular death, but not stroke and re-infarction, compared to non-current smokers. However, we found that current smokers were younger, having fewer comorbidities, used more aggressive medication compared to non-current smokers. These findings could also possibly explain the smoker’s paradox in our patients.

The findings of the present study highlighted the importance of comprehensive life style modification, including smoking cessation in Taiwan for ACS prevention, in particular for young people, given that current smokers accounted for ACS was extremely high as more than three quarters of the ACS patients were aged below 35 years. In addition, other major risk factors of ASCVD, such as hypertension, diabetes and dyslipidemia were also prevalent in our young patients with ACS. For example, the prevalence of dyslipidemia in the ACS patients below 35 years old exceeded those of the general population and even the elderly in Taiwan.[24,25]

4.1. Limitations

This was an observational study based on data from the Taiwan ACS Full Spectrum registry. In addition, several details such as smoking amount, duration, and quitting or not after the ACS were not available due to the original study design. Therefore, we cannot clearly show the relationship between smoking and one-year outcomes after ACS.

In addition, the initial ACS treatment strategy was not recorded in the original study design. Thus, we cannot know the influence on outcomes. Lastly, our study is limited due to its short follow up duration. Certain long-term effects of smoking will not be observed in such a short period.

5. Conclusion

This study demonstrated that, in Taiwan, smoking is a major risk factor in ACS patients and is especially so in younger patients. Smoking is associated with a nearly 10 year earlier development of CAD presented with ACS. Although smokers had better one-year outcomes regarding the combined results for all-cause mortality, reinfarction and stroke, it may be attributed to young age, fewer comorbidity, and more aggressive medical treatment.

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Declaration of any potential financial and non-financial conflicts of interest

No potential financial and non-financial conflicts of interest.

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Supplement

Supplementary Table 1
Distribution of risk factors and current smoker prevalence by different age groups.

	< 35 years (N = 57)	35–44years (N = 237)	45–54 years (N = 627)	55–64 years (N = 768)	≥ 65 years (N = 1439)	p value
Current smoker	44 (77.2)	172 (72.6)	396 (63.2)	359 (46.7)	342 (23.8)	< 0.0001
Male	54 (94.7)	224 (94.5)	558 (89.0)	637 (82.9)	963 (66.9)	< 00001
BMI (kg/m^2)	28.5 ± 6.1	27.3 ± 4.0	26.6 ± 3.8	25.7 ± 3.4	24.2 ± 3.6	< 0.0001
ESRD	0 (0.0)	6 (2.5)	16 (2.5)	43 (5.6)	58 (4.0)	0.0145
Hypertension	17 (29.8)	89 (37.7)	332 (53.4)	487 (64.2)	1054 (74.1)	< 0.0001
DM	6 (10.5)	46 (19.5)	180 (28.9)	291 (38.1)	595 (41.6)	< 0.0001
Dyslipidemia	15 (26.3)	93 (39.6)	254 (41.0)	327 (42.9)	526 (36.9)	0.0146
STEMI	39 (68.4)	156 (65.8)	383 (61.1)	412 (53.6)	651 (45.2)	< 0.0001
NSTEMI	10 (17.5)	58 (24.5)	172 (27.4)	246 (32.0)	570 (39.6)	< 0.0001
UA	5 (8.8)	21 (8.9)	70 (11.2)	102 (13.3)	194 (13.5)	0.1781

Abbreviations are as in Table 1. Data are mean (%) except BMI, which is mean ± SD.