Ventricular Arrhythmias in Patients with Spontaneous Coronary Artery Dissection

Findings from the Gulf Spontaneous Coronary Artery Dissection (Gulf SCAD) Registry

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Background: Spontaneous coronary artery dissection (SCAD) is an increasingly recognized cause of acute coronary syndrome in young women, with a wide clinical spectrum of severity. Ventricular arrhythmia (VA) can occur and worsen prognosis. The current study compared in-hospital and follow-up adverse cardiovascular events in patients with and without VA at presentation.

Methods: Eighty-three cases of SCAD were collected retrospectively from 4 Gulf countries (KSA, UAE, Kuwait, and Bahrain) during the period from January 2011 to December 2017. We divided the patients into 2 groups: those with and without VA at presentation. VA was defined as ventricular tachycardia and/or ventricular fibrillation. In-hospital (recurrent VA, cardiogenic shock, death, implantable cardioverter-defibrillator placement, dissection extension) and follow-up (MI, de novo SCAD, death, spontaneous superior mesenteric artery dissection) events were compared among the 2 groups.

Results: The median age of patients in the study was 44 (37–55) years. Forty-two (51%) were women. VA occurred in 10 (12%) patients in the first 24-hour of hospitalization, and 5 (50%) of those patients had recurrent in-hospital VA. Among those with recurrent VA, 1 died during hospitalization and 1 died within the first year following hospital discharge.

Conclusions: In-hospital adverse cardiovascular events were significantly more frequent for patients with SCAD who presented with VA. However, follow-up events were not statistically significant between those with and without VA at presentation.

Key Words: gulf, ventricular arrhythmias, SCAD

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INTRODUCTION

The risks of ventricular arrhythmia (VA) and sudden cardiac arrest vary in specific populations with different underlying cardiac conditions, specific family histories, and genetic variants. This variation has important implications for studying and applying therapies.[29]

Spontaneous coronary artery dissection (SCAD) was first described in 1931 by Pretty in the case of a 42-year-old woman with sudden cardiac death (SCD) following repetitive retching and vomiting. Autopsy revealed dissecting aneurysm of the right coronary artery.[44]

SCAD accounts for about 16% of non-atherosclerotic causes of SCA[13] and can cause life-threatening VA with ventricular tachycardia (VT) or ventricular fibrillation (VF) being the initial clinical presentation in about 3%–11% of cases.[1–3,23,33] SCD accounted for <1% of SCAD cases although this may be underestimated.[2,3,13,23]

The true incidence and prevalence of SCAD in the general population remains uncertain. According to recent publications, SCAD was diagnosed on coronary angiography in 1%–4% of all acute coronary syndromes (ACSs).[33] SCAD is an important cause of ACS and SCD: particularly among young women and individuals with few conventional atherosclerotic risk factors.[1–4] SCAD is defined as an epicardial coronary artery dissection that is not iatrogenic and is not associated with atherosclerosis or trauma.[5] Coronary artery obstruction occurs from formation of an intramural haematoma (IMH), intimal disruption rather than atherosclerotic plaque rupture or intraluminal thrombus.[6–22] The causes are hypothesized to be multifactorial; contributing factors include genetics, hormonal influences, systemic inflammatory diseases, inherited or acquired arteriopathies often compounded by environmental precipitants or stressors.[33] The presentation of SCAD is similar to atherosclerotic ACS[2–4,23,24] with chest pain as the most common symptom,[25] electrocardiography (ECG) evidence of acute ischemia or infarction and cardiac enzyme elevation.[3,23,26] Left ventricular (LV) wall motion abnormalities are common but LV ejection fraction is usually preserved.[27] Prognosis of patients with SCAD is more favorable compared with other patients presenting with acute myocardial infarction which may be related to a high burden of comorbidities and risk factors in the latter group.[43]

At an intermediate-term follow-up of 2–3 years major adverse cardiac events are common, occurring in 10%–30% of cases, mostly caused by recurrent MI from recurrent SCAD reported in 15%–22% of patients.[3,23,42] The impact of VA at presentation in patients with SCAD on prognosis is not well defined. In the present study, we compared in-hospital and follow-up adverse cardiovascular events in patients with and without VA at presentation.

METHODS

Patient Data

The details regarding the design, methods, and endpoints of this multicentre, observational study came from the SCAD in relation to physical and emotional stress.[40] A retrospective review was conducted from January 2011 to December 2017 by way of medical record review and telephone follow-up for all ACS patients from

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30 centers including 25 tertiary referral facilities and 5 regional hospitals in four Arab Gulf countries (Kingdom of Saudi Arabia, United Arab Emirates, Kuwait, and Bahrain). Subsequently, the diagnosis of SCAD was based on angiographic and intravascular imaging whenever available for ambiguous lesions and according to physician discretion. Coronary angiograms were reviewed by the primary cardiologist from each center and confirmed by the principal cardiologist from King Faisal Specialist Hospital and Research Center (Jeddah branch). If SCAD was confirmed, findings were classified as type 1, type 2, or type 3 angiographic SCAD.[22,39] Type 1 angiographic SCAD appears as the classic contrast dye staining of arterial wall with multiple radiolucent lumens with or without the presence of dye hang-up or slow contrast clearing from the lumen. Type 2 angiographic SCAD appears as diffuse (typically 20–30 mm) and smooth narrowing that can vary in severity and length, with type 2A being a narrowing bordered by a normal proximal and distal lumen and type 2B being narrowing that extends to the distal tip of the artery. Type 3 angiographic SCAD mimics atherosclerosis with focal or tubular stenosis. Optical coherence tomography and intravascular ultrasound are required to confirm the presence of IMH or double-lumen. The following patients were excluded: patients found to have coronary artery dissection due to atherosclerosis, blunt trauma, surgical instruments, or those that are catheter-induced, and in case of disagreement between the primary and principal cardiologist. None of the patients declined to participate in this registry. The study protocol was approved by the Institutional Review Board of King Faisal Specialist Hospital and Research Center and each of the participating hospitals. The patients were divided into 2 groups: those presenting with VA (VA group), and those without (non VA group).

Definitions

VA was defined as sustained VT and/or VF assessed by electrocardiogram on admission and during hospitalization.

De novo coronary artery dissection is defined as a new dissection in different epicardial vessels with resolution of the prior dissection in the originally affected vessels.

Extension of the dissection is defined as a continuation of an already-established dissection, either spontaneously or iatrogenically.

Initial Clinical Evaluation

At the time of hospitalization, a complete medical history and episodes of arrhythmias were recorded. A 12-lead ECG and routine laboratory studies were obtained. Telemetry and 12-lead ECG data were analyzed by cardiologists. Transthoracic echocardiographic and coronary angiography reports were obtained for all patients with confirmed diagnosis of SCAD.

VAs were assessed on admission and during hospital stay. Any episode of VT lasting longer than 30 seconds was considered sustained VT.

Follow-up Clinical Data

Follow-up data were obtained for all patients using standardized telephone interviews and treating physician reports. In cases of patient death, the cause was ascertained by thorough review of all available clinical information at the time of death. For patients treated with an implantable cardioverter-defibrillator (ICD), reports from outpatient visits were collected and reviewed.

Statistical Analysis

Categorical variables were summarized with absolute numbers and percentages. Continuous variables were summarized with mean and SD or median and interquartile range. Comparison between different groups was performed using the χ^2 test or Fisher exact test for categorical variables and independent-sample *t*-test or Mann-Whitney *U* test for continuous variables. All analyses were performed using SAS/STAT software, Version 9.2 (SAS Institute Inc., Cary, NC, and R foundation for Statistical Computing, Vienna, Austria). A 2-sided *P*-value <0.05 was considered statistically significant.

RESULTS

Baseline Characteristics, Hospital Presentation, and Management of SCAD Patients Presenting With and Without VA

Eighty-three patients with SCAD were identified retrospectively over a period of 6 years with a median follow-up duration of 18.8 months (interquartile range: 9.06–40.1 months). Ten of these patients (12%) had VA at presentation. Compared to patients without VA, patients with VA were similar in terms of age, sex, and cardiovascular risk factors as shown in Table 1. As shown in Table 2, STEMI was diagnosed at presentation in 90% of patients in the VA-group and 44% of patients in the non VA group ($P = 0.007$). Non-ST-elevation ACS was more common in the non VA group compared to VA group (52% vs. 10%, $P = 0.016$). There was no significant difference in reduced ejection fraction between the VA and non VA groups. In the VA group, admission levels of troponin and white blood cell count (WBC) were significantly increased compared to the non VA group ($P < 0.011$ and $P < 0.001$). As shown in Table 3, SCAD lesion characteristics, distribution, stenosis severity, length, and TIMI grade flow were not significantly different between the 2 groups. Forty percent of the patients were treated with medical management only which included aspirin (99%), P2Y12 inhibitors (90%), beta-blockers (89%), statins (84%), and ACE-I/ARB agents (65%). Revascularization was performed in 60% of the entire cohort, 53% with PCI and 7% with CABG. In the VA group, 6 patients (60%) underwent PCI and none had CABG. Table 4 demonstrates the characteristics of SCAD patients presenting with VA.

In-hospital and Follow-up Events

During hospital stay, 5 patients (50%) in the VA group had recurrent in-hospital VA as shown in Table 5. All patients with cardiogenic shock were in the VA group, and received inotropic support, and intra-aortic balloon pumps (4 vs. 0, $P < 0.001$). Five patients (50%) in the VA group received amiodarone while none received it in the non VA group. As shown in Table 5, the average hospital length of stay was longer in the VA group compared to non VA group (7.2 ± 3.2 days vs. 3.7 ± 0.98 days, $P < 0.001$). Among patients with VAs, 1 (10%) died during hospitalization and 1 (10%) had death following hospital discharge within the first year as shown in Table 5. There were no deaths in the non VA group during the study period. During follow-up, 4 patients (5.5%) had recurrent de novo SCAD. All of these occurred in the non VA group as shown in Table 5. No VA was observed in these 4 patients.

Device Implantation and Follow-up

In the entire cohort, 1 patient underwent ICD implantation because of recurrent VA during the hospital stay and low LVEF as shown in Tables 4 and 5. The patient was a 37-year-old male who presented with STEMI and on admission developed VT/VF and required defibrillation three times. He developed recurrent VT/VF during hospitalization and had a LVEF of 30%. A dual-chamber ICD was implanted. He was lost to follow up but a phone call to his family confirmed his death 10 months post-discharge.

DISCUSSION

Our study provides a unique examination of patients with SCAD in the Arab Gulf countries compared to western registries, as described by Daoulah et al.[40] The percentage of male and female with SCAD

were relatively balanced. Amongst enrolled females, we found a higher percentage of multiparity (21.4%) and pregnancy-associated SCAD (28.5%). Exogenous hormonal therapy was used in 12% of patients. In our cohort, no patients were diagnosed with fibromuscular dysplasia (FMD), connective tissue disorder, or vasculitis. However, no additional imaging or lab tests were performed to diagnose these associated conditions due to the retrospective nature of this study. Consequently, the prevalence of FMD in the Gulf countries remains unknown. In this study, female patients with SCAD demonstrated more cardiovascular events compared to males with higher average age (48 vs. 39 years). In addition, cardiovascular events were numerically higher in peripartum and multiparous females compared to other females during follow-up, as opposed to in-hospital events. Lastly, 2 out of 5 patients on exogenous hormonal therapy had cardiovascular events in non VA group as opposed to VA-group. The first patient was on postmenopausal therapy, and had an in-hospital event (Extension of Dissection) and (De novo SCAD) during follow-up. The second patient was on oral contraceptives, and had (De novo SCAD) during follow-up.

TABLE 1. Demographic Characteristic of SCAD Patients Presenting With and Without VA on Admission

	All Patients (n = 83)	Group Without VA (n = 73)	Group With VA (n = 10)	*P*
Demographics				
Age (years)	44 (37–55)	42 (25–79)	46 (26–81)	0.737
Female sex	42 (50.6%)	36 (49.31%)	6 (60%)	0.737
Body mass index (kg/m^2)	27 (24.8–30)	26.8 (17.2–43)	28.25 (22.8–31.3)	0.515
Smoker	37 (44.58%)	31 (42.46%)	6 (60%)	0.329
Comorbidities				
Insulin dependent diabetes mellitus	4 (4.82%)	2 (2.73%)	2 (20%)	0.069
Noninsulin dependent diabetes mellitus	17 (20.48%)	15 (20.54%)	2 (20%)	0.999
Arterial hypertension	26 (31.33%)	21 (28.76%)	5 (50%)	0.273
Dyslipidaemia	32 (38.55%)	28 (38.35%)	4 (40%)	0.998
Atrial fibrillation	0 (0.00%)	0 (%)	0 (%)	0.999
Congestive heart failure	2 (2.41%)	1 (1.36%)	1 (10%)	0.227
Chronic kidney disease	2 (2.41%)	2 (2.73%)	0 (%)	0.998
Hypothyroidism	6 (7.23%)	6 (8.21%)	0 (%)	0.999
Anxiety	28 (33.73%)	26 (35.61%)	2 (20%)	0.482
Depression	12 (14.46%)	11 (15.06%)	1 (10%)	0.999
Migraines	19 (22.89%)	17 (23.28%)	2 (20%)	0.816
Stress factors				
Emotional stress	33 (39.76%)	28 (38.35%)	5 (50%)	0.508
Physical stress	9 (10.84%)	8 (10.95%)	1 (10%)	0.927
Physical and emotional stress	7 (8.43%)	7 (9.58%)	0 (%)	0.589

TABLE 2. Clinical Presentation of SCAD Patients on Admission

	All Patients (n = 83)	Group Without VA (n = 73)	Group With VA (n = 10)	*P*
Hospital presentation				
Chest pain	3 (3.61%)	3 (4.10%)	0 (%)	0.513
STEMI	41 (48%)	32 (43.83%)	9 (90%)	0.007
NST-ACS	39 (46.99%)	38 (52.05%)	1 (10%)	0.016
2-D echocardiogram on admission				
Left ventricular ejection fraction (%)				
EF < 20%	1 (1.2%)	1 (1.36%)	0 (%)	0.709
EF 20-35%	13 (15.66%)	10 (13.69%)	3 (30%)	0.186
EF 35-50%	45 (54.21%)	38 (52.05%)	7 (70%)	0.331
EF > 50%	24 (28.91%)	24 (32.87%)	0 (%)	0.056
Lab test on admission				
WBC (g/L), median (IQR)	10 (6.45–12)	9 (4–18)	12.5 (10–17)	<0.001
Troponin (ng/L), median (IQR)	2.69 (0.05–20)	2.05 (0–1510)	22.5 (0.5–50)	0.011
CK (U/L), median (IQR)	265 (108.5–737)	26 (12–2014)	617.5 (14.7261)	0.512
CKMB (ug/L), median (IQR)	33 (5.85–84.5)	24 (0.657–569)	152.8 (0.6–700)	0.038

CK, creatine kinase; CKMB, creatine kinase muscle/brain; CRP, c-reactive protein; EF, ejection fraction; NST-ACS, Non-ST-elevation acute coronary syndrome; STEMI, ST-elevation myocardial infarction; WBC, white blood cell count.

TABLE 3. Angiographic Characteristics of SCAD Patients and Their Management

	All Patients (n = 83)	Group Without VA (n = 73)	Group With VA (n = 10)	*P*
Angiographic characteristics of the SCAD lesions				
Coronary artery territory involved				0.275
Left main	10 (12.05%)	9 (12.32%)	1 (10%)	
Left anterior descending artery	36 (43.37%)	31 (42.46%)	5 (50%)	
Left circumflex artery	8 (9.64%)	8 (10.95%)	0 (%)	
Right coronary artery	18 (21.69%)	17 (23.28%)	1 (10%)	
Multi-vessel	8 (9.64%)	5 (6.84%)	3 (30%)	
Branch vessel	3 (3.61%)	3 (4.10%)	0 (%)	
Lesion characteristics				0.152
Type 1	43 (51.81%)	36 (49.31%)	7 (70%)	
Type 2	35 (42.17%)	33 (45.20%)	2 (20%)	
Type 3	3 (3.61%)	3 (4.10%)	0 (%)	
Multi-type	2 (2.41%)	1 (1.36%)	1 (10%)	
TIMI coronary grade flow				0.306
TIMI 0	8 (9.64%)	8 (10.95%)	0 (%)	
TIMI 1	13 (15.68%)	13 (17.80%)	0 (%)	
TIMI 2	21 (25.30%)	17 (23.28%)	4 (40%)	
TIMI 3	41 (49.40%)	35 (47.94%)	6 (60%)	
Maximum stenosis severity (%)	80 (50–95)	80 (10-100)	80 (40-100)	0.955
Maximum dissection length (mm)	25 (18–36)	25 (5-80)	24 (20-85)	0.547
Acute management				
Medical management only	33 (39.76%)	29 (39.72%)	4 (40%)	0.998
Percutaneous coronary intervention	44 (53.01%)	38 (52.05%)	6 (60%)	0.742
Coronary artery bypass grafting	6 (7.23%)	6 (8.21%)	0 (%)	0.346

TIMI, thrombolysis in myocardial infarction.

Although VA has been described as a complication of SCAD, the exact incidence and mechanisms have not been well studied. The previously reported incidence of VA in SCAD patients varied from 3% to 11%.[1–3,23] We reported a similar percentage of VA at presentation (12%) in our patients. These reports must be interpreted with care. Not included in these studies are patients with missed or misdiagnosed SCAD, those who did not survive to initial evaluation and those in whom coronary imaging or postmortem evaluation was not performed. In the VA group, all VT degenerated into VF requiring at least 2 external defibrillations. In patients with VA, electrical instability leading to subsequent shock and the use of inotropic support and intra-aortic balloon pumps were more frequent. Five of 10 (50%) patients had recurrent VA during hospitalization. All episodes of recurrent VA occurred within 4 days of hospitalization. Prior

TABLE 4. Characteristics of SCAD Patients Presenting With VA

Age (years)	Sex	Hospital Presentation	Troponin (ng/L)	WBC (/L)	LVEF (%)	Coronary Artery Distribution With SCAD	TIMI Flow	Lesion Characteristics	Stenosis Severity (%)	Length Category	In-hospital Events	Acute Management	Follow-up Events
39	F	STEMI	1.0	11	40	LAD	2	Type 2	50	Intermediate	No event	Medical	No event
26	F (PP)	STEMI	47	13	45	LAD	2	Type 2	60	Diffuse	No event	Medical	No event
37	M	STEMI	15	13	30	LAD and RCA	3	Type 1	80	Diffuse	VT/VF, ICD	Medical	Death
51	M	NST-ACS	0.5	15	45	RCA	3	Type 1	50	Diffuse	No event	Medical	No event
81	F (PM)	STEMI	35	12	30	LAD and LCX	2	Type 1	80	Intermediate	VT/VF, Shock	PCI	No event
45	F (M)	STEMI	50	17	40	LM	3	Type 1	95	Diffuse	VT/VF, Shock	PCI	No event
34	F	STEMI	20	12	25	LAD	3	Type 1	75	Diffuse	VT/VF, Shock, Death	PCI	
70	M	STEMI	25	12	40	LAD	3	Type 1	95	Intermediate	No event	PCI	No event
57	F (PM)	STEMI	30	14	45	LCX and RCA	2	Type 1 and 2	100	Intermediate	VT/VF, Shock	PCI	No event
48	M	STEMI	2.5	10	45	LAD	3	Type 1	80	Diffuse	No event	PCI	No event

F, female; LAD, left anterior descending coronary artery; LCX, left circumflex coronary artery; LM, left main coronary artery; M, male; M, Multiparous; NST-ACS, non-ST segment acute coronary syndrome; PCI, percutaneous coronary intervention; PM, Post-Menopausal; PP, Post-Partum; RCA, right coronary artery; STEMI, ST-segment elevation myocardial infarction; WBC, white blood cell count.

TABLE 5. In-hospital Events and Follow-up Events of SCAD Patients Presenting With and Without VA

	All Patients (n = 83)	Group Without VA (n = 73)	Group With VA (n = 10)	*P*
In-hospital events				
Recurrent VA	5 (6.02%)	0 (0.00%)	5 (50%)	<0.001
Cardiogenic shock	4 (4.81%)	0 (0.00%)	4 (40%)	<0.001
Death	1 (1.2%)	0 (0.00%)	1 (10%)	0.120
ICD	1 (1.2%)	0 (0.00%)	1 (10%)	0.120
Extension of dissection	3 (3.61%)	3 (4.1%)	0 (0.00%)	0.998
Composite events	8 (9.63%)	3 (4.1%)	5 (50%)	<0.001
Hospital length of stay (days)	4.15 ± 1.7	3.7 ± 0.98	7.2 ± 3.2	<0.001
Follow-up events	n = 82	n = 73	n = 9	
Myocardial infarction	6 (7.31%)	5 (6.84%)	1 (11.11%)	0.513
De novo SCAD	4 (4.87%)	4 (5.47%)	0 (0.00%)	0.999
Death	1 (1.21%)	0 (0.00%)	1 (11.11%)	0.109
Spontaneous superior mesenteric artery dissection	1 (1.21%)	1 (1.36%)	0 (0.00%)	0.998
Composite events	6 (7.31%)	5 (6.84%)	1 (11.11%)	0.513

studies suggest 3–5 days of in-hospital monitoring for ischemia and/or significant VA is justified to detect a small but important (5%–10%) early hazard of dissection extension or new recurrent SCAD.[3,33] However, the duration of VA risk and the optimal duration of monitoring are still unclear.[28,29] Notably, patients presenting with VA who survive to hospital discharge may or may not have a good prognosis.[30–32] In our study, one patient (10%) died within the first year following hospital discharge. A previously described SCAD registry of 102 patients included 14 patients who experienced SCD. These patients were more likely to be peripartum, tobacco users, have presented with STEMI, have received ICD placement and had higher incidence of repeat SCAD.[38] In our study patients with SCD who presented with VA were more likely to have ST-segment elevation myocardial infarction, elevated troponin T, and elevated (WBC) count. Additionally, they had longer hospital stays due to recurrent VA and/or cardiogenic shock. In contrast to the above-mentioned study, we found no recurrent de novo SCAD in our patients who presented with VA.

Considering mechanisms for VA in SCAD, catecholamine discharge has been postulated to lead to coronary artery shear stress that in part contributes to the pathophysiology of SCAD. Catecholaminergic surge and automatic mechanisms are the most probable contributors for arrhythmogenesis in SCAD. Although this hypothesis has not been tested in SCAD patients, a similar mechanism has been proposed in other stress-induced cardiovascular conditions such as takotsubo cardiomyopathy.[34,35] Catecholamine release can also induce inflammation, which plays an important role in the acute setting and during myocardial recovery after myocardial infarction.[36] Inflammation may contribute to maladaptive remodeling processes following infarction.[37] Anti-inflammatory strategies may be potential therapeutic approaches to reduce inflammation and subsequent hazards. Indications for the placement of an ICD for VA in patients with SCAD remain uncertain. We found that 1 patient with ICD placement died less than 1 year after discharge. This calls into question the survival benefit of ICD placement in patients with SCAD and VA, and prevents us from offering specific recommendations on indications for device implantation. This must be decided on a case-by-case basis.

Based on our findings, we believe future large scale prospective and epidemiological studies will help further our understanding of the demographic, pathogenesis, treatment, and anatomic factors associated with recurrence and allow more accurate prediction and ultimate prevention of recurrent SCAD.

Limitations

Limitations of this study include the small sample size and selection, referral, and attrition biases due to the unavoidable nature of a retrospective registry. For instance, the current cohort, by default, did not incorporate patients who did not survive their initial SCAD. Similar to other registry studies, our study may have underestimated the prevalence of SCAD by failing to include patients with missed or misdiagnosed SCAD, those who did not survive to initial evaluation, and those in whom coronary imaging or postmortem evaluation was not performed. The retrospective analysis may have underestimated the prevalence of VA. Measurement bias could not be formally excluded. Nevertheless, the data collection was standardized based on the computerized database with data entry and analysis performed by physicians blinded to the eventual categorization of patient groups. Patients were acutely managed in the intensive or coronary care units, allowing standardized and close monitoring of heart rhythm and hemodynamics. The current data do not allow specific recommendations for optimal management of VA in patients with SCAD.

CONCLUSIONS

VA is not uncommon in patients with SCAD, both on admission and during initial hospital stay, and VA impacts short-term prognosis. Further study is needed to guide the use of ICD in patients with SCAD.

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DISCLOSURES

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