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Review Article

Immediate versus delayed detection of Takotsubo syndrome after epileptic seizures

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ABSTRACT

Takotsubo syndrome(TTS) is often preceded by emotional or physical stress. Epileptic seizures are described in > 100 cases. It is unknown whether patients with immediate and delayed detection of seizure-induced TTS differ.

We screened the literature and compared clinical and electrocardiographic (ECG) findings. In 48 cases with seizure-associated TTS, the time between seizure and TTS-detection was reported. Troponin levels were elevated in 37/40. ECG abnormalities were negative T-waves(40%), ST-elevations(33%) and ventricular fibrillation/flutter(10%). Immediate detection was reported in 23 patients, in the remaining 25 patients, TTS was detected 5–288 h postictally. Patients did not differ in gender, age or symptoms. Negative T-waves were more frequent in patients with delayed detection(64 vs. 13%, $p = .0009$), whereas ECG-abnormalities suggesting acute myocardial infarction tended to be more prevalent in patients with immediate detection.

Due to lack of typical symptoms, seizure-induced TTS can be overlooked. Postictally, an ECG should be recorded and troponin levels measured. New T-wave inversions might indicate seizure-induced TTS.

1. Introduction

Takotsubo syndrome (TTS) is characterized by transient systolic left ventricular dysfunction, most frequently in the apical region, which cannot be explained by coronary artery disease. The clinical presentation, electrocardiographic (ECG) findings, and biomarker profiles are often similar to those of an acute coronary syndrome [1]. TTS predominantly affects elderly women and is often preceded by emotional or physical stress [1]. Epileptic seizures as triggering events of TTS are, so far, described in > 100 cases [2–16]. Seizure-induced TTS may occur immediately after the seizure and can be diagnosed at hospital admission. However, TTS may also occur delayed, several hours, after the seizure. Postictal somnolence or lack of cardiologic surveillance impedes the diagnosis of TTS after a seizure. So far, it is unknown whether patients with immediate and delayed detection of seizure-induced TTS differ. Thus aim of the present review was to screen the literature for information about the interval between epileptic seizure and TTS.

2. Methods

A literature research was carried out in PubMed with the search terms “Takotsubo/Tako-tsubo/ apical ballooning/ transient left ventricular dysfunction” and “Epilepsia/ epilepsy/ seizure”. Clinical trials, case series and case reports, published in English, German, French, Spanish, Polish and Japanese were considered. Articles which contained information about the interval between epileptic seizure and detection of TTS were included for the analysis. Furthermore, we included only articles which reported 12-lead ECG findings. We compared cases with immediate versus delayed detection of TTS regarding demographic, clinical, ECG findings and, if reported, troponin levels. For statistical analysis of categorical variables, the Chi-Square-test was used. Additionally, we applied the Fisher's exact test to check the robustness. Continuous variables were compared using Welch's *t*-test.

3. Results

Our research disclosed 45 articles, reporting 48 cases with seizure-associated TTS where the time interval between seizure and detection of TTS was clearly described. For all cases, the data were collected retrospectively. Most articles were reports about a single case, 5 articles comprised case series [17–21]. In 23 cases, TTS was reported to develop immediately after the seizure and signs of TTS were already present at hospital admission (Table 1) [4–6,8,9,13,14,16,20–34]. In the remaining 25 cases, detection of TTS was reported 5–288 (mean 50)

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Table 1
Cases with detection of Takotsubo syndrome immediately after seizure (n = 23).

Cause of epilepsy/ neurologic disease	Sex	Age	ST	AET	TTST	TSy	ASy	Intu	Trop	ECG	OC	Author
Cerebrovascular disease	F	75	TCS	LEV	Glo	None	Conf	No	Nm	VT/F	FR	[16]
Genetic	F	68	TCS, SE	None	Ap	None	None	Yes	↑	ST↑	FR	[9]
Nm	F	50	TCS	None	Bas	None	None	No	↑	Qw	FR	[22]
Left frontal hypodensity	M	39	Nm	Nm	Bas	Nm	Nm	Nm	↑	ST↑	FR	[20]
Right mesial temporal sclerosis	F	51	TCS	CBZ, PHT, ZNS, TPM	Ap	None	Nm	Nm	Nm	VT/F	FR	[23]
Genetic	F	47	SE	None	Ap	None	None	Yes	⊥	VT/F	FR	[24]
PRES	F	82	SE	None	Ap	None	None	No	↑	ST↑	FR	[25]
PRES	M	69	TCS	None	Ap	None	None	Yes	↑	LBB	FR	[8]
Alkohol	F	25	Nm	None	Ap	None	None	No	⊥	VT/F	FR	[26]
Stroke	F	83	TCS, SE	None	Ap	None	Hypo	No	↑	ST↑	FR	[5]
Nm	F	75	Nm	Nm	Mid	Nm	Coma	Nm	Nm	ST↑	Nm	[21]
Stroke	F	54	TCS, SE	LMT	Ap	Dysp	Hypo	No	↑	Neg T	FR	[27]
Nm	F	49	TCS, SE	Nm	Mid*	None	None	Yes	↑	⊥	FR	[4]
Nm	F	64	TCS	None	Ap	CP	None	No	↑	Qw	FR	[6]
Stroke	F	75	CPS	Nm	Ap	CP	Nm	No	↑	ST↑	FR	[28]
Stroke	F	67	TCS	None	Ap	None	None	No	↑	ST↑	FR	[29]
Medication withdrawal	F	42	Nm	CBZ	Mid	None	Conf	No	↑	Neg T	FR	[30]
Cavernoma mesiotemporal	F	71	TCS	GBT	Ap	CP, Dysp	None	No	↑	ST↑	†	[31]
Nm	F	72	TCS	None	Ap	RI	None	Nm	↑	ST↓	FR	[13]
Nm	F	68	TCS, SE	CBZ	Ap	None	Coma	Nm	↑	Neg T	FR	[32]
Meningeoma	F	76	TCS	None	Ap	CP	Conf	No	↑	ST↓	FR	[14]
Genetic	F	64	TCS	CBZ, PHT	Ap	Dysp	Conf	No	↑	ST↑	FR	[33]
Multiple sclerosis	F	69	TCS	None	Ap	None	Conf	Yes	↑	ST↑	FR	[34]

Abbreviations: *no coronary angiography
† = death.
⊥ = normal.
AET = antiepileptic therapy.
AF = atrial fibrillation.
Age = Age in years.
Ap = apical type of TTS.
ASy = atypical symptoms of TTS.
Bas = basal type of TTS.
CBZ = carbamazepine.
CLB = clobazam.
Coma = comateuse.
Conf = confused.
CP = Chest pain.
CPS = complex partial seizures.
Dysp = Dyspnea.
ECG = electrocardiographic abnormalities.
F = female.
FR = full recovery.
GBT = gabapentine.
Glo = globally reduced left ventricular function.
h = hours after the seizure.
Hypo = arterial hypotension.
ICB = intracerebral bleeding.
Intu = Intubation because of airway protection or respiratory insufficiency.
LEV = levetiracetam.
LMT = lamotrigine.
M = male.
Mid = midventricular.
Neg T = negative T waves in the electrocardiogram.
Nm = not mentioned.
OC = outcome.
PHT = phenytoin.
PRES = posterior reversible encephalopathy syndrome.
PRM = primidone.
QT = QT interval at electrocardiogram.
Qw = Q waves.
rever = reverse type of TTS.
SAB = subarachnoid bleeding.
SE = status epilepticus.
ST = seizure type.
TCS = tonic clonic seizure.
TPM = topiramate.
Trop = Troponin levels after seizure.
TTST = TTS type.
VPA = valproic acid.
VT/F = ventricular tachycardia/fibrillation.
ZNS = zonisamide.

Table 2
Cases with detection of Takotsubo syndrome ≥5 h after seizure (n = 25).

Cause of epilepsy/ neurologic disease	Sex	Age	ST	AET	TTST	TSy	ASy	Intu	Seizure-TTS	Trop	ECG	OC	Author
PRES	F	55	TCS	None	Ap	None	Conf	No	72 h	↑	Neg T	FR	[35]
Nm	F	79	SE	LEV, CLB	Ap	None	Aphasia	No	24 h	↑	*Neg T	FR	[36]
Mesiotemporal sclerosis	F	61	TCS, SE	CBZ, LEV	Ap	CP	None	No	12 h	↑	ST↑, Neg T	FR	[37]
Previous SAB	F	50	TCS	Nm	Mid[a]	None	None	Yes	48 h	↑	Neg T	FR	[38]
Genetic	F	67	TCS	CBZ, VPA, PRM	Ap	None	Hypo	No	48 h	↑	VT/F, Neg T	FR	[39]
ICB	F	55	TCS	PHT, LEV	Ap	CP	None	No	5 h	↑	Neg T	FR	[40]
Nm	F	73	Nm	Nm	Ap	CP	None	No	12 h	↑	ST↓	FR	[41]
Alkohol withdrawal	M	53	TCS	None	Ap	None	Hypo	No	72 h	↑	*Neg T	FR	[10]
Nm	F	18	SE	Nm	Glo	Nm	None	Yes	96 h	Nm	QT↑	FR	[17]
Nm	F	47	SE	Nm	Glo	Nm	None	Yes	288 h	Nm	Neg T	FR	[17]
Nm	M	25	SE	Nm	Glo	Nm	None	Yes	264 h	↑	QT↑	FR	[17]
Nm	F	61	TCS	Nm	Ap	None	None	No	12 h	↑	ST↑	FR	[3]
Genetic	F	50	TCS	Nm	Ap	None	Hypo	Yes	5 h	↑	ST↓	FR	[18]
Schizophrenia	F	63	TCS	Nm	Ap	None	Hypo	Yes	5 h	↑	ST↑	FR	[18]
Stroke	F	62	TCS	Nm	Ap	None	None	No	10 h	↑	ST↑, Qw	FR	[42]
Water intoxication	F	41	TCS	None	Glo	Dysp	Conf	Yes	12 h	↑	ST↑	FR	[11]
Alkohol withdrawal	F	49	Nm	Nm	Ap	None	Conf, Hypo	No	12 h	⊥	Neg T	FR	[43]
Cavernoma bleeding	F	74	TCS	None	Ap	CP	Conf	No	28 h	↑	*Neg T, AF	FR	[19]
SAB	F	60	TCS	ZNS	Ap	None	None	No	24 h	Nm	Neg T	FR	[44]
Stroke	F	44	TCS	PHT	Ap	None	None	Yes	12 h	↑	*Neg T	FR	[45]
Alkohol withdrawal	M	63	TCS	None	Ap	Dysp	None	No	48 h	↑	Neg T	FR	[46]
Nm	F	61	SE	Nm	Ap	None	Nm	Nm	48 h	Nm	*Neg T	Nm	[12]
Genetic	F	69	TCS	None	Ap	None	Conf	No	26 h	Nm	Neg T	FR	[47]
Opiate withdrawal	F	58	TCS	None	Ap	None	Coma	Yes	8 h	↑	*ST↑	FR	[48]
Genetic	F	67	TCS	LMT, PRM, CLB	Ap	CP	Hypo	No	48 h	↑	*Neg T	FR	[49]

[a] Abnormalities were not reported in a previous ECG, the abbreviations are the same as for Table 1.

hours after the seizure (Table 2) [3,10–12,17–19,35–49].

Most patients (90%) were females and the age ranged between 18 and 82 years. There were no significant differences in gender or age between patients with immediate and delayed detection of postictal TTS, although in the delayed group, the patients tended to be younger (Table 3). The apical type of TTS was most frequently reported in 77%. The typical symptoms of TTS, chest pain (19%) and dyspnea (10%) were not frequently reported, whereas atypical symptoms like confusion (21%) or hypotension (17%) were rather frequent. Twenty-nine% of the patients were intubated at onset of TTS because of respiratory insufficiency which was either postictal, due to the underlying neurologic disorder or a side effect of the therapy which they received for the seizure.

Table 3
Patients with seizure-associated Takotsubo syndrome: Comparison of immediate versus delayed (≥ 5 h) detection of symptoms.

Characteristic	Immediate (n = 23)	Delayed (n = 25)	p
Age, years, mean ± SD	62 ± 15	56 ± 14	0.1483
Females %	91	88	1.0000
Cause of epilepsy			
Alcohol %	4	12	0.6161
Stroke %	17	8	0.3307
Genetic %	13	16	1.0000
PRES %	9	4	0.6069
Seizure type			
Status epilepticus %	30	24	0.8602
Tonic clonic %	65	72	0.8456
Takotsubo type			
Apical %	74	80	0.8748
Midventricular %	13	4	0.3307
Takotsubo symptoms			
Chest pain %	17	20	1.0000
Dyspnoea %	13	8	0.6547
Confusion %	22	20	1.0000
Coma %	9	4	0.6026
Hypotension %	9	24	0.2593
Intubation %	28	38	0.7409
Troponin ↑ %	78	76	1.0000
Electrocardiogram			
Negative T waves %	13	64	0.0009
ST↑ %	44	24	0.2612
ST↓ %	9	8	1.0000
Ventricular tachycardia/ fibrillation %	17	4	0.1699

The abbreviations are the same as for Table 1.

Postictal serum troponin T or I levels were reported in 40/48 patients (Tables 1 and 2). In 37 of these 40 patients, troponin levels were elevated. No significant differences in the prevalence of elevated troponin levels could be detected between patients with immediate and delayed detection of TTS (Table 3).

The ECG was normal in only one patient with immediate detection of TTS (Table 1) [4]. In the remaining patients, one or more ECG abnormalities were detected, as listed in Tables 1 and 2. The most frequent ECG abnormalities were negative T-waves (40%), ST-elevations (33%) and ventricular fibrillation/flutter (10%). ECG-abnormalities like ST-elevations, ST-depressions, left bundle branch block and ventricular fibrillation/flutter, suggesting acute myocardial infarction due to coronary artery disease, tended to be more prevalent in the group of patients with immediate than delayed detection of TTS (Tables 1, 2 and 3). The prevalence of negative T-waves was higher among patients with delayed than with immediate detection of TTS (Table 3). In 7 of the 25 cases with delayed detection of TTS, an earlier ECG performed at admission was reported which showed no ST- or T-wave abnormalities [10,12,19,36,45,48,49].

In 12/48 cases, no cause of epilepsy was reported. Among the reported cases, the most frequent causes were genetic (n = 7), stroke (n = 6), alcohol withdrawal (n = 4) or posterior reversible encephalopathy syndrome (PRES) (n = 3). The antiepileptic therapy was reported for 14 cases, in further 15 cases, it was not mentioned in the report and in the remaining 19 cases the seizure occurred without previous antiepileptic therapy (Tables 1 and 2).

4. Discussion

This study about the interval between the triggering seizure and the development of TTS in published cases with TTS did not show any differences between patients with immediate and delayed detection of TTS regarding underlying neurologic disorders, demographic

characteristics, TTS-type and symptoms. The only difference was the prevalence of T-waves, which was more frequent among patients with delayed than immediate detection of TTS.

Up to now, the pathogenesis of TTS is not completely clarified [1]. Elevated catecholamine levels seem to play an important role. In patients with epileptic seizure, a significant increase in plasma norepinephrine, epinephrine, noradrenaline, prolactin, vasopressin and oxytocin levels has been observed [50,51]. Furthermore, it may be possible that there is an additional external trigger leading to a catecholaminergic storm causing both a seizure and TTS. Up to now it is unknown if there are risk factors to develop TTS after a seizure. The mechanism of the delay between seizure and TTS may be related to autonomic instability or more prolonged catecholamine release despite seizure control. Alternatively, it is possible that clinically silent seizures continued despite apparent resolution. Also the postictal period may be associated with increased catecholamine release.

ECG abnormalities in TTS have a typical pattern (Fig. 1): The initial ST-elevation may be subtle, lasts only for several hours and is followed by T-wave inversion which persists for several weeks [52]. ECG-abnormalities like ST-elevations, ST-depressions, left bundle branch block and ventricular fibrillation/flutter, suggesting acute myocardial infarction, tended to be more prevalent in the group of patients with immediate than delayed detection of TTS (Tables 1, 2 and 3). Probably, these findings may have induced acute coronary angiography in these patients and thus facilitated the diagnosis of TTS. The persistence of negative T-waves for weeks may have facilitated the diagnosis of TTS in the patients with delayed detection, especially when a previous ECG was available without T-wave abnormalities. Negative T-waves in TTS are assumed to be due to myocardial edema. This assumption is based on clinical observations such as the parallel time course of development and resolution of ventricular repolarization abnormalities and the demonstration of myocardial edema on initial and follow-up cardiac magnetic resonance images [53].

Troponin levels were elevated in most of the patients included in the present review and their prevalence did not differ between TTS with immediate and delayed detection. Troponin elevation is reported to occur in 12% of patients after generalized tonic-clonic seizures [54,55]. Unfortunately, the ECG findings and follow-up data of patients from these two studies are incompletely reported, thus it cannot be assessed, how many of them eventually developed TTS [54,55]. Troponin elevation after a seizure, however, may not only be due to TTS but also due to rhabdomyolysis occurring sometimes as a consequence of a seizure [54]. When interpreting troponin level elevations it should be considered that different assays were used in the past. In the meantime, high-sensitivity cardiac troponin assays with their superior analytical performance were designed [56].

The prevalence of seizure-induced TTS, so far, is unknown. In a study, published 2010, troponin levels in 279 consecutive patients admitted to an emergency room with cerebral seizures were investigated. Troponin levels were elevated in 18 patients (7%). Among these 18 patients, TTS was diagnosed in 5 patients (27%), thus the prevalence of TTS was 1.8% [57].

Unfortunately, it is not possible to compare the type of antiepileptic medication between patients with immediate and delayed detection of TTS since the medication was not mentioned in a third of the cases. Thus it cannot be assessed whether the type of medication – with sedating or non-sedating effects – may have influenced the time interval between seizure and TTS.

The mean interval between seizure and detection of TTS was 50 h. As a consequence of this finding, cardiologic surveillance for at least 72 h, relatively long period, has to be considered, which would be an enormous challenge to the health care system [58]. This stresses the need to obtain data from prospective studies including large numbers of consecutive patients about the prevalence of seizure-induced TTS, risk factors and time course of TTS after seizures.

Limitation of our review is that all conclusions are only based on

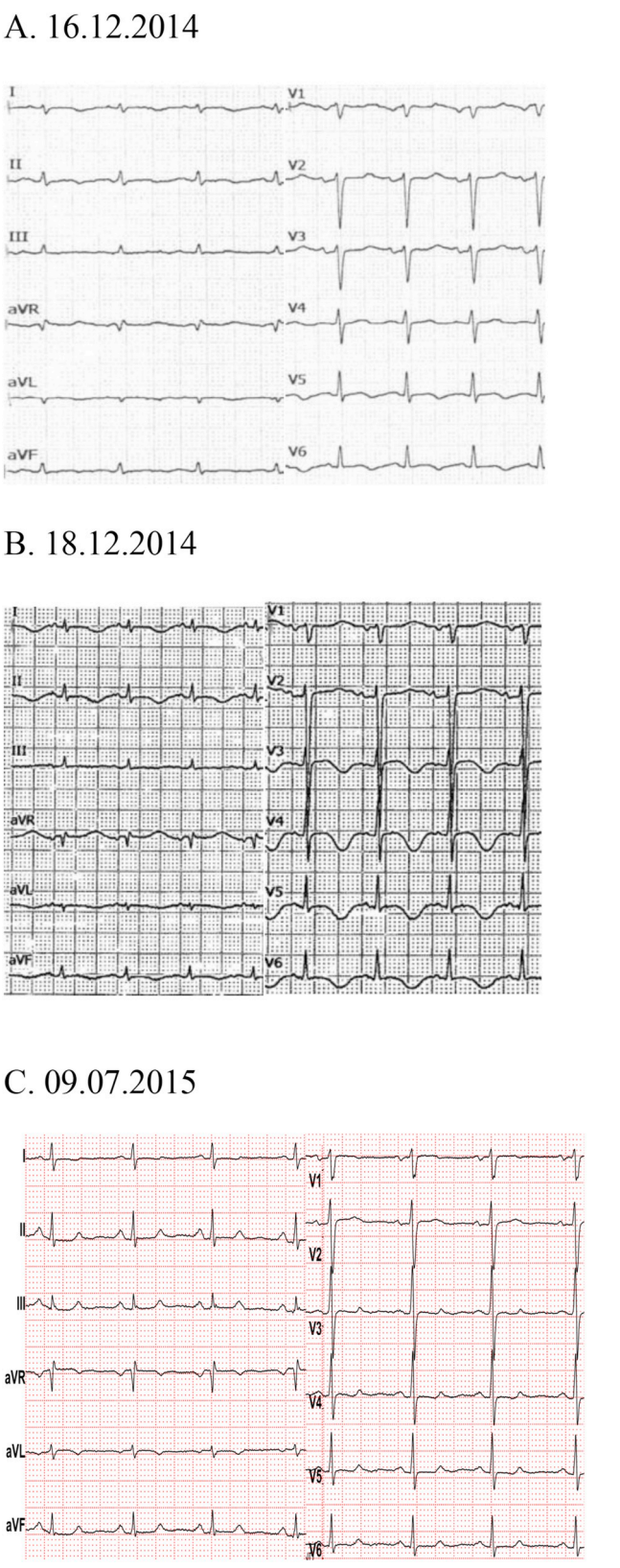

Fig. 1. Typical pattern of electrocardiographic changes in a patient with Takotsubo syndrome. Initially, there are only slight T – wave abnormalities (A). Two days later, negative T-waves are present in leads I, II, aVL, aVF, $V_{3–6}$ and a prolonged QTc interval (590 ms) (B). After six months, the abnormalities have regressed (C).

case reports or case series. Most of the reports did not detail the time interval between onset of the seizure and the first cardiac symptoms. Furthermore, the duration estimates in retrospective reports are arbitrarily. Recall bias will play an important role and the definition of symptom detection may be variable. Thus, a part of cases with TTS onset immediately after the seizure may be recognized as “delayed detection group” due to delayed diagnosis.

5. Conclusion

Seizure-induced TTS may occur immediately or delayed. Since no risk factors for delayed detection of TTS after seizures are known, it seems prudent to observe patients cardiologically after a seizure. Since patients often do not typically experience symptoms of angina pectoris or dyspnea after epileptic seizures or may even be asymptomatic, TTS can easily be overlooked. Thus, a postictal ECG should be recorded, troponin levels should be measured and their hemodynamic and rhythmologic condition should be monitored. In cases with troponin elevation and normal postictal EGC, especially looking for T-wave inversion, is recommended. Cases with postictal ECG abnormalities should undergo assessment of brain natriuretic peptides and echocardiography [59]. If wall motion abnormalities or ECG abnormalities are detected, coronary angiography should be carried out to confirm or exclude the diagnosis of TTS or coronary artery disease. Due to lack of data, the optimal duration for postictal cardiologic surveillance is, so far, unclear.

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