



Review

Seizure-triggered Takotsubo syndrome rarely causes SUDEP

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ABSTRACT

Since almost 20 y it is known that seizures may trigger Takotsubo syndrome (TTS). Since then it has been repeatedly proposed that TTS could be the cause of sudden unexpected death in epilepsy (SUDEP).

A review of the so far reported cases of seizure-triggered TTS was carried out to see how often seizure-triggered TTS is fatal.

Altogether 59 papers were identified which reported altogether 74 patients with seizure-triggered TTS. Age was reported in 70 patients and ranged from 18 to 82 y. Gender was reported in 70 cases and was female in 60 cases (86%). The type of triggering seizure was reported in 47 cases. In 28 patients (60%) the trigger was a generalized tonic clonic seizure, in 15 cases (32%) a generalized status epilepticus, and in 3 cases a complex partial seizure. The outcome was mentioned in 63 of the 74 patients. Full recovery was reported in 61 cases (97%), incomplete recovery in none of the patients, and a fatal outcome in 2 patients (3%).

Fatalities are rare in patients experiencing seizure-triggered TTS. This is why seizure-triggered TTS does not seem to play a major role in the pathogenesis of SUDEP.

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1. Introduction

Central nervous system (CNS) disease is increasingly recognized as a trigger of Takotsubo syndrome (TTS), also known as Tako-tsubo cardiomyopathy, ampulla cardiomyopathy, apical ballooning, neurogenic stress cardiomyopathy, broken heart syndrome, neurogenic stunned myocardium, transient regional left ventricular dysfunction, transient myocardial dysfunction, transient systolic dysfunction, neurogenic stressed myocardium, catecholamine cardiomyopathy, or reversible acute heart failure [1,2]. TTS mimics myocardial infarction clinically, electrocardiographically, and chemically. Clinically, TTS is characterized by acute onset chest pain and dyspnea, occasionally associated with palpitations, coughing, edema, tiredness, syncope, fever, nausea, vomiting, or anxiety [3]. Creatine-kinase (CK), troponin-T, and proBNP may be elevated. Electrocardiography (ECG) may show initial ST-elevation, which dynamically changes to negative T-waves, and finally complete resolution of the abnormalities [4]. Echocardiography may show segmental hypokinesia or akinesia and reduced systolic function and compensatory hyperkinesia of segments not affected by the stunning. Most frequently, stunning affects the apical or

midventricular segments of the left ventricular myocardium (classical type), but rarely also the midventricular segments (midventricular type), the basal or midventricular segments (inverted type), or all segments (global type) are affected [2]. According to a recent review [3], the CNS disorder most frequently triggering TTS is subarachnoid bleeding but the CNS disorder second most frequently triggering TTS is epilepsy [3]. This mini-review aimed at highlighting recent advances concerning the prevalence, age at occurrence, gender distribution, TTS type, and outcome of TTS triggered by epileptic seizures or an epileptic state. Additionally, we were interested in the cause of epilepsy, the seizure type, and the antiepileptic drug (AED) therapy prior to the TTS.

2. Methods

Data for this review were retrieved by searches of MedLine and references from relevant articles using the search terms “epilepsy” and “seizures” combined with “Takotsubo syndrome”, “Takotsubo cardiomyopathy”, “ampulla cardiomyopathy”, “stress cardiomyopathy”, “apical ballooning”, “broken heart syndrome”, and “stunned myocardium”. Randomized (blinded or open label) clinical trials, longitudinal studies, case series, and case reports were pondered. Only articles published in English between 1966 and 2015 were considered. Appropriate papers were studied and discussed for their suitability to be incorporated in this review.

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3. Results

Altogether 59 papers matching the search terms were selected. Full access was available to 47 articles (80%) and 12 papers were accessible only as an abstract. Fifty-one papers were case reports or single cases with epilepsy in a cohort study. Eight papers reported more than 1 patient but none of the papers reported more than

5 patients (Table 1). Altogether, 74 patients with epilepsy-triggered TTS were identified (Table 1). Among the nine papers reporting >2 patients, all were accessible as full papers. In the study with 5 patients nothing is reported about the cause, type, or treatment of seizures [5]. None of the five patients experienced cardiac symptoms prior or during hospitalization [5]. The only indication for cardiac disease in these 5 patients was elevated

Table 1
Seizures triggering TTS.

Cause of epilepsy	ST	AED at TTS	NOP	Sex	Age	TTST	TTSTr	OC	Reference	FP/AB
Nm	nm	nm	5	f	49–78	nm	nm	FR	[5]	FP
Nm	nm	nm	3	nm	nm	nm	nm	nm	[9]	FP
Nm	TCS	nm	3	f	43–80	Classic	nm	FR	[10]	FP
Nm	SE	nm	3	2f, 1 m	18–47	Global	nm	nm	[11]	FP
Stroke, alcohol, amitryptillin	nm	nm	3	2f, 1 m	40–73	nm	nm	FR	[6]	FP
Nm	nm	nm	2	f	41 + 78	Classic	nm	FR	[12]	FP
Nm/psychosis	TCS	PHT/LEV, CBZ	2	f	63/50	Classic	BB, ACEI	FR	[13]	FP
SAB/ICB	TCS	nm	2	1 m, 1 f	50 + 62	Mid	HFT	FR	[14]	FP
Nm	nm	nm	1	nm	nm	nm	nm	nm	[15]	AB
Nm	nm	nm	1	f	73	Classic	None	FR	[16]	FP
Previous stroke	nm	VPA	1	f	68	Classic	nm	na	[17]	AB
Bleeding right frontotemporal	sTCS	PHT, LEV	1	f	55	Classic	Stent	FR	[18]	FP
Genetic	CPS	ZNS, LMT	1	f	19	nm	None	nm	[7]	FP
Previous stroke	TCS	PHT	1	f	44	Classic	ACEI	FR	[19]	FP
Genetic	SE	None	1	f	47	Classic	nm	FR	[20]	FP
Alcohol withdrawal	nm	LEV	1	f	57	Classic	HFT	FR	[21]	FP
Genetic	TCS	CBZ, VPA, PRM	1	f	67	Classic	None	FR	[22]	FP
Nm	TCS	None	1	f	50	Classic	BB, ACEI	FR	[23]	FP
Cryptogenic, left temporal	SE	VPA, ZNS, DZP	1	f	43	Classic	None	FR	[24]	FP
Nm	TCS, SE	CBZ	1	f	68	Classic	None	FR	[25]	FP
Subarachnoid bleeding	TCS	None	1	f	60	Classic	None	FR	[26]	FP
Nm	SE	LEV, CLB	1	f	79	Classic	None	FR	[27]	FP
Na	SE	na	1	f	50	Classic	nm	FR	[28]	AB
Trauma	SE	nm	1	m	50	Classic	None	FR	[29]	FP
Alcohol	TCS	None	1	m	63	Classic	None	FR	[30]	FP
Genetic	TCS	None	1	f	69	Classic	nm	FR	[31]	AB
Stroke	nm	nm	1	m	78	Classic	nm	Death	[32]	FP
Anesthesia	NES	na	1	f	67	Mid	nm	FR	[33]	AB
Previous stroke	TCS	None	1	f	67	Classic	None	FR	[34]	FP
Genetic	TCS	VPA	1	f	81	Classic	nm	FR	[35]	FP
Na	na	na	1	f	59	nm	nm	nm	[36]	AB
Subdural hematoma	CPS	CBZ	1	m	73	nm	nm	nm	[37]	AB
Nm	TCS	None	1	f	50	Classic	nm	FR	[38]	AB
Cavernoma bleeding	TCS	None	1	f	74	Classic	None	FR	[39]	FP
Right mesial temporal sclerosis	sTCS	CBZ, PHT, ZNS, TPM	1	f	51	Classic	None	FR	[40]	FP
Alcohol	nm	None	1	f	25	Classic	nm	FR	[41]	AB
Nm	nm	nm	1	f	75	Mid	nm	nm	[42]	FP
Cavernoma mesotemporal	TCS	GBT	1	f	71	Classic	None	Death	[43]	FP
Na	Na	na	1	f	42	Mid	nm	FR	[44]	AB
Stroke	CPS	nm	1	f	75	Classic	nm	FR	[45]	FP
Nm	nm	nm	1	f	79	Classic	nm	FR	[46]	FP
Opiate withdrawal	TCS	None	1	f	58	Classic	BB	FR	[47]	FP
PRES	SE	None	1	f	82	Classic	None	FR	[48]	FP
Nm	nm	nm	1	m	57	Classic	Dobutamine	FR	[49]	FP
Electroconvulsive therapy	TCS	None	1	f	71	Classic	None	FR	[50]	FP
Nm	nm	nm	1	f	77	Mid	nm	FR	[51]	FP
Ischemic stroke	TCS	nm	1	f	62	Classic	ACEI, DR	FR	[52]	FP
Ischemic stroke	TCS, SE	nm	1	f	54	Classic	ACEI, DR	FR	[53]	FP
Genetic	TCS, SE	nm	1	m	62	Mid	None	FR	[54]	FP
Genetic	TCS, SE	DZP, PHT, VPA	1	f	75	Classic	Nitro	FR	[55]	FP
PRES	TCS	None	1	f	55	Classic	None	FR	[56]	FP
Mesiotemporal sclerosis	TCS, SE	CBZ, LEV	1	f	61	Classic	BB, DR	FR	[57]	FP
Genetic	TCS	CBZ, PHT	1	f	64	Classic	BB, DR	FR	[58]	FP
Multiple sclerosis	TCS	None	1	f	69	Classic	None	FR	[59]	FP
Astrocytoma surgery	TCS, SE	na	1	f	59	Classic	nm	FR	[60]	AB
Alcohol withdrawal	na	na	1	f	49	Classic	nm	FR	[61]	AB
Genetic	TCS	None	1	f	82	Classic	None	FR	[62]	FP
Left frontal hypodensity	nm	nm	1	m	39	Inverted	nm	FR	[63]	FP
Nm	TCS	nm	1	f	67	Classic	nm	FR	[64]	FP

AED, antiepileptic drug; ST, seizure type; NOP, number of patients; TTST, TTS-type; TTSTr, treatment of TTS; OC, outcome; FP/AB, full paper or only abstract available; SAB, subarachnoid bleeding; ICB, intracerebral bleeding; PRES, posterior reversible encephalopathy syndrome; CPS, complex partial seizures; (s)TCS, (secondary) tonic clonic seizure; SE, status epilepticus; NES, non-epileptic seizure; f, female; m, male; FR, full recovery; IR, incomplete recovery; *, epileptic state; nm, not mentioned; na, not accessible; CLB, clobazam; DZP, diazepam; PHT, phenytoin; CBZ, carbamazepine; VPA, valproic acid; LEV, levetiracetam; LMT, lamotrigine; ZNS, zonisamide; PRM, primidone; TPM, topiramate; BB, beta-blockers; ACEI, angiotensin converting enzyme inhibitors; HFT, heart failure therapy; DR, diuretics.

troponin, why further cardiologic investigations were carried out, revealing TTS. The first paper about seizure-triggered TTS was provided by Sharkey et al. in 1998. In a series of 22 patients with TTS, three had generalized seizures prior to the development of TTS [6]. Two of them were female, age ranged between 40 and 73 y, and all had a favorable outcome. Unfortunately, no information about the cause and treatment of epilepsy and the TTS type were provided [6]. Among all 74 patients with seizure-triggered TTS so far reported, age was reported in 70 patients, ranging from 18 to 82 y. Gender was reported in 70 cases and was female in 60 (86%) and male in 10 (14%) cases. The type of triggering seizure was reported in 47 cases. In 28 patients (60%) the trigger was a generalized tonic clonic seizure (TCS), in 15 cases (32%) a generalized status epilepticus, and in 3 cases a complex partial seizure (Table 1). Data about the antiepileptic medication prior to TTS were provided for 18 patients. In some studies it was unclear if the given AED was taken already prior to TTS or prescribed after the triggering seizure. The type of TTS was reported in 59 patients, of which 48 (81%) presented with the classical type, 7 with the midventricular type, 1 with the inverted (inverse) type, and 3 with the global type (Table 1). Treatment for TTS was given in 15 cases (Table 1). No treatment for TTS was provided in 19 cases. Only 1 of those who received no treatment died. The outcome was reported in 63 of the 74 patients so far published. Full recovery of TTS was reported in 61 cases (97%), incomplete recovery in none of the patients, and a fatal outcome in 2 patients (3%).

4. Discussion

This mini-review shows that TTS is increasingly recognized as a possible complication of generalized seizures. Diagnosing TTS in patients after a seizure is essential since treatment of TTS may improve the outcome of affected patients. However, of the 19 patients who did not receive treatment only one died and of the 14 patients who received treatment, none died. Though it is speculated that TTS is one possible mechanism to explain sudden unexpected death in epilepsy (SUDEP), the present review argues against a strong causal relation. Only 3% of the patients with seizure-triggered TTS died during or after the event. In 97% of the cases the outcome of seizure-triggered TTS was favorable, irrespective if TTS was treated or not, suggesting that TTS without complications is not responsible for SUDEP in a significant number of cases. Only if TTS is complicated by non-sustained ventricular arrhythmias or asystole it is conceivable that TTS secondarily causes SUDEP. More frequently than uncomplicated TTS, ventricular arrhythmias or a pulmonary complication may cause SUDEP. A further argument against a strong role of TTS in the pathomechanism of SUDEP is that TTS generally carries a favorable outcome, disregarding the underlying trigger. Only in patients with subarachnoid bleeding the outcome of TTS is fatal in 34%, and thus higher compared to patients with seizure-triggered TTS, possibly due to the increased mortality of the underlying neurologic disease.

In conclusion this minireview shows that seizure-triggered TTS has been reported in 74 cases since its first description in 1998 [6]. No information about the prevalence of TTS among epilepsy patients has been provided so far. Age at occurrence of TTS ranges from 18 to 82 y [7,8]. There is a striking female preponderance. All types of TTS may be triggered by seizures but the classical type prevails. The outcome is favorable in 97% of the cases, irrespective of treating or not treating TTS, making it rather unlikely that TTS plays a strong role in the pathomechanism of SUDEP. It appears that patients with generalized seizures do not need to receive beta-blockers for prophylactic reasons to prevent TTS.

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Conflict of interest statement

The authors declare that there are no conflicts of interest.

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