Takotsubo cardiomyopathy

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Takotsubo cardiomyopathy, also known as transient apical ballooning syndrome, apical ballooning cardiomyopathy, stress induced cardiomyopathy, Gebrochenes-Herz-Syndrom, is a type of non-ischemic cardiomyopathy in which there is a sudden temporary weakening of the myocardium.

Because this weakening can be triggered by emotional stress, such as the death of a loved one, a break-up, or constant anxiety, it is also known as broken-heart syndrome.

It is a poorly understood condition associated with periods of emotional and physical stress. The clinical approaches for management of SIC are supportive and reactive to patient symptoms.

Types

There are two major types of takotsubo cardiomyopathy: the classical type with left ventricular apical ballooning and a type with midventricular ballooning. Both types show different electrocardiographic patterns at presentation.

Etiology

Takotsubo cardiomyopathy is a complication of cardiac dysfunction after stroke, including subarachnoid hemorrhage (SAH).

The onset of the disease occurs early in the course of aSAH and an elevated BNP and troponin may be associated with the onset of NCM. Cardiac function often remains impaired during the acute recovery phase potentially impeding resuscitation during this period. The routine use of short term follow-up echocardiography may be recommended [1)].

A sudden surgical pain stimulus combined with insufficient analgesia are hypothesized to probably cause a catecholamine surge in Takotsubo cardiomyopathy [2)].

High circulating levels of catecholamines seem to cause catecholamine-induced microvascular spasms with subsequent myocardial dysfunction and stunning [3].

However, elevated blood catecholamine levels are not always present [4].

Kalani et al performed exome sequencing of 7 white female patients. Filtering of the identified variants was performed to limit the investigation to those sequences that passed quality control criteria, were rare or novel, were determined algorithmically to have high impact on the associated protein, and were within regions of high species conservation. All variants were verified by using Sanger sequencing.

Exome-sequencing analysis revealed that each patient carried predicted deleterious variants affecting known cardiomyopathy genes. In each case, the identified variant was either not previously found in public human genome data or was previously annotated in a database of clinical variants associated with cardiac dysfunction.

Patients harbor deleterious mutations in established cardiomyopathy genes at a level higher than healthy controls. They hypothesize that patients at highest risk likely live in a compensated state of cardiac dysfunction that manifests clinically only after the myocardium is stressed. In short, they will propose that this is another example of an occult cardiomyopathy with a distinct physiological trigger and suggest that alternative clinical approaches to these patients may be warranted [5].

Clinical Features

The clinical presentation is similar to that of an acute myocardial infarction.

Symptoms are acute substernal chest pain, dyspnea, syncope, shock, or electrocardiographic abnormalities. In contrast to these clinically impressive symptoms, further diagnostics cannot confirm a coronary syndrome with the signs of coronary occlusion and ischemic lesions. The onset of Takotsubo can be triggered by emotional stress or constant anxiety and unexpected catastrophic conditions [6] [7]

Diagnosis

Clinicians should suspect takotsubo cardiomyopathy in patients with subarachnoid haemorrhage who have an ECG abnormality. Echocardiography is needed to detect the distinctive regional wall motion abnormality. [8].

Takotsubo is characterized by ST segment elevations especially in the anterior precordial leads, deep T wave inversions or abnormal Q waves [9].

Classical electrocardiographic findings of the acute and subacute phases of takotsubo cardiomyopathy are descibed by Bonnemeier [10].

Differential diagnosis

Stress-induced cardiomyopathy may mimic myocardial infarction and is an important condition to recognize in patients with underlying stress states, particularly neurologic injuries [11].

Treatment

There are no guidelines regarding the optimal treatment of subarachnoid hemorrhage (SAH) patients complicated by Takotsubo cardiomyopathy (TCM).

There are no controlled data to define an optimal medical regimen, but it seems reasonable to treat these patients with standard medications for left ventricular systolic dysfunction. These include ACE inhibitors, β-blockers and diuretics to prevent volume overload. However, because the condition may recur, on-going adrenergic blockade with β-blockers is suggested. Consequently, Takotsubo is a transient disorder managed with the resolution of physical or emotional stress and supportive therapy. Due to the personal needs in case of elective neurosurgery and the possibility of neurogenic mechanisms in triggering of Takotsubo, surgical interventions and perioperative care should be realized under stress-free conditions as far as possible [12].

Neurosurgeons treating SAH must take into account the various general treatment strategy options on a case by case basis after prompt recognition [13].

Despite its severity in the acute phase, takotsubo cardiomyopathy is self-limiting and its management is conservative [14].

Treatment methods in Takotsubo cardiomyopathy that use extracorporeal circulatory support and medications that do not rely on β-receptor stimulation and preemptive blockade of β receptors or calcium channels before brain death may be relevant to donor care [15].

Outcome

Stress cardiomyopathy is a well-recognized cause of acute heart failure, lethal ventricular arrhythmias, and ventricular rupture.

Patients who survive the acute episode typically recover to normal ventricular function within four weeks. The prognosis is determined by the acute complications of Takotsubo. Despite the severity of the acute illness, Takotsubo is a transient disorder manageable with supportive therapy [16].

In some the condition proves fatal. Seizure-associated takotsubo cardiomyopathy may cause sudden unexplained death in epilepsy (SUDEP), and EEG measurements should be carefully checked for seizure patients [17].

Case series

2016

Between January 2008 and December 2014, 371 consecutive patients with aneurysmal SAH underwent transthoracic echocardiography after admission, and 30 with TCM (7.7 %) were identified. We reviewed the incidence and type of perioperative complications among clipped (n = 11) and coiled (n = 19) patients. The 30 patients were dichotomized based on their 90-day modified Rankin scale (mRS) scores into favorable (mRS: 0-2) and unfavorable (mRS: 3-6) groups, and their demographic, laboratory and echocardiographic variables were compared.

Neither clipped nor coiled patients developed serious perioperative cardiopulmonary complications, but coiled patients had a higher incidence of fatal procedure-related complications. Among the 30 patients, 13 (43 %) had favorable 90-day outcomes, and the favorable group was significantly younger. Age, but not the degree of cardiac dysfunction, correlated with outcomes by multivariate regression analysis.

Clipping was shown to be a safe treatment modality in our cohort, and treatment selection may better be made on a case-by-case basis in most patients with SAH-induced TCM. The lack of correlation between the degree of cardiac dysfunction and outcomes indicates that aggressive intervention is justified in patients with severely impaired cardiac function [18].

2015

In a retrospective institutional chart review of 800 patients with aneurysmal SAH from 2007 to 2014. Eighteen patients were identified to have both aneurysmal SAH and TCM based on echocardiogram. Demographic data, clinical parameters, radiographic findings, treatment modalities, and laboratory results were analyzed.

The incidence of typical TCM in our patients was 2.2%. Mortality rate of TCM in SAH was 22% compared to the total mortality rate of all non-traumatic SAH patients of 15% in our institution over the same time period. Use of beta blockers prior to or after the diagnosis of TCM did not seem to affect their outcome. Majority of patients (61%) were on vasopressors prior to the diagnosis of TCM. Of those, 73% had good outcomes. Even after the diagnosis of TCM, good outcomes were observed in 6 of 7 patients who remained on vasopressors.

Despite the general agreement on the importance of the avoidance of vasopressors in TCM, our experience showed that the use of vasopressors is safe in these patients. The use of beta blockers in our patients was not associated with significantly better outcomes despite multiple previous reports on beta blocker usage in TCM [19].

Case reports

a rare case of a patient who was found to have a subarachnoid hemorrhage that incited the development of Takotsubo cardiomyopathy, which subsequently progressed to an acute myocardial infarction. The aim of this case report is to explore the underlying pathophysiology of how cerebral hemorrhage can result in apical ballooning of the left ventricle through various mechanisms including sympathetic-induced surge in catecholamines and neurogenic damage to the myocardium. We also intend to highlight the importance for clinicians to consider brain bleeds in the differential diagnosis when a patient presents with an acute myocardial infarction as treatment with heparin is generally contraindicated [20]

2017

A 69-year old female patient was admitted to the neurosurgical department and scheduled for elective resection of a cerebellar metastasis in sitting position. After craniotomy and opening of the posterior fossa a venous air embolism was detected via transesophageal echocardiography. Immediately, the patient presented a cardiac decompensation with signs of TakoTsubo or stress-induced cardiomyopathy.

Being aware of stress induced cardiomyopathy as a probably underdiagnosed disease entity should be mandatory to the intensivists and anesthesiologists in the operating room and on intensive care units, especially since its management will differ significantly from other forms of cardiogenic shock. Diagnosis can quickly be approached by bedside echocardiography emphasizing the need for availability of this tool and the integration in diagnostic algorithms on the ICU [21)].

2015

A case of a 53 year-old female with a spinal neurinoma and surgery-associated Takotsubo cardiomyopathy is demonstrated. The patient developed typical signs of a myocardial infarction with circulation depression and ST elevation, but normal cardiac enzymes at the end of surgery. Cardiac catheterization and levocardiography confirmed the absence of any critical coronary disease but the presence of a typical apical ballooning and midventricular hypokinesis. The patient recovered completely under supportive conservative and cardiological therapy, showing regular left ventricular pump function [22)].

A 61 years old female patient (height 1.65 m; weight 70 kg) presented with a haemorrhagic pituitary neuroendocrine tumor with compression of the optic chiasm and was scheduled for transnasal endoscopic tumour resection. We report a case series with five consecutive anaesthesia procedures in the same patient for neurosurgery. This case series is remarkable since the severe symptoms occurred during every anaesthesia procedure. The female patient was resuscitated two times including therapeutic hypothermia, but fortunately no neurological or cognitive deficit was detectable. CONCLUSIONS: TTC may initially present in the perioperative period with pulmonary oedema, electrocardiographic (ECG) changes, elevation of cardiac enzymes, and cardiogenic shock or cardiac arrest. Since the risk of recurrence is considered to be low in TTC, this case report is of high interest. In each procedure similar clinical signs were found which resulted in severe haemodynamic derangements in every manifestation and cardiac arrest in two of the manifestations. Despite cardiopulmonary resuscitation twice, the patient survived without any neurological deficiency [23)].

2014

A 68-year-old woman was admitted for symptomatic seizure. She had a history of cerebral infarction in the right fronto-temporal lobes, and was medicated for the symptomatic seizure with valproic acid. Her electrocardiogram(EEG)showed ST-segment elevation in leads II, III, aVF, and V2-V6. Emergency coronary angiography showed normal coronary arteries, however, left ventriculography showed apical ballooning in the systolic phase. She had no chest pain or dyspnea, and takotsubo cardiomyopathy was diagnosed due to ECG abnormality [24)].

A 67-year-old woman was admitted with aneurysmal subarachnoid haemorrhage and a 12-lead ECG showed ST segment elevation. Transthoracic echocardiography confirmed akinesis of the left ventricular mid-apical segment, with an ejection fraction of 26%, features characteristic of takotsubo cardiomyopathy. Five days later, we identified thrombus in the apex of the left ventricle. Sixteen days after onset, the thrombus had disappeared and wall motion improved (ejection fraction 58%) without evidence of cardioembolism [25].

2011

A 90-year-old woman presented with aneurysmal subarachnoid hemorrhage (SAH) corresponding to Hunt and Hess grade II. Acute congestive heart failure and pulmonary edema developed following uneventful surgical clipping. Serial electrocardiography and echocardiography led to a diagnosis of neurogenic stress cardiomyopathy (NSC), also known as tako-tsubo cardiomyopathy. The outcome was favorable after supportive therapy with respiratory management and diuretic administration [26].

The first case of neurogenic stunned myocardium presenting with heart left ventricle noncompaction requiring intensive care in the perioperative period of tension tumor-induced hydrocephalus.

A 12-yr-old female with intracranial astrocytoma and hypertensive hydrocephalus presented with severe heart dysfunction and life-threatening ventricular ectopies intraoperatively. A severe heart failure developed requiring hemodynamic and ventilatory support for 10 days. Echocardiography showed a transient noncompaction aspect of the left ventricular wall, further confirmed by a cardiac magnetic resonance image. The noncompaction aspect lasted until 15 days postadmission, as was the case for the QT interval prolongation; no life-threatening ectopies were demonstrated on the subsequent Holter electrocardiogram monitoring.

This report describes a unique presentation of myocardial stunning in association with an intracranial illness, namely, a hypertensive hydrocephalus complicating an intracranial neoplasm [27].

2010

A 65-year-old female with subarachnoid hemorrhage (SAH) developed takotsubo cardiomyopathy induced by dobutamine infusion for vasospasm 9 days after onset of SAH. She underwent neck clipping of the ruptured cerebral aneurysm on day 1. Course after surgery was uneventful, but she developed motor aphasia on day 9. Hypertensive therapy was carried out under the diagnosis of symptomatic vasospasm. Half an hour after initiation of dobutamine infusion at 6 microg/kg/min, sudden symptoms of takotsubo cardiomyopathy developed. Fortunately, her symptoms recovered in a few days with supportive therapy without any consequences. Takotsubo cardiomyopathy is one pattern of cardiac dysfunction occasionally encountered after SAH. Possible mechanisms of this disorder include epicardial catecholamine cardiotoxicity. Therefore, generally, cardiac function is worst at the early stage of SAH, when sympathetic activity is highest, and recovers thereafter. Dobutamine infusion seems to have triggered the takotsubo cardiomyopathy in the present patient even 9 days after onset of SAH. Inotropic agents including dobutamine are often used during the course of SAH, and since takotsubo cardiomyopathy can occur in patients with SAH, this complication

must be considered [28].

Gologorsky and Gologorsky present the case of a 43 year-old female that developed the signs of Taksotsubo intraoperatively during lumbar interbody fusion [29].

2004

A 66-year-old woman presented with subarachnoid hemorrhage(SAH) caused by a ruptured aneurysm of the left middle cerebral artery. Electrocardiography (ECG) disclosed abnormalities resembling acute myocardial infarction. She underwent neck clipping of the aneurysm uneventfully. Sixteen days after admission, ECG again disclosed abnormalities resembling acute myocardial infarction, and echocardiography suggested heart failure. Coronary angiography showed no abnormalities, but left ventriculography showed severe hypokinesia in the apex of the heart consistent with so-called ampulla (takotsubo) cardiomyopathy. The heart failure was treated with catecholamines and her heart function gradually recovered. Ampulla (takotsubo) cardiomyopathy associated with SAH requires careful management of heart function [30].

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