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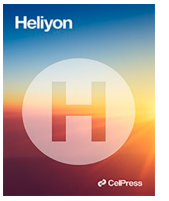

Case report

Subarachnoid hemorrhage with Takotsubo syndrome as the prominent manifestation: A case and literature review

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ABSTRACT

Background: Takotsubo syndrome, which is often induced by physical or psychological stress, is typically a cardiac syndrome with transient left ventricular dysfunction in the absence of obstructive coronary artery disease. Subarachnoid hemorrhage with typical symptoms and signs is frequently reported, whereas the incidence of subarachnoid hemorrhage with Takotsubo syndrome as the prominent manifestation without a typical headache is rarely reported.
Case description: We present a rare case of a 63-year-old male patient with cough and fever as the first manifestations, accompanied by mild dizziness, headache, and mental discomfort; however, the patient was eventually diagnosed with atypical subarachnoid hemorrhage with Takotsubo syndrome. The patient underwent general anesthesia downwards stent-assisted spring coil embolization and was discharged from the hospital after postoperative treatment consisting of anti-cerebrovascular spasm, anti-platelet aggregation, and cerebrospinal fluid replacement.
Conclusion: This case demonstrates the association between Takotsubo syndrome and subarachnoid hemorrhage. When patients present with unexplained pulmonary edema with mild neurologic symptoms, clinicians should be alerted to subarachnoid hemorrhage and Takotsubo syndrome.

1. Introduction

Subarachnoid hemorrhage (SAH) is a type of stroke caused by bleeding into the subarachnoid space after the rupture of blood vessels at the base or surface of the brain, and it accounts for approximately 5–10 % of all strokes. The main feature of SAH is a severe headache that starts suddenly [1]; in addition, other accompanying neurological symptoms and signs include an altered level of consciousness, cranial neuropathy, focal weakness, and meningitis [2]. SAH with typical symptoms and signs is frequently reported, and the incidence of SAH with Takotsubo syndrome (TS) as the prominent manifestation without a typical headache is rarely reported. As clinicians generally lack knowledge about the atypical manifestations of SAH, to avoid misdiagnosis and delay of the disease, a case

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of SAH with TS as the prominent manifestation was reported below, and a literature review was subsequently conducted.

2. Case presentation

A 63-year-old male patient was admitted to the Emergency Department due to a cough and low fever for one day. One day before admission, the patient experienced paroxysmal coughing without an obvious cause, accompanied by a small amount of thick white sputum, chills, and a fever, with a maximum temperature of 37.9 °C.In addition to cough and fever, the patient also had dizziness, general pain (Including mild headache, and continuous throbbing pain at the top of the head, without symptoms such as dizziness, nausea, or vomiting, and no apparent aggravating or relieving factors.), and poor appetite. Previous medical history included hypertension (with the highest blood pressure measurement being 165/95 mmHg), and disease control was unknown. He had not yet received any treatment. Chest computed tomography (CT) showed bronchial disease and multiple exudates in both lungs (A,B,C in Fig. 1). He was admitted to the Department of Respiratory Medicine. Physical examination on admission: T: 37.3 °C, P: 70 bpm, R: 22 bpm, BP: 125/75 mmHg, and SPO_2: 98 % (breathing air). The patient was acutely ill-looking and had a clear mind, spontaneous position, no cyanosis of the lips, no deflection of the angle of the mouth, soft neck with no resistance, moist rales in both lower lungs, a heart rate of 70 bpm, regular heart rhythm with no murmur, soft abdomen, no swelling of both lower extremities, and negative pathological signs.

After admission, the patient received anti-infective, expectorant, and other symptomatic and supportive treatments. Laboratory tests (Tables 1 and 2) were characterized by mildly elevated white blood cell, C-reactive protein, and neutrophil percentages, as well as varying degrees of elevated myocardial enzymes, significantly elevated D-dimer and N-terminal prohormone of brain natriuretic peptide (NT-proBNP), and mildly decreased potassium. Bedside electrocardiogram (ECG) showed a sinus rhythm with a prolonged QT interval (A in Fig. 2). With highly suspected acute myocardial infarction, the patient was orally administered a loading dose of aspirin (300 mg) and clopidogrel (300 mg) for anti-platelet aggregation and given a secondary preventive treatment for coronary heart disease. However, myocardial enzyme results (Table 2) and ECG (B and C in Fig. 2) were not consistent with the dynamic changes characteristic of acute myocardial infarction on several retests during the observation period. Echocardiography was performed and showed an ejection fraction (EF) of 65 %, mitral and tricuspid regurgitation, decreased left ventricular compliance, and normal systolic function (Fig. 4). During the entire treatment process, the patient did not complain of chest tightness, chest pain, or pressure in the precordial area, but he still had a persistent mild headache and a gradually deteriorated attitude. As nonneurologists, we first considered whether the patient had an acute cerebral infarction. Due to the presence of few or atypical clinical symptoms and the relatively low sensitivity of CT scans, many cerebellar infarctions may only be detected with MRI [3]. We decided to perform the patient's head MRI examination, and the results suggested intracerebral hemorrhage(A,B in Fig. 3); therefore, the Neurology Department was invited for consultation, and physical examination showed a BP of 123/77 mmHg, an HR of 60 bpm, a RR of 16 bpm, a Glasgow Coma Scale (GCS) score of 15, clear mind and fluent language, equal roundness and same size of both pupils, normal eye movements, suspected positive cervical resistance, normal muscle strength and muscle tone of extremities, Chaddock sign (+),

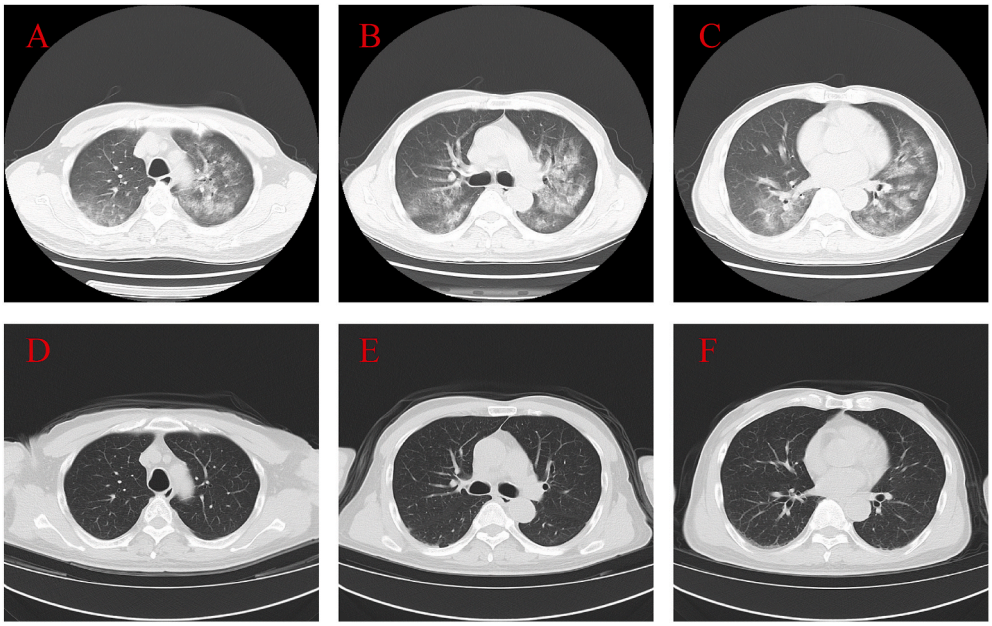

Fig. 1. Pulmonary Imaging
A, B, and C represent chest CT scans at admission, which show exudative lesions in both lungs; D, E, and F represent chest CT scans after five days, which show that the multiple exudative lesions in both lungs were markedly absorbed.

Table 1
Laboratory test.

List	Standard	Value
Blood gas analysis (Breath in the air)		
pH	7.35–7.45	7.445
$PaCO_2$ (mmHg)	34–45	28.1
PaO_2 (mmHg)	80–100	71.6
SaO_2 (%)	92–99	95.5
Blood routine		
WBC (10^9/L)	4–10	11.35
N (%)	40–70	89.4
L (%)	20–40	5.6
CRP (mg/L)	0–6	18.92
TBIL (umol/L)	3.4–20.6	32.3
DBIL (umol/L)	0–8.6	10.1
K^+ (mmol/L)	3.5–5.3	3.39
D-D (ng/ml)	0–500	3680
PCT (ng/ml)	0–0.10	0.091
ESR (mm/h)	0–15	5.85
NT-proBNP (pg/ml)	0–250	6700.12

Abbreviations: $PaCO_2$, partial pressure of carbon dioxide in the artery; PaO_2, partial pressure of oxygen in the artery; SaO_2, arterial oxygen saturation; WBC, white blood cell; N, neutrophil; L, lymphocyte; CRP, C-reactive protein; TBIL, total bilirubin; DBIL, direct bilirubin; D-D, D-dimer; PCT, procalcitonin; ESR, erythrocyte sedimentation rate; NT-proBNP, N-terminal prohormone of brain natriuretic peptide.

Table 2
Myocardial enzyme test.

List	Standard	Admission (Value)	Retest after 2 h (Value)	Retest after 11 h (Value)	Retest after 5 d (Value)
CK (U/L)	0–190	357	341	447	94
CK-MB (U/L)	0–24	31	25	21	17
cTnI (ng/ml)	0–0.15	4.151	3.639	3.039	0.093

Abbreviations: CK, creatine kinase; CK-MB, creatine kinase isoenzyme; cTnI, cardiac troponin I.

Brudzinski sign (−), and Kernig sign (−) of the right lower extremity, and grade 1 on the Hunt-Hess scale. Moreover, a head CT scan (C, D in Fig. 3)and head and neck computed tomography angiography (CTA) showed the following measurements. 1. SAH was considered in the bilateral frontotemporal lobe, right parietal lobe, and tentorium of the cerebellum. 2. There was an aneurysm in the M2 segment of the left middle cerebral artery (E in Fig. 3). The patient was diagnosed with SAH and transferred to the stroke ICU. Emergency coronary angiography showed plaques in the proximal and middle segments of the left anterior descending artery, 85 % stenosis of the opening of the first diagonal branch, and TIMI grade 3 flow. Cerebral angiography showed an aneurysm in the M2 segment of the left middle cerebral artery (F in Fig. 3), which was considered the cause of this SAH; After completing coronary angiography with no significant coronary artery stenosis observed, and in accordance with the 2018 ESC international diagnostic criteria consensus, the patient was diagnosed with Takotsubo syndrome (TS) based on a comprehensive evaluation of clinical manifestations, physical signs, electrocardiogram, and laboratory investigations including cardiac enzyme studies. Subsequently, stent-assisted coil embolization was performed under general anesthesia, Postoperative angiography is shown in the G in Fig. 3. After the operation, the patient was treated with symptomatic and supportive treatments, such as nimodipine for anti-cerebral vasospasm, mannitol for dehydration, aspirin enteric-coated tablets and clopidogrel tablets for anti-platelet aggregation, and esomeprazole for stomach protection. After five days, the reexamination showed that routine blood tests, myocardial enzymes, NT-proBNP, and D-dimer values were normal. Moreover, the head CT showed the following results: 1. less SAH and intraventricular hemorrhage than was observed before treatment; 2. post-operative changes of the aneurysm in the M2 segment of the left middle cerebral artery. Furthermore, chest CT showed bronchial disease, and the multiple exudates in both lungs that were previously observed were markedly absorbed (D,E,F in Fig. 1); thus, the patient was discharged. The final diagnosis was as follows: 1. ruptured aneurysm in the M2 segment of the left middle cerebral artery with SAH (Takotsubo syndrome); and 2. grade 2 hypertension representing a high-risk group. Two months later, the patient came to the hospital again for aneurysmal subarachnoid hemorrhage (aSAH), but neither the ECG (D in Fig. 2) nor cardiac troponin I showed obvious abnormalities.

3. Discussion

SAH is a serious and life-threatening medical emergency that accounts for 5–10 % of all strokes. The average age of SAH patients is 55 years, and increasing age is an independent risk factor for SAH. Patients with SAH present with a sudden onset of severe headaches that are classically described as “the worst headache of my life” [1,4]. Some patients present with atypical symptoms of a lack of

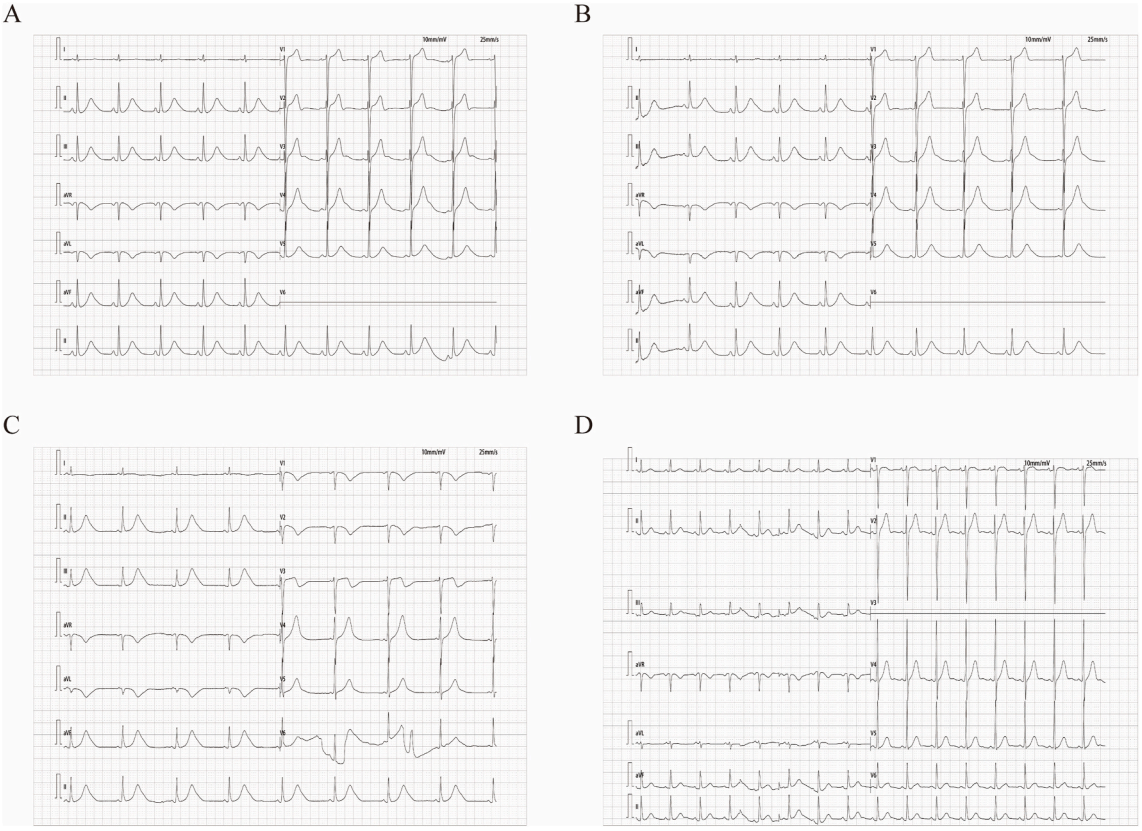

Fig. 2. Electrocardiogram
A, B, and C were ECGs at the time of the first hospitalization, which A was the ECG at the time of admission, B and C were the ECGs re-examined during treatment, all suggested QT interval prolongation; D was ECG on the second admission, showing normal ECG.

sudden headache and/or reduced consciousness, such as nausea or vomiting, dizziness or lightheadedness, and neck or back pain [4]. In this case, the patient was a 63-year-old male with respiratory symptoms as the most prominent manifestation and had a mild headache, dizziness, and poor mentality. He was finally diagnosed with a ruptured aneurysm in the M2 segment of the left middle cerebral artery with SAH by head and neck CTA and cerebral angiography, thus suggesting that the patient had SAH with atypical symptoms.

The most common cause of SAH is an intracranial aneurysm, which accounts for approximately 85 % of SAHs. Secondary complications in patients with an aneurysm are important causes of morbidity and mortality [5].

In addition to brain injury, there are also a variety of extracerebral complications, among which cardiopulmonary complications are the most common [5–7]. In recent years, TS secondary to SAH has gradually attracted the attention of clinicians. It has been reported that the prevalence of TS after aneurysmal SAH (aSAH) ranges from 0.8 % to 30 % [7–9]. Originally described by Sato et al., in 1990 [10], TS is a cardiac syndrome characterized by transient left ventricular dysfunction in the absence of obstructive coronary artery disease, which usually spontaneously recovers within days or weeks. TS was named because the shape of the left ventricle at the end of the systolic period is similar to the Japanese octopus pot (“tako-tsubo”) [11]. It is typically triggered by physical or psychological stress and is correspondingly referred to as ‘stress cardiomyopathy’ or ‘broken-heart syndrome’. The TS caused by neural stressors can also be referred to as neurogenic stunned myocardium and neurogenic stress cardiomyopathy, among other conditions [11]. Among neurogenic TSs, SAH is the most common etiology [11,12]. Studies have shown that approximately 90 % of TS patients are women aged 67–70 years [13]. A total of 78.5 % of Chinese TS patients are women, with an average age of approximately 60 years [14], thus suggesting that postmenopausal women are more likely to develop TS. In addition, it is speculated that this may be related to estrogen deficiency [13]. Male, atypical Takotsubo types and acute neurological and/or psychiatric disorders are more common in younger TS patients [15].

In this case, the patient was a relatively young (63-year-old) man with a TS that was induced by aSAH, which is similar to those reported in the literature. The pathogenesis of TS has not been fully elucidated, and it is currently believed to be closely related to coronary plaque rupture, epicardial vasospasm, microcirculation disturbances, and catecholamine myocardial toxicity caused by the hyperactivation of sympathetic nerves [16]. Some scholars have suggested that the mechanism of TS after SAH may be related to the degeneration of the vagal complex and the overactivity of sympathetic nerves [17].

The clinical features of TS may be similar to those of acute coronary syndromes, such as severe chest pain, retrosternal crushing

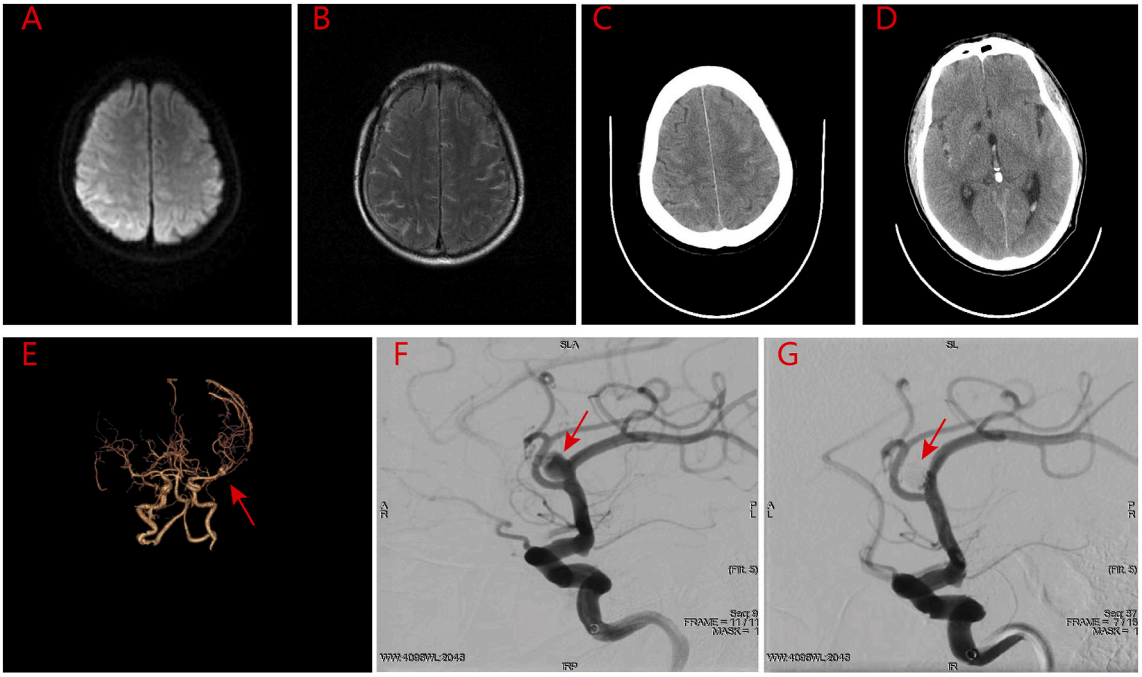

Fig. 3. Head Imaging
A and B represent MRI scans that showed no cerebral infarction but suspicious SAH; C and D represent nonenhanced head CT scans, which show SAH; E represents the head and neck CTA, which shows an aneurysm in the M2 segment of the left middle cerebral artery; F represents cerebral angiography before the stent-assisted coil embolization, which shows an aneurysm in the M2 segment of the left middle cerebral artery and was considered the cause of this SAH; and G represents angiography after the operation.

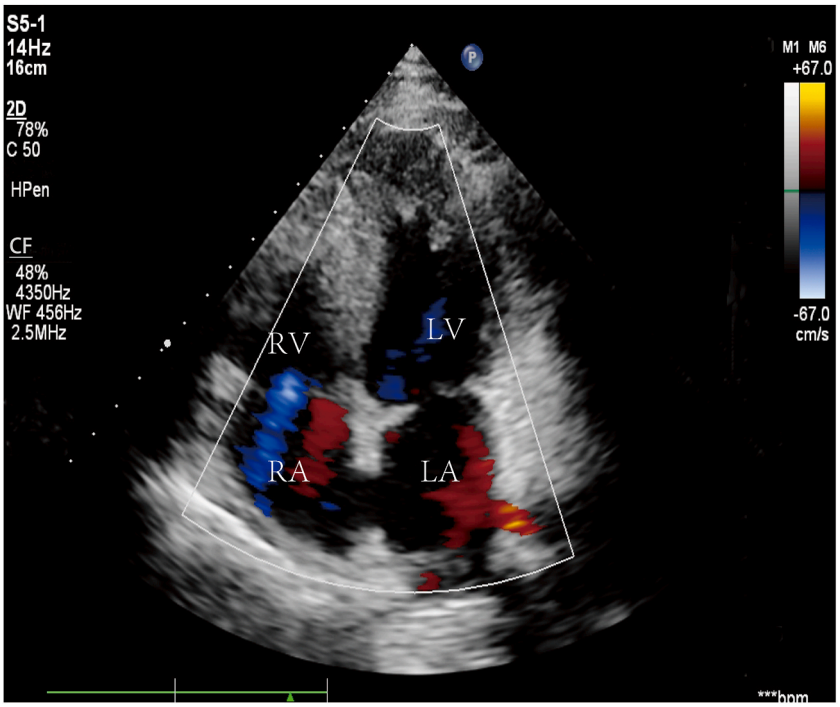

Fig. 4. Transthoracic echocardiogram
Transthoracic echocardiogram with a four-chamber view showing no typical anteroseptal apical dyskinetic "ballooning" of the left ventricle. LV, left ventricle; LA, left atrium; RV, right ventricle; RA, right atrium.

pain, dyspnea, and syncope; in addition, patients with these clinical features may have left ventricular dysfunction, abnormal ECG, and elevation of myocardial injury markers, whereas coronary angiography is generally normal [18]. Studies have shown that emotional stressors are more common in women, with patients presenting with chest pain and having a better prognosis; additionally, TS caused by physical stressors (such as infection, surgery, trauma, neurological disease, or hypoxia, etc.) are more common in men, with patients presenting with syncope and dyspnea [10]. The plasma troponin concentrations are usually elevated, but the peak is lower than in ST-segment elevation myocardial infarction; in addition, the concentrations of brain natriuretic peptide (BNP) and NT-proBNP in the plasma are usually much higher than in patients with myocardial infarction [10]. The most common ECG abnormalities are ST-segment elevation, T-wave inversion, and left bundle branch block. Previous studies have shown that the ECG of TS can evolve dynamically; specifically, ST-segment deviation occurs within the first few hours of the onset of symptoms. Subsequently, progressive T-wave inversion and QTc prolongation often occur within 1–3 days and reach a peak in 2–6 days. In the following weeks or months, the inverted T-wave and prolonged QTc gradually recover [10,19]. Echocardiography can be used to support the diagnosis (for determining the extent, severity, and location of abnormal ventricular wall motion) and to identify potential complications of TS; in addition, approximately 20 % of patients have evidence of left ventricular outflow tract obstruction [20].

The 2018 European Society of Cardiology (ESC) International Expert Consensus on TS Diagnostic Criteria include [21] the following criteria: (1) transient left ventricular dyskinesia presenting as apical ballooning or midventricular, basal, or focal wall motion abnormalities, with right ventricular involvement possibly being present and transitions between all types possibly existing; (2) an emotional, physical, or combined trigger can precede the TS event (but this scenario is not obligatory); (3) neurologic disorders (e.g., subarachnoid hemorrhage, stroke/transient ischemic attack, or seizures), as well as pheochromocytoma, may serve as triggers for TS; (4) new ECG abnormalities are present (ST-segment elevation, ST-segment depression, T-wave inversion, and QTc prolongation), but rare cases exist without any ECG changes; (5) levels of cardiac biomarkers (troponin and creatine kinase) are moderately elevated in most cases, and a significant elevation of NT-proBNP is common; (6) coronary angiography is generally normal, and significant coronary artery disease is not a contradiction in TS; (7) patients with no evidence of infectious myocarditis; and (8) postmenopausal women are predominantly affected. This patient was an elderly male with aSAH. The ECG showed a prolonged QT interval; moreover, the myocardial zymogram showed increased CK and cTnI without dynamic evolution and significantly increased NT-proBNP. Furthermore, emergency coronary angiography showed no significant coronary stenosis. There was no obvious evidence of infective myocarditis because he was not a young adult, and no conduction block or ventricular arrhythmia was found on the ECG. The erythrocyte sedimentation rate (ESR) and procalcitonin (PCT) were normal, and there was no history of cold or diarrhea before onset [22]. According to the 2018 ESC International Expert Consensus on Diagnostic Criteria, the patient was diagnosed with TS.

We summarised a clinical features table regarding other published cases of TS secondary to SAH (See Supplementary Table 3). We identified 28 published articles involving 31 patients. Among these articles, the majority of patients were female (accounting for 28 of the patients), with only three being male. Twenty-eight patients presented with typical neurological symptoms (i.e., headache, vomiting, collapse, and loss of consciousness, among other symptoms). In contrast to the literature, our patient did not present with typical neurological symptoms, which is noteworthy, especially for nonneurologists. Among the 31 patients with SAH, aSAH accounted for approximately 65 %. This patient was no exception, and an aneurysm was discovered in the M2 segment of the left middle cerebral artery by CTA. Most cases have varying degrees of cardiac enzyme elevation and new electrocardiographic changes (e.g., ST-segment abnormalities, T-wave inversions, and prolonged QT intervals). Our patient's presentation was consistent with the literature. All of the patients (100 %) were observed to have varying degrees of left ventricular dyskinesia by echocardiogram, left ventriculography, or coronary angiography. However, this patient's echocardiography showed an EF of 65 %, and left ventricular systolic function was within the normal range. There were no apical ballooning or midventricular, basal, or focal wall motion abnormalities, which was considered to be related to the longer onset time before the echocardiography was performed because the wall motion abnormalities in patients with TS usually return to normal within hours to weeks [23]. In addition, the left ventricular EF of TS patients can be rapidly normalized; moreover, early cardiac imaging is required to detect such abnormalities [10].

Due to the fact that there are many similarities between the clinical features of TS and acute myocardial infarction, the disease is prone to be initially misdiagnosed as acute myocardial infarction [21]. In this case, the abnormal elevation of myocardial enzymes after admission was also misdiagnosed as acute non-ST-elevation myocardial infarction in combination with the ECG results, which is similar to those reported in the literature. Developed in the community, the patient presented with fever, cough, white viscous sputum, and moist rales in both lower lungs, and chest CT showed exudative lesions in both lungs. On admission, the patient was initially diagnosed with community-acquired pneumonia. However, the clinical diagnosis of community-acquired pneumonia can only be established after the exclusion of noninfectious factors, such as pulmonary edema [24]. In this case, there were physical factors (such as aSAH), and NT-proBNP was significantly increased. After diuresis and active treatment of the primary disease, the lesions in both lungs were significantly absorbed in a short period (within 5 days), thus suggesting that the pulmonary lesions were considered to be pulmonary edema rather than pulmonary infection. The patient's fever may be related to damage to the hypothalamus, midbrain, or pons caused by aSAH, thus resulting in thermoregulation disorders, acute or delayed cerebral ischemia caused by intracranial vasospasm, and stimulation of haem degradation products [25]. After admission, a routine blood examination of the patient showed increased white blood cells (WBCs) and C-reactive protein (CRP), which may be related to the massive secretion of catecholamines in the TS patient [26].

At present, there is no strong evidence-based medicine (EBM) for the treatment of TS, and such treatment is mainly based on clinical experiences and expert consensus [10]. In the acute phase, symptomatic and supportive treatment is the primary treatment. Diuretics and nitrates may be used (as appropriate) in TS patients with pulmonary edema. For aSAH, etiological treatment should be performed as soon as possible, and embolization is recommended as the first choice according to guidelines to improve the long-term functional prognoses of patients [27]. After the patient was confirmed to have aneurysm SAH, stent-assisted coil embolization was immediately

performed under general anesthesia, and the patient was treated with anti-cerebral vasospasm, anti-platelet aggregation, and cerebrospinal fluid replacement after the operation, after which his condition improved, and he was discharged.

Therefore, for clinical cases with elevated myocardial enzymes but dynamic evolution that is not consistent with acute myocardial infarction, TS should be excluded, and coronary angiography can be used to assist in the diagnosis. In diagnosing TS patients, it is necessary to actively identify the precipitating factors. If there are no psychological factors, it is necessary to investigate physical factors, especially for male patients. In clinical practice, any acute headache (regardless of its severity or previous history) should be suspected of SAH [2], especially in the presence of unexplained pulmonary edema with mild neurological symptoms. We should be mindful of SAH and complete head CT and cerebrospinal fluid examinations in a timely manner [1] to avoid misdiagnosis and delays in diagnosis and treatment.

4. Conclusion

Takotsubo syndrome is closely associated with subarachnoid hemorrhage.

Subarachnoid hemorrhage should be considered when patients present with unexplained pulmonary edema with mild neurological symptoms.

CRediT authorship contribution statement

Shihong Qin: Writing – original draft, Data curation. **Huifang Teng:** Writing – review & editing, Resources. **Aiping Li:** Writing – review & editing, Resources. **Lile Wang:** Writing – original draft, Data curation. **Ruicheng Hu:** Visualization. **Daiyan Fu:** Methodology, Conceptualization.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:Daiyan Fu reports financial support was provided by Changsha Science and Technology Program Project (No. kq2004112) and Key Project of Hunan Provincial Department of Education (No. 20A298). If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2024.e30057.

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