

# Tako-Tsubo Syndrome in a Patient with Sudden Visual Loss: Case Report

## Ani Görme Kaybı Gelişen Bir Hastada Tako-Tsubo Sendromu

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**ABSTRACT** Cortical visual loss is characterized by decreased vision from involvement of the occipital cortex and develops most commonly due to cerebral embolism. Left ventricular thrombus in the dysfunctional wall segment is one of the important potential cardiac sources of cerebral emboli. The transient left ventricular apical ballooning syndrome, or also known as Tako-tsubo syndrome (TTS) or stres-induced cardiomyopathy is a transient form of left ventricular dysfunction characterized by transient wall-motion abnormalities especially involving the left ventricular apex and mid-ventricle in the absence of obstructive epicardial coronary disease. Although increased cases complicated by a left ventricular thrombus are defined, systemic embolic events were reported rarely in TTS. In this case report, a patient with TTS and bilateral sudden cortical visual loss is reported.

**Key Words:** Blindness, cortical; Tako-tsubo cardiomyopathy; embolism

**ÖZET** Kortikal görme kaybı oksipital korteks tutulumuna bağlı görmede azalma ile karakterizedir ve sıklıkla serebral emboliye bağlı gelişen bir durumdur. Serebral embolinin potansiyel kardiyak sebeplerinden biri sol ventrikülde disfonksiyone duvar segmenti içinde oluşmuş trombüstür. Geçici sol ventriküler apikal balonlaşma sendromu veya aynı zamanda Tako-tsubo sendromu (TTS) veya stress ile tetiklenen kardiyomyopati olarak da bilinen sendrom, tıkaçıcı epikardiyal koroner arter hastalığı yokluğunda özellikle sol ventrikül apeks ve mid ventriküler bölgeyi kapsayan geçici duvar hareket bozuklukları ile karakterize sol ventrikül disfonksiyonunun geçici bir formudur. Sol ventrikülde trombus oluşumu ile komplike olan artan sıklıkta vakalar tanımlanmasına rağmen, TTS'lu olgularda sistemik embolik olaylar nadiren bildirilmiştir. Bu vaka sunumunda, TTS'ü olan ve çift taraflı kortikal görme kaybı gelişen bir olgu sunulmuştur.

**Anahtar Kelimeler:** Körlük, kortikal; Tako-tsubo kardiyomyopatisi; embolizm

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Cortical visual loss is characterized by decreased vision from involvement of the occipital cortex. This situation is most commonly due to cerebral embolism.<sup>1,2</sup> Emboli arising from the heart account for at least 20% of ischemic strokes.<sup>3</sup> Left ventricular thrombus with dysfunctional or aneurysmal wall segment is one of the important potential cardiogenic sources of cerebral embolism.<sup>4</sup> Tako-tsubo syndrome (TTS) is a transient form of left ventricular dysfunction without coronary artery disease.<sup>5,6</sup> Although increased numbers of cases with TTS and left ventricular thrombus are defined, systemic embolic events have been rarely reported.<sup>5-7</sup> Neurological events precipitating with the syndrome are also rare. In

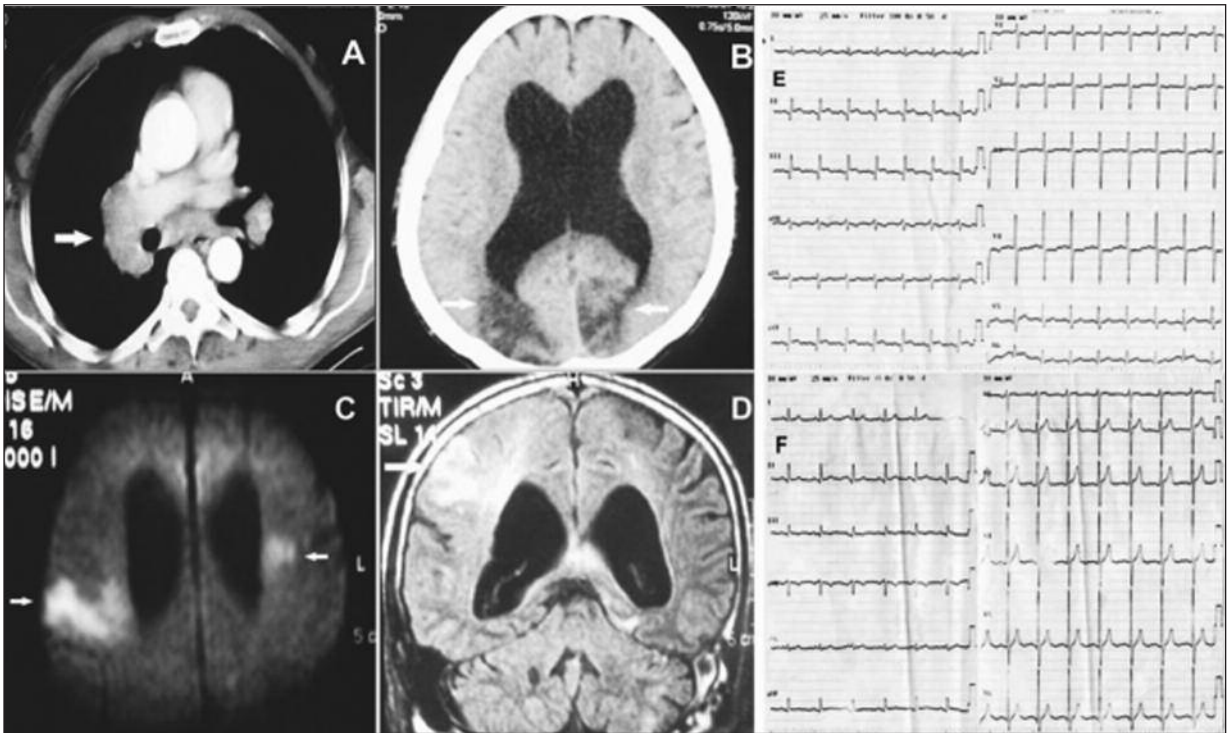
the present case, TTS in a patient with bilateral sudden cortical visual loss is reported.

## CASE REPORT

A 53-year-old male patient was hospitalized to neurology clinic with the complaints of dyspnea, headache, dizziness and bilateral sudden painless visual loss. Past medical history of the patient included stage IV non-small cell lung carcinoma and he was treated with chemotherapy, for 1 year (Figure 1A). On neuro-ophthalmologic examination, there was no light perception in either eye. Both pupils were equally reactive to light and accommodation, ocular movements were full and optic fundi appeared normal. Cranial computerized tomography (CT) showed bilateral occipital infarction (Figure 1B). Electrocardiography (ECG) on the

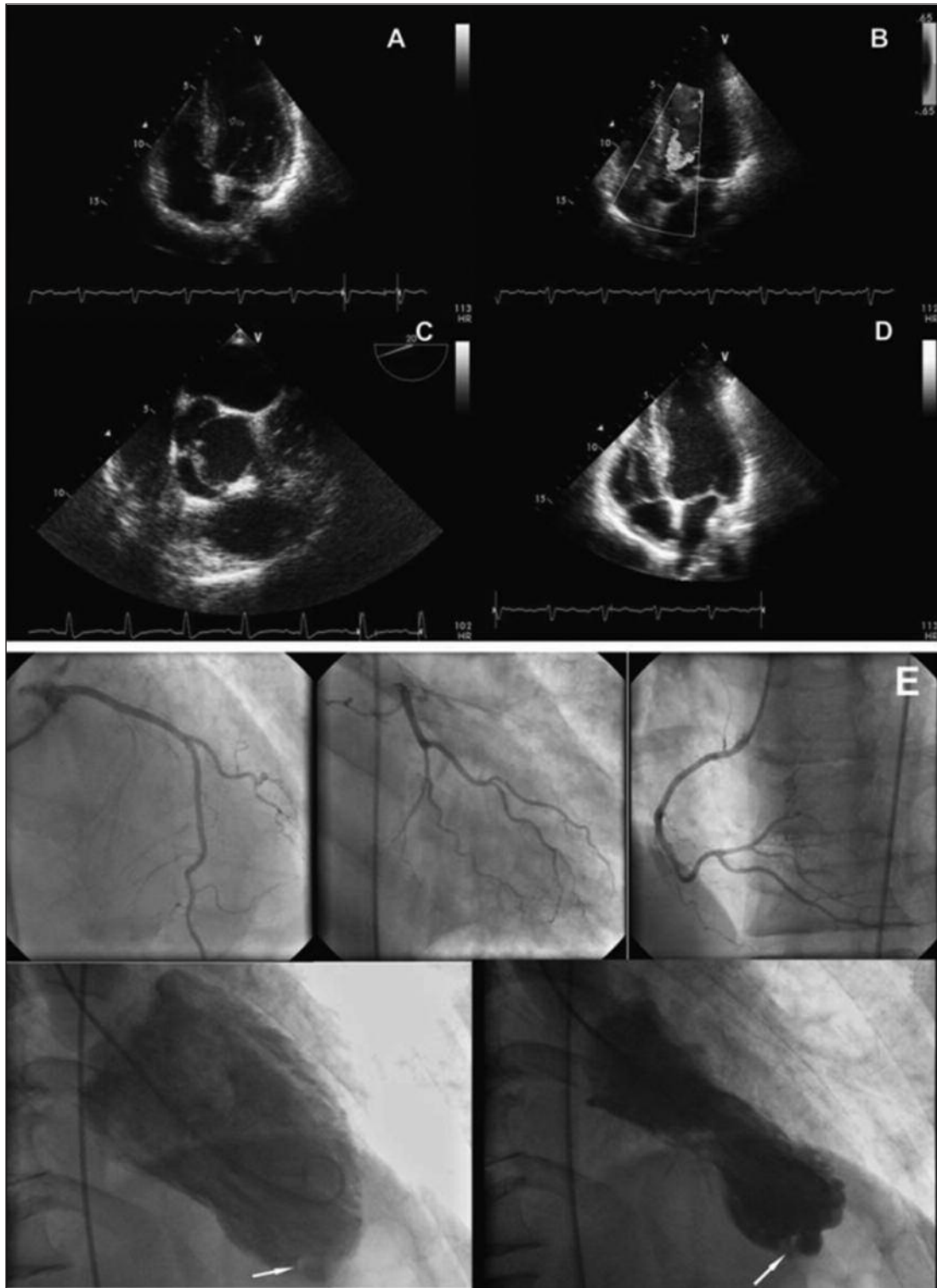
admission was normal. Transthoracic echocardiography (TTE) revealed left ventricular hypertrophy, normal wall motion, mild to moderate aortic regurgitation and bicuspid aorta (Figure 2A and figure 2B). Transesophageal echocardiography (TEE) confirmed the diagnosis of the bicuspid aorta (Figure 2C). Holter ECG, carotid and vertebral artery Doppler examination were normal. Aspirin and clopidogrel was given to the patient. Early warfarin therapy was considered but was not given because of the threat of hemorrhagic transformation of acute cerebral infarction.

On the twelfth day of the admission, dyspnea, facial paralysis and dysarthria developed in the patient. Cranial magnetic resonance imaging (MRI) showed acute infarction in the right parietal region (Figure 1C, Figure 1D). ECG showed ST seg-



**FIGURE 1:** Radiologic images of recurrent cerebral embolism, lung cancer and ST segment changes on electrocardiography

- A- Contrast enhanced thorax CT image reveals right hilar mass lesion
- B- Unenhanced axial computed tomography (CT) image of brain depicts sub-acute ischemic changes in both occipital lobes.
- C- Diffusion weighted magnetic resonance (MR) image: Restricted diffusion in right parietal lobe and left deep peri-ventricular white matter which are concordant with acute infarction.
- D- Coronal FLAIR (fluid attenuated inversion recovery) image: Right parietal hyper-intense region is consistent with edematous changes in brain tissue due to acute cerebral emboli
- E- ECG during the second attack of cerebral emboli, ST segment elevation in inferior leads
- F- Discharge ECG of the patient



**FIGURE 2:** Echocardiographic and angiographic findings

- A- Initial transthoracic echocardiography: mild hypertrophy of mid ventricular septum, no wall motion abnormality, no thrombus
- B- Transthoracic echocardiography with Color Doppler: Mild to moderate aortic regurgitation
- C- Transesophageal echocardiography: Bicuspid aorta
- D- Discharge transthoracic echocardiography: no wall motion abnormality, no thrombus
- E- Coronary angiography and left ventriculography: Normal coronary arteries and the typical apical ballooning, a hypercontractile base during systole and an apical filling defect suggesting thrombus during systole and diastole

ment elevation and pathologic Q waves in inferior leads (Figure 1E). The QT interval and analysis of repolarization duration (QTc) was normal.

The patient was transferred to the coronary care unit with the diagnosis of inferior myocardial infarction. Myoglobin, creatine-kinase-MB and cardiac troponin-I were normal. Repeated measures of high sensitive C reactive protein (hs-CRP) showed a sustained elevation such as 22.6 mg/L, 26.4 and 30.0 mg/L. Aspirin, clopidogrel, enoxaparin,  $\beta$  blocker, statin and angiotensin-converting enzyme inhibitor were started. Coronary angiogram showed normal coronary arteries, but left ventriculography demonstrated the typical apical ballooning, a hypercontractile base and an apical filling defect suggesting thrombus (Figure 2E). TTS was considered and warfarin therapy was given. The patient was discharged 2 weeks later under warfarin therapy. Discharge ECG was normal (Figure 1F) and TTE showed no wall motion abnormality and no thrombus (Figure 2D).

## DISCUSSION

TTS has been gradually recognized worldwide.<sup>5,6</sup> Increased number of cases with TTS and left ventricular thrombus is defined but systemic embolic events have been rarely reported.<sup>5-8</sup> Sasaki et al described one case with TTS, bicuspid aorta and renal embolism.<sup>7</sup> The present case is the second one with bicuspid aorta, TTS and systemic embolism. Neurological events precipitating with the syndrome are also rarely reported. Transient ischemic attack (TIA), epileptic seizures and cerebral hemorrhage were described as precipitating events for the syndrome.<sup>9-11</sup> However, TTS due to acute cerebral embolism or acute cerebral embolism due to TTS has not been clearly defined.

Bybee et al have proposed the Mayo Clinic diagnostic criteria, shown in Table 1.<sup>12</sup> The present case had all of the four criteria of TTS and had interesting clinical findings: sudden painless visual loss due to cerebral embolism, bicuspid aorta and lung cancer. The patient's first symptom was sudden visual loss due to bilateral occipital infarction and neither apical ballooning nor any cardiac so-

**TABLE 1:** Proposed Mayo Clinic Criteria for the clinical diagnosis of TTS.

1. Transient hypokinesis, akinesis, or dyskinesis of the left ventricular mid segments with or without apical involvement. The regional wall-motion abnormalities extend beyond a single epicardial vascular distribution.
2. Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.
3. New ECG abnormalities (either ST-segment elevation and/or T-wave inversion) or elevated cardiac troponin.
4. Absence of recent significant head trauma, intracranial bleeding, pheochromocytoma, myocarditis, hypertrophic cardiomyopathy

urce of the emboli was found during this period. However, left ventricular thrombus was detected during the invasive evaluation of coronary artery disease after the second attack of cerebral embolism.

One possibility is that the patient might have initially cerebral emboli due to several other sources of thrombus formation such as lung cancer or moderate to severe aortic regurgitation rather than TTS. Due to the mental stress induced by the first embolic event, he might be developed TTS.

Second possibility is that the patient initially might have embolization in the brain and then later on developed another embolization to the coronary arteries. The fact that normal coronary arteries were found does not exclude the possibility that coronary embolism or spasm might be the initial source for the development of TTS.

There is limited data about initiation time and duration of anticoagulation to prevent systemic embolism in patients with TTS.<sup>5,6,8</sup> In the present case, early usage of warfarin therapy after the first attack could prevent the recurrence of cerebral embolism. Nevertheless, it was not given because of the threat of hemorrhagic transformation of acute cerebral infarction.

The most important triggering mechanism for TTS is emotional stress and Cancer is a well known state of emotional or physiological stress.<sup>5,6</sup> There is only one case of TTS associated with brain metas-

tasis of seminoma and one with neurofibromatosis.<sup>13,14</sup> The present case had lung cancer and this condition may also be attributed as an underlying situation for development of TTS due to malignancy-induced stress.

Elevated CRP levels and thrombocytosis were accused as potential risk markers of thrombus formation in TTS.<sup>8</sup> Although there were numerous causes of CRP elevation in the present case, this

finding also suggested that inflammation may also be an interrelated mechanism in TTS.

## CONCLUSION

During the evaluation of a patient with sudden cortical vision loss due to cerebral emboli, the possibility of the presence or occurrence of TTS should always be kept in mind, especially if the source of the emboli was not found.

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