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КЛИНИЧЕСКИЙ СЛУЧАЙ СИНДРОМА ТАКОЦУБО В РАННЕМ ПОСЛЕОПЕРАЦИОННОМ ПЕРИОДЕ РИНОПЛАСТИКИ

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A Clinical Case of Takotsubo Syndrome in the Early Postoperative Period of Rhinoplasty

Резюме

Синдром такоцубо (СТ) представляет собой острою обратимую дисфункцию миокарда левого желудочка, вызванную эмоциональным или физическим триггером. В периоперационном периоде СТ в некоторых случаях индуцируется различными психологическими факторами, такими как стресс до/после операции, и непсихологическими факторами, например — введение лекарственных препаратов. В данной статье приводится описание клинического наблюдения синдрома такоцубо, развившегося в раннем послеоперационном периоде ринопластики.

Ключевые слова: *вторичный синдром такоцубо, инвертированный тип, ринопластика*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

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Abstract

Takotsubo syndrome (TS) is an acute reversible left ventricular myocardial dysfunction caused by an emotional or physical trigger. In the perioperative period, TS is in some cases induced by various psychological factors, such as stress before/after surgery, and non-psychological factors, such as drug administration. This article describes the clinical observation of takotsubo syndrome that developed in the early postoperative period of rhinoplasty.

Key words: *acute coronary syndrome, takotsubo syndrome, inverted type, clinical case, rhinoplasty*

Conflict of interests

The authors declare no conflict of interests

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BNP — brain natriuretic peptide, BP — blood pressure, CVG — cardiac ventriculography, ECG — electrocardiography, Echo — echocardiography, EF — ejection fraction, ICU — intensive care unit, LV — left ventricle, MRI — magnetic resonance imaging, ThO — thoracic organs, TS — takotsubo syndrome, VCR — ventricular contraction rate

Introduction

Takotsubo syndrome (TS) is an acute reversible heart disease with clinical presentation and results of laboratory tests and instrumental examinations similar to acute coronary syndrome. A prior significant stressor is a typical feature of TS. According to the International Takotsubo Registry (InterTAK Registry), TS in most cases (36 %) develops as a result of exposure to the so-called "physical" stress: somatic diseases, medical interventions, use of drug products [1]. Emotional stress due to various life situations, both negative and positive, is a less common TS trigger; it is found in 27.7 % of patients; in other cases, TS is either of mixed origin (7.8 %) when a patient had experienced both physical and emotional stress, or a definite trigger cannot be established (28.5 %) [1]. Among the "physical" factors acute respiratory failure is the most common — 20.2 %, with surgical interventions and injuries occupying the second position — 18.4 % [1]. The studies conducted by Guzzo G. et al. (2021) demonstrated that among 305,906 patients who have undergone various types of surgeries in the hospital of Buenos Aires in the period of 2008-2017, TS developed in 21 patients: it occurred during surgery in 6 (29 %) patients, within the first 72 hours in 7 (33 %) patients, and on day 4 and later in 8 (38 %) patients; TS in the perioperative period developed more often in men. Moreover, the author emphasizes the fact that 13 surgeries (60 %) were elective, and 10 surgeries (49 %) were deemed as low or medium risk of TS [2, 3]. According to Brooks JK et al., the literature sources for the period from 1991 to 2018 describe 28 TS episodes that developed during surgical interventions in maxillofacial region, with atypical TS forms in several cases [4]. This article presents a clinical case of inverted (reverse) variant of TS that developed in a 38-year-old man during elective rhinoplasty.

Case report

Patient P., 38 years old, diagnosed with "deviated nasal septum, nasal obstruction syndrome", was admitted to an inpatient department in St. Petersburg for elective rhinoplasty. Pre-operative examinations, as well as laboratory test results, chest X-ray, electrocardiography (ECG), presented no abnormalities. According the patient's history, he had a well-controlled intermittent asthma, chronic gastritis with no exacerbation; no known hereditary diseases; no history of allergies, or epidemiological anamnesis without abnormalities; the patient denied bad habits.

Surgery was performed on the scheduled day under general anesthesia, fentanyl, esmeron and propofol were administered in standard doses without abnormalities, hemodynamics was stable, the patient was intubated. Septoplasty with left sinusectomy was performed. At the final stage of the surgery, in order to prevent postoperative bleeding and edema, 1 mL of 0.1 % adrenaline diluted in 20 ml of saline was injected into the submucosal layer of the nasal septum of the patient. The total duration of the surgical intervention was 1 hour 45 minutes. 20 minutes after adrenaline injection, an increase in blood pressure (BP) up to 210/120 mm Hg was observed, at this time, cardiac monitor demonstrated no rhythm or conduction abnormalities; no changes in repolarization processes, ST segment deviation, or QT interval prolongation were observed. This situation was regarded as an acceptable short-term effect of the local administration of adrenaline. During the next 5 minutes, the patient's blood pressure returned to acceptable values, the patient was extubated (total duration of anesthesia was 2 hours 35 minutes). The patient in clear consciousness was transferred to the intensive care unit (ICU) for follow-up.

In the ICU, the patient almost immediately started to complain of feeling of not getting enough air and constricting discomfort in chest. Physical examination revealed the following: BP 90/60 mm Hg, regular pulse with VCR 100 bpm;RR 20 per minute; heart tones are muffled, no murmurs, harsh breathing, no rales; examination of other organ systems presented no abnormalities. 10 minutes after transfer to the ICU, the patient developed syncope, BP was 80/50 mm Hg, due to unconsciousness and unstable hemodynamics, the patient was re-intubated. ECG demonstrated sinus tachycardia with HR 100 bpm, ST elevation in leads I, II, aVL, V3-V6, corrected QT prolongation (Bazett's formula) up to 465 ms (Fig. 1).

Taking into account the clinical presentation and ECG results, the patient's condition was regarded as an acute coronary syndrome with ST elevation complicated by cardiogenic shock, therefore, the patient was urgently transferred to the vascular unit for cardiac ventriculography (CVG). CVG results demonstrated no hemodynamically significant stenosis of coronary arteries, however, 30 % reduction of left ventricular ejection fraction (LV EF)

and akinesia of all basal segments, as well as hypoakinesia of all midline LV segments were observed (Fig. 2). In view of the persistently severe patient's condition, he was transferred to ICU for further treatment; due to persistent severe hypotension, inotropic support was administered (adrenaline 0.01 μg/kg/min, noradrenaline 0.3 μg/kg/min).

CVG results were in line with of echocardiography (echo) findings: akinesia of all basal segments, hypoakinesia of all LV midline segments, LVEF calculated by Simpson's method was 35 %, with no significant changes in heart valvular apparatus. Complete blood count was indicative of neutrophilic leukocytosis: WBC 33.9×10^9/L, neutrophils 30.9×10^9/L. Among blood chemistry parameters, there was increased troponin I up to 10,376.0 pg/mL (normal up to 26.0 pg/mL), creatine phosphokinase up to 346 U/L (30–200), aspartate aminotransferase up to 43 U/L (5–34), lactate dehydrogenase to 238 U/L (125–220), total bilirubin up to 29.7 μmol/L (3.4–20.5), glucose up to 15.6 mmol/L (3.9–5.5), creatinine up to 90 μmol/L and C-reactive protein up to 9.17 mg/dL (reference range 0–0.5). Coagulogram parameters and blood electrolytes were within reference range, urinalysis presented no abnormalities.

The patient's condition on day 2 in the course of ongoing inotropic support (dopamine 4.5 μg/kg/min; noradrenaline 0.15 μg/kg/min) continued to be severe, hemodynamics was unstable (severe hypotension). Echo: EF 46 %, hypokinesia of LV basal segments persisted; ECG: sinus rhythm with HR 66 bpm, QRS axis in normal position, corrected QT 413 ms, ST elevation in chest leads significantly decreased and was regarded as an early repolarization syndrome, no pathological Q waves, negative T waves were detected in leads I, aVL (Fig. 3); chest (ThO) X-ray: no focal or infiltrative changes, signs of pulmonary interstitial edema; MSCT of the chest: no data for pulmonary embolism; signs of bilateral pulmonary edema.

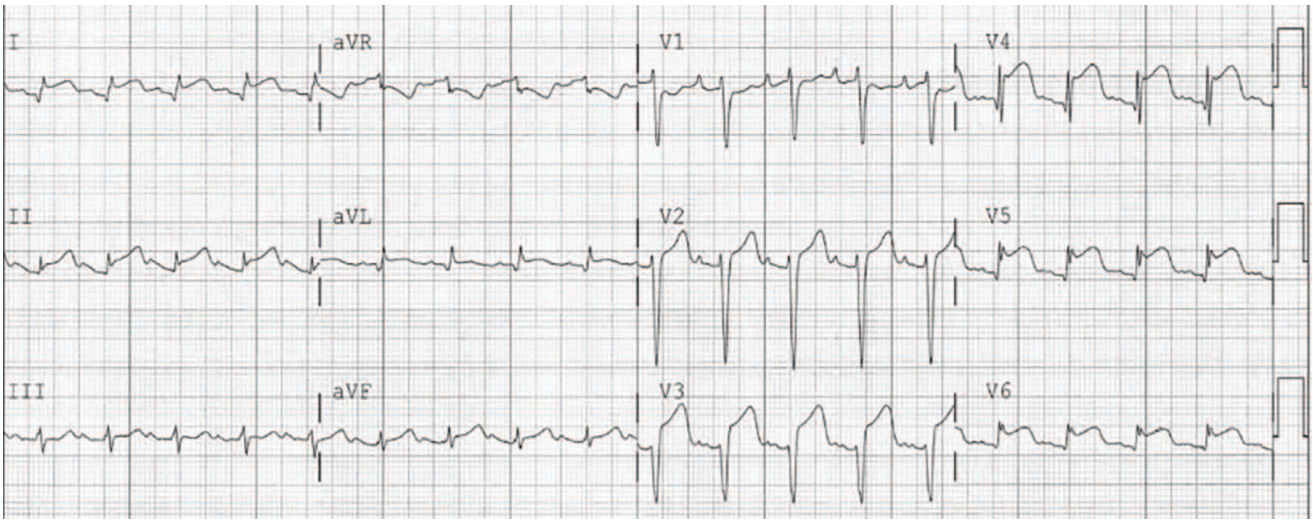

Figure 1. *ECG in the ICU*

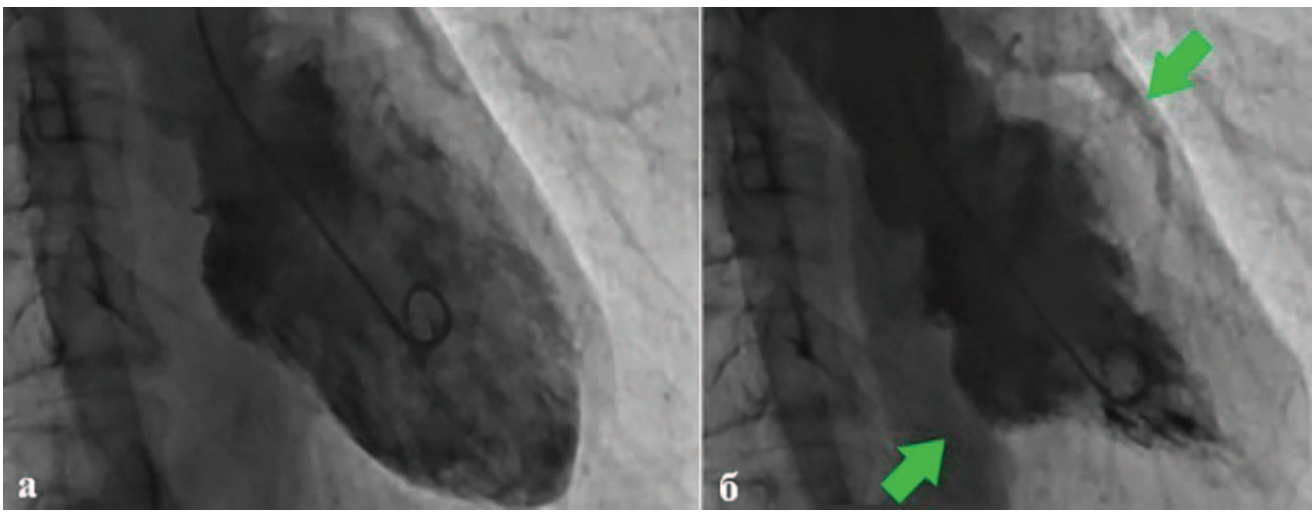

Figure 2. *Coronary ventriculography: a — diastole; b — systole (arrows indicate akinesia of all basal segments, hypo-akinesia of all median LV segments)*

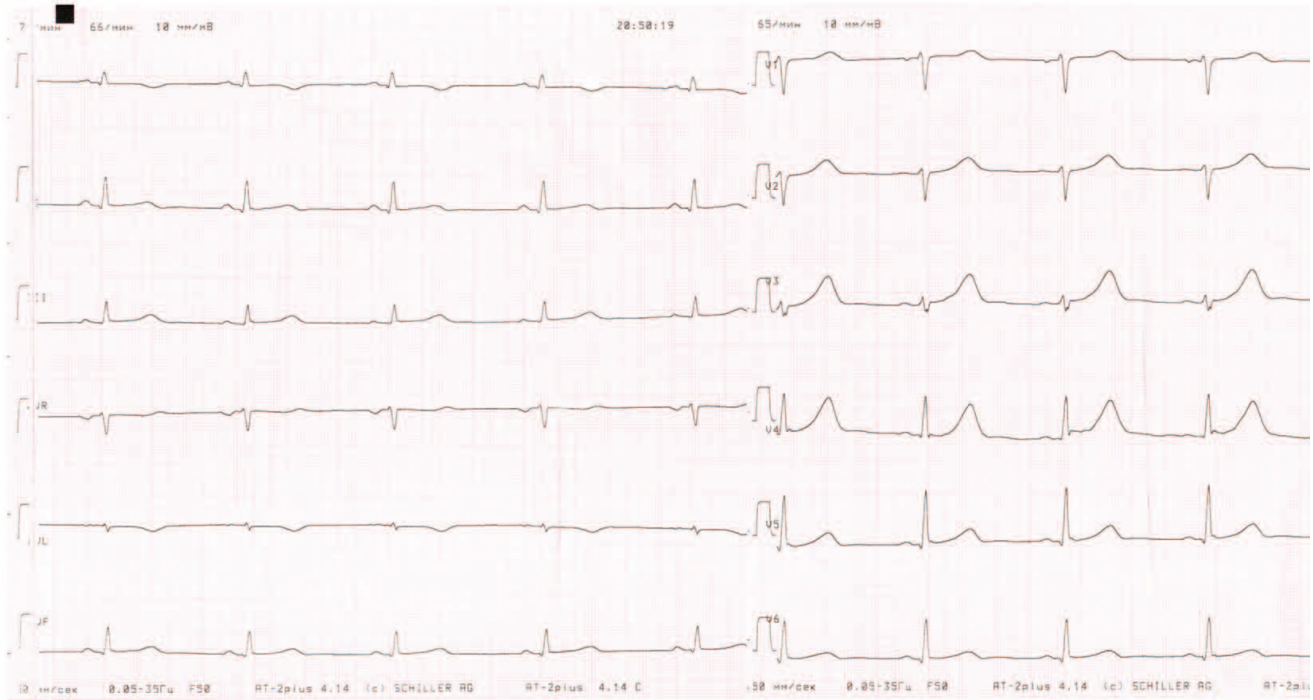

Figure 3. *ECG on day 2*

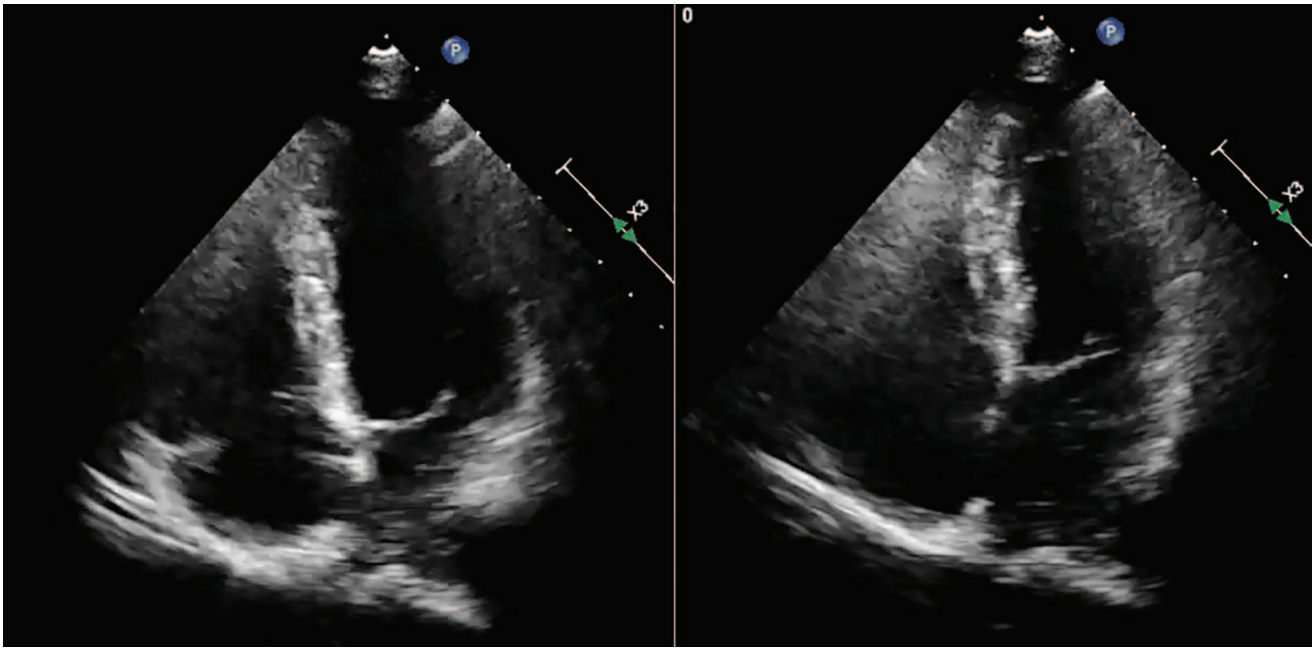

Figure 4. *EchoCG on day 9 (left-systole; right-diastole). Absence of local impairment of LV contractility*

Starting from day 3 of hospitalization, due to the suspicion of TS, vasopressors administration was discontinued (dopamine 5 μg/kg/min; noradrenaline 0.18 μg/kg/min); levosimendan (0.1 μg/kg/min) was prescribed as inotropic support (0.1 μg/kg/min), due to this measure, BP was normalized; on day 4 of the disease, levosimendan was discontinued due to the patient's stabilization.

Over time, on day 9 of hospitalization, all blood parameters returned to normal; chest X-ray demonstrated resolution of interstitial edema; ECG: sinus rhythm with HR of 65–75 bpm, normal position of ORS axis, shortening of corrected QT interval to 350 ms, moderate signs of the early repolarization syndrome of ventricles in chest leads; echo: EF 65 %, restoration of the kinetics of all LV walls (Fig. 3).

Considering clinical signs (dyspnea, chest pain); ECG results (ST elevation in leads I, aVL, V3–V6 with no reciprocal changes); no damage of coronary arteries according to CVG; impaired myocardial kinetics

according to ventriculography and echo-akinesia of all basal segments and hypo/akinesia of all midline segments, followed by normalization of ECG parameters and complete restoration of myocardial contractility by day 9 (Fig. 4), as well as the presence of a provoking stress factor (surgery and adrenaline administration), the patient was diagnosed with inverted TS.

A month after discharge from the hospital, the patient felt good, there were no complaints, no decrease in tolerance to physical activity, results of the examinations of organ systems were within normal. 2 months after TS development, the patient underwent a follow-up echo (EF 63 %, no local contractility impairment was found) and contrast-enhanced cardiac MRI (no impairment of LV myocardial kinetics, no areas of pathological accumulation of a contrast agent were found).

Discussion

In the presented clinical case, both emotional stress reaction to the planned surgical treatment, and all stages of surgery — from anesthesia up to surgical incision — could have triggered TS development. Adrenaline injection into the submucosal layer of the nasal cavity on its own could precipitate the development of TS. Literature describes about 40 cases of adrenaline-induced TS [5–8], in should be noted that adrenaline dose was often relatively low — from 0.3 mg to 1 mg [7].

According to the patient, he did not fear the upcoming surgery; the surgery proceeded smoothly until the injection of adrenaline into submucosal layer of the nasal cavity, therefore, in the presented case, it was probably the local administration of adrenaline that caused TS.

This clinical case demonstrates once again that local administration of adrenaline, even at low doses, should be performed with caution, with thorough monitoring of the patient's the vital functions. If the patient develops sudden hemodynamic disorders following adrenaline administration, ECG demonstrates ST deviation, negative T waves and prolonged QT interval, high troponin and, more typically, proBNP/BNP level, the clinicians should consider the possibility of TS development. In such event, echo as early as practicable and visualization of typical TS presentation will provide early diagnosis and determine patient management approach. The use of vasopressors in TS, including cases of hypotension, should be avoided; the drug of choice is levosimendan [9].

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