CASE REPORT

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A Rare Clinic Related to Paclitaxel Use: Type 2 Kounis Syndrome

Paklitaksel Kullanımı ile İlgili Nadir Bir Klinik: Tip 2 Kounis Sendromu

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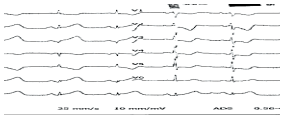
GRAPHICAL ABSTRACT

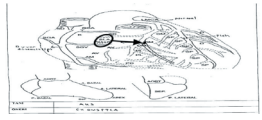
We recommend that chest pain should be questioned when allergy, anaphylaxis and angioedema develop in patients receiving chemotherapeutic drugs and that they should be alert for Kounis Syndrome

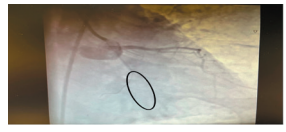
Kounis Syndrome is an acute coronary condition that arises during allergic or anaphylactic reactions. In this report, we present a case of anaphylaxis while receiving paclitaxel treatment for lung malignancy in the chemotherapy unit and the diagnosis of Type 2 Kounis Syndrome was established in the emergency department.



Patients with Kounis Syndrome typically present to the emergency department with cardiac symptoms associated with either acute or chronic allergic reactions. If left untreated, Kounis Syndrome can result in cardiorespiratory arrest or sudden death. Our case represents Type 2 Kounis Syndrome because of ST segment elevation in inferior leads and occlusion in coronary angiography. Previously reported cases in the literature were Type 1 Kounis Syndrome and our case is Type 2 Kounis Syndrome. Therefore, this case report is a rare case report. Our case is a rare case report because the culprit vessel of inferior wall ST segment elevation was not the right coronary artery (RCA) but the circumflex artery (CX).

From the perspective of public health and patient benefit, cardiac toxicity, cardiovascular hypersensitivity, and Kounis Syndrome are three critical conditions in cardio-oncology that every general practitioner and specialist should be aware of.





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Kuş B et al. A Rare Clinic Related to Paclitaxel Use… Med J West Black Sea 2025;9(1).

ABSTRACT

Paclitaxel is a member of the taxane class of chemotherapy medications and is utilized in the treatment of ovarian, breast, advanced non-small cell lung cancer, and Kaposi's Sarcoma associated with AIDS. Hypersensitivity reactions are relatively common and may range from mild clinical manifestations to severe, treatment-resistant, and even fatal outcomes. Approximately 30% of patients receiving taxane-based chemotherapeutic agents experience such reactions. Proposed pathophysiological mechanisms include IgE-mediated anaphylaxis—characterized by elevated serum tryptase levels—direct activation of mast cells and/or basophils, and the involvement of the complement cascade. Kounis syndrome is an acute coronary condition that arises during allergic or anaphylactic reactions. In the pathogenesis of Kounis syndrome, a variety of inflammatory mediators are thought to be involved, including proteases, tryptase, arachidonic acid metabolites, platelet-activating factor, as well as various cytokines and chemokines released during mast cell activation. The incidence in patients un-

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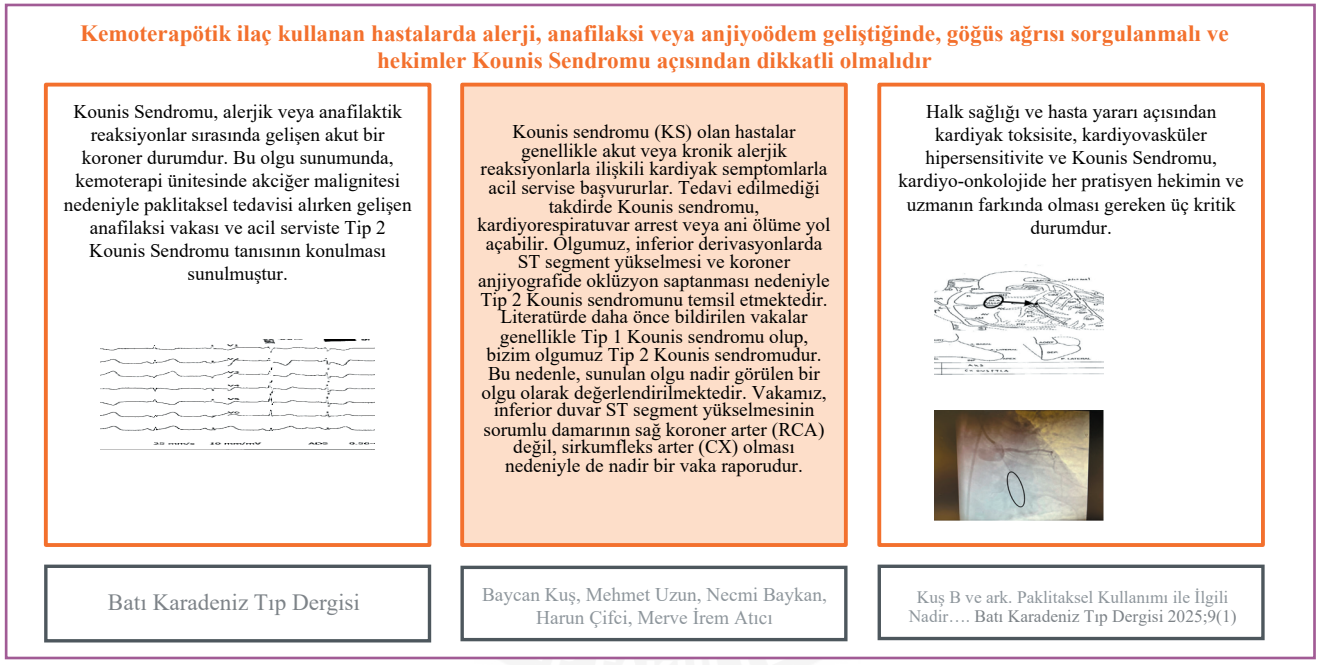
dergoing an allergic, hypersensitive, anaphylactic, or anaphylactoid reaction ranges from 1.1% to 3.4%. The most common heart complaint during application is chest pain (incidence: 86.6%). Diagnosing Kounis syndrome in the emergency department can be challenging due to the variety of clinical symptoms. It should rely on the presence of cardiovascular, allergic, or anaphylactic symptoms and signs, along with supporting evidence from laboratory tests, electrocardiograms, echocardiograms, and angiograms. In this report, we present a case of anaphylaxis while receiving paclitaxel treatment for lung malignancy in the chemotherapy unit and the diagnosis of Type 2 Kounis syndrome was established in the emergency department. Our case represents Type 2 Kounis syndrome because of ST segment elevation in inferior leads and occlusion in coronary angiography. Previously reported cases in the literature were Type 1 Kounis syndrome and our case is Type 2 Kounis syndrome. Therefore, this is a rare case report.

Our case is a rare case report because the culprit vessel of inferior wall ST segment elevation was not the right coronary artery but the circumflex artery.

Keywords: Anaphylaxis, Kounis Syndrome, lung adenocarcinoma, paclitaxel

GRAFİKSEL ÖZET



ÖZ

Paklitaksel, taksan sınıfı kemoterapi ilaçlarının bir üyesidir ve over, meme, ileri evre küçük hücreli dışı akciğer kanseri ile AIDS'e bağlı Kaposi Sarkomu'nun tedavisinde kullanılmaktadır. Aşırı duyarlılık reaksiyonları nispeten yaygındır ve hafif klinik bulgulardan şiddetli, tedaviye dirençli ve hatta fatal sonuçlara kadar geniş bir spektrumda gözlemlenebilir. Taksan bazlı kemoterapötik ajanlar alan hastaların yaklaşık %30'unda bu tür reaksiyonlar meydana gelmektedir. Öne sürülen patofizyolojik mekanizmalar arasında IgE aracılı anafilaksi, serum triptaz düzeylerinin artışı ile karakterize mast hücreleri ve/veya bazofillerin doğrudan aktivasyonu ve kompleman sisteminin devreye girmesi yer almaktadır. Kounis Sendromu, alerjik veya anafilaktik reaksiyonlar sırasında gelişen akut bir koroner durumdur. Kounis Sendromu'nun patogenezinde, mast hücresi aktivasyonu sırasında salınan proteazlar, triptaz, araşidonik asit türevleri, trombosit aktive edici faktör, çeşitli sitokinler ve kemokinler gibi farklı inflamatuvar mediatörlerin rol oynadığı düşünülmektedir. Alerjik, hipersensitif, anafilaktik veya anafilaktoid reaksiyon geçiren hastalarda insidans %1,1 ile %3,4 arasında değişmektedir. Uygulama sırasında en sık görülen kalp şikayeti göğüs ağrısıdır (insidans: %86,6). Acil serviste Kounis Sendromu'nun tanısı, çeşitli klinik semptomlar nedeniyle zorlu olabilir. Tanı, kardiyovasküler, alerjik veya anafilaktik semptomların ve bulguların varlığına ve laboratuvar testleri, elektrokardiyogram, ekokardiyogram ve anjiyogramdan elde edilen destekleyici kanıtlara dayanmalıdır. Bu raporda, kemoterapi ünitesinde akciğer malignitesi nedeniyle paklitaksel tedavisi alırken gelişen anafilaksi vakası ve acil serviste Tip 2 Kounis Sendromu tanısının konulması sunulmuştur. Vakamız, inferior derivasyonlarda ST segment yükselmesi ve koroner anjiyografide tıkanıklık olması nedeniyle Tip 2 Kounis Sendromu'nu temsil etmektedir. Literatürde daha önce bildirilen vakalar Tip 1 Kounis Sendromu iken, vakamız Tip 2 Kounis Sendromu'dur. Bu nedenle, bu nadir bir olgu sunumudur.

Vakamız, inferior duvar ST segment yükselmesinin sorumlu damarının sağ koroner arter değil, sirkumfleks arter olması nedeniyle de nadir bir vaka raporudur.

Anahtar Sözcükler: Akciğer adenokarsinomu, anaflaksi, Kounis Sendromu, paklitaksel

INTRODUCTION

Anaphylaxis is a severe, life-threatening systemic allergic reaction that manifests with various clinical symptoms such as urticaria, respiratory distress, nausea, vomiting, diarrhea, and hypotension (1). The annual incidence of severe, life-threatening anaphylaxis with circulatory symptoms is roughly 7.9 to 9.6 cases per 100,000 people (2).

Kounis Syndrome (KS) is an acute coronary syndrome triggered by allergic or anaphylactic reactions. Inflammatory mediators released during mast cell degranulation, along with those derived from the interaction of other immune cells such as T lymphocytes, macrophages, eosinophils, and platelets, play a pivotal role in the pathogenesis of KS. Substances including chymase—which acts as a conversion enzyme—tryptase, histamine, and arachidonic acid derivatives may contribute to acute ischemic events by promoting coronary vasospasm, atheromatous plaque erosion or rupture, and platelet activation. Importantly, KS is not limited to the coronary arteries; it may also involve cerebral, mesenteric, and peripheral vascular beds (3). The incidence in patients undergoing an allergic, hypersensitive, anaphylactic, or anaphylactoid reaction ranges from 1.1% to 3.4% (4). Three types of KS have been described so far:

1. Type I or MINOCA type (myocardial infarction with non-obstructive coronary arteries), which affects 76.6% of patients with normal or nearly normal coronary arteries and is induced by histamine, chymase, or arachidonic acid products (leukotrienes, platelet- activating factor).
2. Type II, which affects 22.3% of patients with quiescent preexisting coronary disease and is induced by the same factors as type I plus platelet activation.
3. Type III is a less common variant, affecting approximately 5.1% of patients, and is characterized by the development of stent thrombosis (subtype IIIa) and/or in-stent restenosis (subtype IIIb). This form is associated with hypersensitivity reactions triggered by various factors, including stent polymers, metallic components of the stent, eluted pharmacologic agents, dual antiplatelet therapies, and environmental exposures (5,6).

The most common heart complaint during application is chest pain (incidence: 86.6%). Diagnosing KS in the emergency department can be challenging due to the variety of clinical symptoms. It should rely on the presence of cardiovascular, allergic, or anaphylactic symptoms and signs, along with supporting evidence from laboratory tests, electrocardiograms, echocardiograms, and angiograms (7). The most common cause of KS is drugs (8) (Table 1). Paclitaxel is a member of the taxane class of chemotherapy medications and is utilized in the treatment of ovarian, breast, advanced non-small cell lung cancer, and Kaposi's Sarcoma associated with AIDS. Although mild allergic reactions such as skin flushing, rash and itching are common, serious allergic reactions such as anaphylaxis, angioedema and heart attacks are uncommon side effects. Hypersensitivity reactions are relatively common and may range from mild clinical manifestations to severe, treatment-resistant, and even fatal outcomes. Approximately 30% of patients receiving taxane-based chemotherapeutic agents experience such reactions. Proposed pathophysiological mechanisms include IgE-mediated anaphylaxis—characterized by elevated serum tryptase levels—direct activation of mast cells and/or basophils, and the involvement of the complement cascade (9). Over 300 cases of KS following exposure to various agents have been reported (2). Although the incidence of KS is quite low, the number of paclitaxel-related KS cases reported in the literature is even lower (10, 11).

In this report, we present a case of anaphylaxis while receiving paclitaxel treatment for lung malignancy in the chemotherapy unit and the diagnosis of Type 2 Kounis Syndrome was established in the emergency department.

CASE

A 62-year-old man with diabetes mellitus, chronic viral hepatitis and non-small cell lung cancer (adenocarcinoma), with no known history of allergies, was diagnosed with lung cancer about 45 days ago. He was admitted to the chemotherapy unit to receive the second dose of carboplatin and paclitaxel one month after his first chemotherapy session. Although he was premedicated with dexamethasone, he developed anaphylaxis during the paclitaxel infusion and was immediately admitted to the emergency department (ED) after receiving adrenaline, pheniramine, dexamethasone, and methylprednisolone in the chemotherapy unit. At the time of admission to the emergency department, the blood pressure was 131/88 mmHg, pulse rate was 88/min, respiratory rate was 16/min, fingertip oxygen saturation was

Table 1: Possible Triggers of Kounis Syndrome

Allergic patients	Drug reactions	Environmental exposure
Angioedema	Antibiotics	Bee stings
Bronchial asthma	Anaesthetics	Anti bites (fire ants)
Urticaria	Radio contrast substances	Latex contact (natural rubber)
Food allergies	Non-steroidal anti-inflammatory drugs	
Serum disease	Corticosteroids	

98%, and temperature was 36.5°C. His general condition was good and Glasgow Coma Scale Score was 15. The patient had mild uvula edema, with no signs of angioedema. Lung sounds were normal and urticarial eruptions were present. The patient described typical chest pain and stated that he had not experienced any chest pain prior to starting chemotherapy. The anamnesis obtained from the patient's relatives also confirmed the patient's statement. An electrocardiogram (ECG) was performed, showing ST segment elevation in the inferior leads and ST segment depression in the reciprocal leads (Figure 1).The patient was immediately consulted with the cardiology department and transferred to the angiography unit for emergency coronary angiography.

Coronary angiography revealed a 100% occlusion of the obtuse marginal artery (OMA) branch of the circumflex artery (Figure 2, 3), and the angiography report indicated that this occlusion was successfully opened. During the follow-up, it was noted that oncologic treatment was planned with the discontinuation of paclitaxel. According to the hospital data system, carboplatin and pemetrexed were administered in combination with dexamethasone and premedication during the next chemotherapy. The treatment continued with gemcitabine approximately 9 months later. It was determined that brain metastases had developed, and the patient also received radiotherapy. The patient was followed up infrequently in the cardiology outpatient clinic for

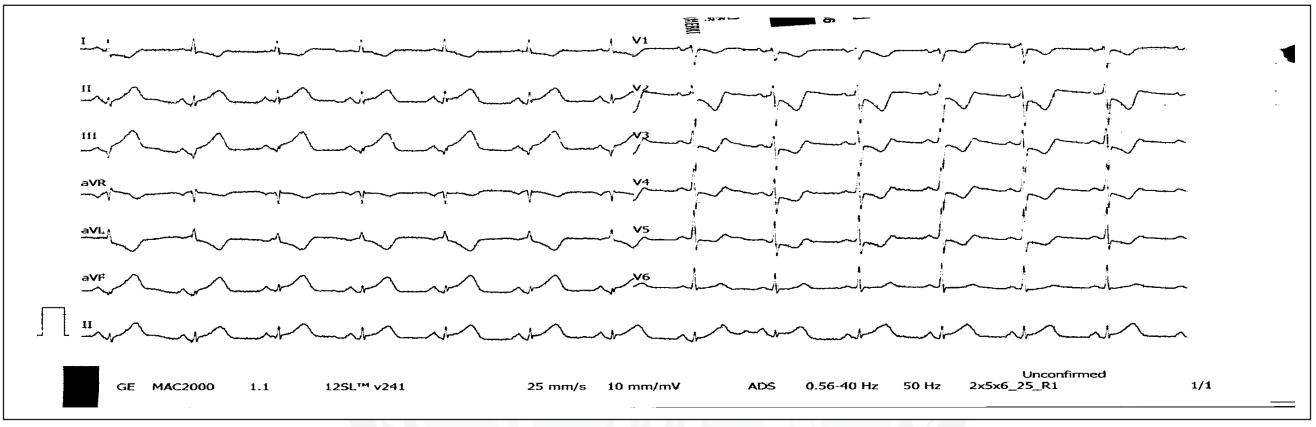


Figure 1: The ECG image of the patient showed ST elevation in the inferior leads and ST depression in the reciprocal leads, indicating acute inferior myocardial ischemia.

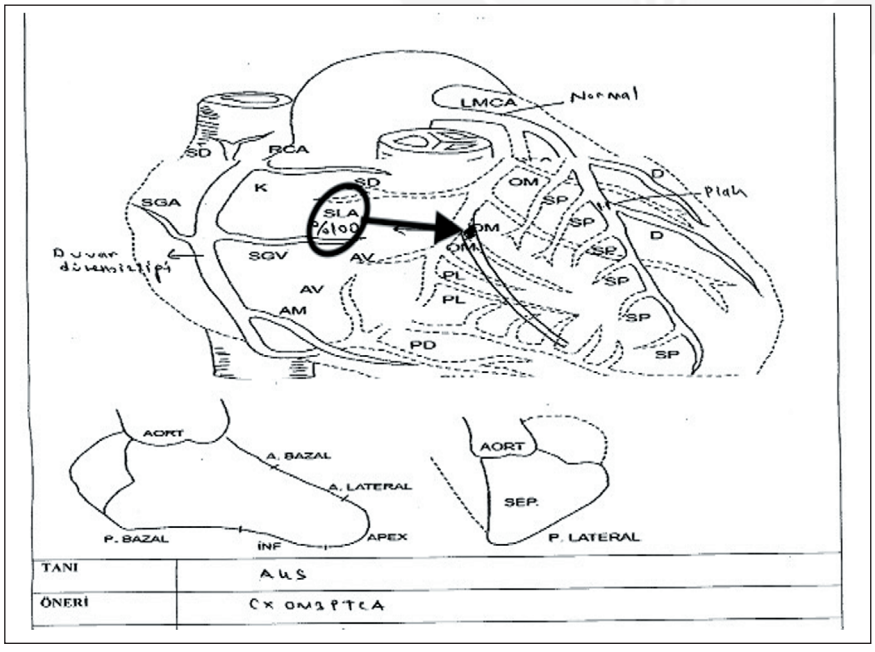


Figure 2: The coronary angiography report of the patient revealed a 100% occlusion of the obtuse marginal artery 3 (OMA3) branch of the circumflex artery.

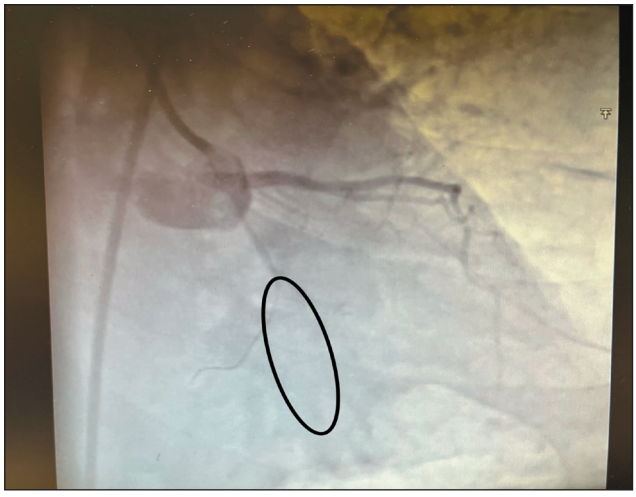


Figure 3: The fluoroscopic image of the patient's coronary angiography shows the area of occlusion, with the circled region indicating a 100% occlusion of the OMA3 (obtuse marginal artery 3) branch.

11 months after the development of KS, but did not require another coronary angiography. Approximately 11 months later, two days after receiving chemotherapy, the patient developed a cerebral hemorrhage due to a fall and passed away after being followed up in the intensive care unit for a period of time.

DISCUSSION

Drug antigens are linked to the formation of specific IgE after repeated exposure in patients, leading to sensitization (12). Through the cross-linking of drug antigens to specific IgE via high-affinity IgE receptors on mast cells and/or basophils, inflammatory mediators are released from these cells. These mediators include vasoactive amines (histamine), proteolytic enzymes (tryptase), arachidonic acid metabolites (prostaglandins and leukotrienes), proteoglycans (heparin) and some symptoms are caused by their release. Organ systems such as cutaneous, respiratory, cardiovascular and gastrointestinal systems may be affected and deaths due to anaphylaxis may occur (13,14).

Early hypersensitivity reactions due to non-immunologic mechanisms are reactions that may occur at the first encounter with the drug antigen without prior sensitization. Mediator release from mast cells and/or basophils occurs without a known IgE mechanism. The responsible mechanism is usually complement activation and/or direct mediator release. Clinical symptoms and signs are similar to IgE-mediated reactions (15).

In the pathogenesis of KS, a variety of inflammatory mediators are thought to be involved, including proteases, tryptase, arachidonic acid metabolites, platelet-activating factor, as well as various cytokines and chemokines released during mast cell activation (5). Recent studies suggest an increased risk of allergic reactions in patients with similar pathogenesis, such as cancer, diabetes, viral hepatitis, and low immune sensitivity, as seen in our case. A recently published study identified 31 immunophenotypes associated with urticaria, with 4 showing a strong causal relationship with the condition. Specifically, the presence of HLA DR^{+} $CD4^{+}$ activated T cells, expression of CD45 in CD8 br, and increased HLA DR expression in plasmacytoid dendritic cells have been associated with a heightened risk. In contrast, CD8dim natural killer T (NKT) lymphocytes appear to exert a protective effect (16).

KS has been categorized into three distinct subtypes in the literature (5, 6). Type I is characterized by coronary vasospasm occurring in individuals with no underlying coronary artery disease or identifiable cardiovascular risk factors, and with angiographically normal coronary arteries. Type II refers to patients with pre-existing coronary atherosclerosis, in whom an acute allergic reaction can trigger plaque erosion or rupture, subsequently leading to acute myocardial infarction. Type III is associated with stent thrombosis occurring after an allergic event in individuals who have previously received a drug-eluting stent (17). Our case represents Type 2 KS because of ST segment elevation in inferior leads and occlusion in coronary angiography. Previously reported cases in the literature were Type 1 KS and our case is Type 2 KS. Therefore, this case report is a rare case report (10,11).

Patients with KS typically present to the emergency department with cardiac symptoms associated with either acute or chronic allergic reactions. The clinical picture may include ischemic chest pain, dyspnea, coronary vasospasm, angina pectoris, myocardial infarction, and syncope secondary to heart failure. Common clinical signs of KS also comprise cold extremities, pallor, palpitations, hypotension, tachycardia, or bradycardia. If left untreated, KS can result in cardiorespiratory arrest or sudden death. In a study conducted in the USA, it was reported that the prevalence of KS was 1.1% and it was responsible for 7% of hospital deaths (18). Our case also had a fatal condition such as ST segment elevation, but the patient survived because of early recognition and rapid intervention. In patients exhibiting systemic allergic reactions, KS should be carefully considered in the differential diagnosis, particularly when clinical, electrocardiographic, echocardiographic, angiographic, or laboratory findings suggest acute myocardial ischemia. A prior history of atopic conditions or allergic episodes should be regarded as a significant diagnostic indicator in such cases. Paclitaxel, docetaxel and other taxanes are commonly used agents in cancer treatment, and hypersensitivity reactions to taxanes are common. In early studies with paclitaxel and docetaxel, reactions of up to 30% were reported in patients. Antihistamine (H1 and H2 histamine receptor antagonists)

and corticosteroid premedication and slow infusion rates reduced the rate of severe reactions to less than 10% (19). However, it should be noted that severe hypersensitivity reactions may occur with both paclitaxel and docetaxel despite premedication. Symptoms typically begin within the first few minutes of infusion and usually occur on the first or second administration of the drug. Symptoms may include dyspnea, urticaria, flushing, severe pain in the back or chest, gastrointestinal symptoms, hypo- or hypertension, musculoskeletal pain, paresthesia and loss of consciousness. There are also cases that report death (15,19).

Although cases of ischemic events or myocardial infarction associated with paclitaxel use have been reported in the literature, the majority of these patients had established cardiovascular risk factors such as hypertension, a history of smoking, or pre-existing coronary artery disease. These confounding variables make it difficult to attribute the observed cardiac effects solely to paclitaxel administration (20). Conversely, reports involving patients who experienced ischemic events in the absence of known coronary artery disease or significant cardiovascular risk factors remain scarce (1). In our case, there was no history of coronary artery disease and no previous symptoms suggestive of acute coronary syndrome.

Hypersensitivity reactions to anticancer chemotherapy can lead to discontinuation of first-line treatment options. Identification of these reactions can lead to rapid drug desensitization and specific diagnosis and treatment. One study investigated the symptoms seen in the presence of immediate hypersensitivity reactions (IHSR) in patients receiving anticancer chemotherapy and found that urticaria was more common with carboplatin, back pain with paclitaxel, dyspnea with oxaliplatin and docetaxel, and cardiovascular symptoms with monoclonal antibodies (22). The debate on the management of IHSR to paclitaxel continues and various management approaches including desensitization have been proposed. In a study of 425 patients, 29.2% of patients experienced IHSR and 11.8% of them met the criteria for anaphylaxis according to the Brighton scale. Of the patients who experienced IHSR, 83.8% completed their treatment by adding antihistamines, leukotriene blockers and corticosteroids and also by slowing the infusion rate (23). In our case, anaphylaxis developed despite desensitization treatment and chest pain was observed in the foreground. The medical oncology unit was informed about the patient, our patient could not continue paclitaxel treatment and the chemotherapeutic drug had to be changed.

It is known that ST segment elevations in the inferior leads usually originate from the right coronary artery (RCA). Although the ECG findings and coronary angiography findings of our case seem to be contradictory, it has been reported in the literature that the circumflex artery (CX) may be the cause of inferior wall STEMI along with the RCA (24,25). Our case is also rare that will contribute to the literature in this respect.

There have been some reports of acute coronary syndromes developing during paclitaxel use. However, many patients also have other important risk factors for cardiovascular diseases. Direct cardiotoxic damage was not reported in these studies (26,27). Gemici et al. suggested that paclitaxel has an allergic effect on the myocardium and causes acute coronary syndrome (20). However, paclitaxel-related Type 2 KS has been reported rarely in the literature. Therefore, we think that our case will contribute to the literature. We recommend that chest pain should be questioned when allergy, anaphylaxis and angioedema develop in patients receiving chemotherapeutic drugs and that they should be alert for KS.

The 2022 European Society of Cardiology (ESC) guidelines on cardio-oncology, developed in collaboration with the European Hematology Association (EHA), the European Society for Therapeutic Radiology and Oncology (ESTRO), and the International Cardio-Oncology Society (IC-OS), do not address hypersensitivity reactions or the coronary form of KS associated with hypersensitivity. This omission is noteworthy, given the frequent reports in the literature of hypersensitivity reactions and KS as significant cardiovascular side effects of cancer therapies (28).

From the perspective of public health and patient benefit, cardiac toxicity, cardiovascular hypersensitivity, and Kounis Syndrome are three critical conditions in cardio-oncology that every general practitioner and specialist should be aware of.

Acknowledgment

None.

Author Contributions

Author contributions are equal.

Conflicts of Interest

Authors declare no conflict of interest.

Financial Support

None.

Ethical Approval

This study is not an experimental and clinical research. Because of it was a case report, the ethical approve was not needed. Written informed consent was obtained from the patient for the publication of the case report.

Review Process

Externally and extremely peer-reviewed.

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