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Contents

Thrice Monthly Volume 9 Number 16 June 6, 2021

REVIEW

- 3796 COVID-19 and the digestive system: A comprehensive review
Wang MK, Yue HY, Cai J, Zhai YJ, Peng JH, Hui JF, Hou DY, Li WP, Yang JS

MINIREVIEWS

- 3814 COVID-19 impact on the liver
Baroiu L, Dumitru C, Iancu A, Leşe AC, Drăgănescu M, Baroiu N, Anghel L
- 3826 Xenogeneic stem cell transplantation: Research progress and clinical prospects
Jiang LL, Li H, Liu L

ORIGINAL ARTICLE

Case Control Study

- 3838 Histopathological classification and follow-up analysis of chronic atrophic gastritis
Wang YK, Shen L, Yun T, Yang BF, Zhu CY, Wang SN

Retrospective Study

- 3848 Effectiveness of sharp recanalization of superior vena cava-right atrium junction occlusion
Wu XW, Zhao XY, Li X, Li JX, Liu ZY, Huang Z, Zhang L, Sima CY, Huang Y, Chen L, Zhou S
- 3858 Management and outcomes of surgical patients with intestinal Behçet's disease and Crohn's disease in southwest China
Zeng L, Meng WJ, Wen ZH, Chen YL, Wang YF, Tang CW
- 3869 Clinical and radiological outcomes of dynamic cervical implant arthroplasty: A 5-year follow-up
Zou L, Rong X, Liu XJ, Liu H

Observational Study

- 3880 Differential analysis revealing APOC1 to be a diagnostic and prognostic marker for liver metastases of colorectal cancer
Shen HY, Wei FZ, Liu Q

Randomized Clinical Trial

- 3895 Comparison of white-light endoscopy, optical-enhanced and acetic-acid magnifying endoscopy for detecting gastric intestinal metaplasia: A randomized trial
Song YH, Xu LD, Xing MX, Li KK, Xiao XG, Zhang Y, Li L, Xiao YJ, Qu YL, Wu HL

CASE REPORT

- 3908** Snapping wrist due to bony prominence and tenosynovitis of the first extensor compartment: A case report
Hu CJ, Chow PC, Tzeng IS
- 3914** Massive retroperitoneal hematoma as an acute complication of retrograde intrarenal surgery: A case report
Choi T, Choi J, Min GE, Lee DG
- 3919** Internal fixation and unicompartmental knee arthroplasty for an elderly patient with patellar fracture and anteromedial osteoarthritis: A case report
Nan SK, Li HF, Zhang D, Lin JN, Hou LS
- 3927** Haemangiomas in the urinary bladder: Two case reports
Zhao GC, Ke CX
- 3936** Endoscopic diagnosis and treatment of an appendiceal mucocele: A case report
Wang TT, He JJ, Zhou PH, Chen WW, Chen CW, Liu J
- 3943** Diagnosis and spontaneous healing of asymptomatic renal allograft extra-renal pseudo-aneurysm: A case report
Xu RF, He EH, Yi ZX, Li L, Lin J, Qian LX
- 3951** Rehabilitation and pharmacotherapy of neuromyelitis optica spectrum disorder: A case report
Wang XJ, Xia P, Yang T, Cheng K, Chen AL, Li XP
- 3960** Undifferentiated intimal sarcoma of the pulmonary artery: A case report
Li X, Hong L, Huo XY
- 3966** Chest pain in a heart transplant recipient: A case report
Chen YJ, Tsai CS, Huang TW
- 3971** Successful management of therapy-refractory pseudoachalasia after Ivor Lewis esophagectomy by bypassing colonic pull-up: A case report
Flemming S, Lock JF, Hankir M, Reimer S, Petritsch B, Germer CT, Seyfried F
- 3979** Old unreduced obturator dislocation of the hip: A case report
Li WZ, Wang JJ, Ni JD, Song DY, Ding ML, Huang J, He GX
- 3988** Laterally spreading tumor-like primary rectal mucosa-associated lymphoid tissue lymphoma: A case report
Wei YL, Min CC, Ren LL, Xu S, Chen YQ, Zhang Q, Zhao WJ, Zhang CP, Yin XY
- 3996** Coronary artery aneurysm combined with myocardial bridge: A case report
Ye Z, Dong XF, Yan YM, Luo YK
- 4001** Thoracoscopic diagnosis of traumatic pericardial rupture with cardiac hernia: A case report
Wu YY, He ZL, Lu ZY

- 4007** Delayed diagnosis and comprehensive treatment of cutaneous tuberculosis: A case report
Gao LJ, Huang ZH, Jin QY, Zhang GY, Gao MX, Qian JY, Zhu SX, Yu Y
- 4016** Rapidly progressing primary pulmonary lymphoma masquerading as lung infectious disease: A case report and review of the literature
Jiang JH, Zhang CL, Wu QL, Liu YH, Wang XQ, Wang XL, Fang BM
- 4024** Asymptomatic carbon dioxide embolism during transoral vestibular thyroidectomy: A case report
Tang JX, Wang L, Nian WQ, Tang WY, Xiao JY, Tang XX, Liu HL
- 4032** Transient immune hepatitis as post-coronavirus disease complication: A case report
Drăgănescu AC, Săndulescu O, Bilașco A, Kouris C, Streinu-Cercel A, Luminos M, Streinu-Cercel A
- 4040** Acute inferior myocardial infarction in a young man with testicular seminoma: A case report
Scafa-Udriste A, Popa-Fotea NM, Bataila V, Calmac L, Dorobantu M
- 4046** Asymptomatic traumatic rupture of an intracranial dermoid cyst: A case report
Zhang MH, Feng Q, Zhu HL, Lu H, Ding ZX, Feng B
- 4052** Parotid mammary analogue secretory carcinoma: A case report and review of literature
Min FH, Li J, Tao BQ, Liu HM, Yang ZJ, Chang L, Li YY, Liu YK, Qin YW, Liu WW
- 4062** Liver injury associated with the use of selective androgen receptor modulators and post-cycle therapy: Two case reports and literature review
Koller T, Vrbova P, Meciarova I, Molcan P, Smitka M, Adamcova Selcanova S, Skladany L
- 4072** Spinal epidural abscess due to coinfection of bacteria and tuberculosis: A case report
Kim C, Lee S, Kim J
- 4081** Rare complication of inflammatory bowel disease-like colitis from glycogen storage disease type 1b and its surgical management: A case report
Lui FCW, Lo OSH
- 4090** Thymosin as a possible therapeutic drug for COVID-19: A case report
Zheng QN, Xu MY, Gan FM, Ye SS, Zhao H
- 4095** Arrhythmogenic right ventricular cardiomyopathy characterized by recurrent syncope during exercise: A case report
Wu HY, Cao YW, Gao TJ, Fu JL, Liang L
- 4104** Delayed pseudoaneurysm formation of the carotid artery following the oral cavity injury in a child: A case report
Chung BH, Lee MR, Yang JD, Yu HC, Hong YT, Hwang HP
- 4110** Atezolizumab-induced anaphylactic shock in a patient with hepatocellular carcinoma undergoing immunotherapy: A case report
Bian LF, Zheng C, Shi XL

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Acute inferior myocardial infarction in a young man with testicular seminoma: A case report

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Abstract

BACKGROUND

Atherosclerosis represents the main cause of myocardial infarction (MI); other causes such as coronary embolism, vasospasm, coronary-dissection or drug use are much rarely encountered, but should be considered in less common clinical scenarios. In young individuals without cardiovascular risk factors, the identification of the cause of MI can sometimes be found in the medical history and previous treatments undertaken.

CASE SUMMARY

We present the case of a 34-year-old man presenting acute inferior ST-elevation MI without classic cardiac risk factors. Seven years ago, he suffered from orchidopexy for bilateral cryptorchidism, and was recently diagnosed with right testicular seminoma for which he had to undergo surgical resection and chemotherapy with bleomycin, etoposide and cisplatin. Shortly after the first chemotherapy treatment, namely on day five, he suffered an acute MI. Angiography revealed a mild stenotic lesion at the level of the right coronary artery with suprajacent thrombus and vasospasm, with no other significant lesions on the other coronary arteries. A conservative treatment was decided upon by the cardiac team, including dual antiplatelets therapy and anticoagulants with good further evolution. The patient continued the chemotherapy treatment according to the initial plan without other cardiovascular events.

CONCLUSION

In young individuals with no cardiovascular risk factors undergoing aggressive chemotherapy, an acute MI can be caused by vascular toxicity of several anti-cancer drugs.

The authors have read the CARE Checklist (2016), and the manuscript was prepared and revised according to the CARE Checklist (2016).

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Core Tip: Atherosclerosis represents the main cause of acute myocardial infarction (MI), but less frequent causes should be evaluated in young individuals not at risk of such cardiac events. Aggressive chemotherapy for testicular seminoma increases vascular toxicity that may induce acute MI, complicating the clinical course of the subject. Under conservative treatment with antiplatelets and anticoagulants, the clinical evolution of subjects is favorable, but with the extant risk of repeating cardiac events on further chemotherapy courses.

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INTRODUCTION

Testicular cancer is a prototypic type of tumor in young men, being at the same time the most frequent solid tumor in the age range of 15 to 40[1]. Despite the negative clinical features and psychological impact[2], the curability rate of testicular cancer with modern therapy is high, with a low mortality at 5- and 10-years[1]. Side effects among testicular cancer survivors are significant, and result from underlying neoplasia and treatment[3].

By all histological subtypes of testicular tumors, seminomas are most frequently encountered in patients with a history of cryptorchidism. In subjects presenting stage I or II seminoma, chemotherapy with cisplatin-based cures assures roughly a 90% rate of curability[4]. Although efficient, the combination of bleomycin, etoposide and cisplatin (BEP) is associated with many side effects that include nephrotoxicity, neuropathy, infertility, vascular toxicity or secondary leukemia[5].

Vascular toxicity can take various forms including the Raynaud phenomenon, myocardial infarction (MI) or cerebrovascular events. Even if a study showed no supplementary risk of acute vascular events in patients receiving cisplatin-based chemotherapy for testicular cancer[6], we present the case of a 34-year-old man who developed inferior MI during the first course of chemotherapy for testicular seminoma. The patient gave informed consent for the presentation of the clinical case.

CASE PRESENTATION

Chief complaints

A 34-year-old man was admitted to the cardiology department for intense chest pain, malaise and diaphoresis. This is the first chest pain episode, the patient describing it as constrictive and not responding to anti-inflammatory medication taken at home.

History of present illness

The chest pain developed 2 h previously, approximately 12 h after he ended his first course of chemotherapy for right testicular seminoma.

History of past illness

Seven years prior, the patient suffered from orchidopexy for bilateral cryptorchidism and had been diagnosed with right testicular seminoma two months prior to the incident under review; surgical resection was performed shortly after the diagnosis and was followed by chemotherapy. The latter was a combination of three anticancer drugs (BEP): Bleomycin 30 mg on days 1-5, 8 and 15, and etoposide 100 mg/mp and

cisplatin 20 mg/mp in the first five days. The chemotherapy course would have been repeated on day 21.

On day 5, shortly after the first chemotherapy session, the patient developed severe acute chest pain, 10 out of 10 on the visual analogue scale.

Personal and family history

He had neither cardiovascular risk factors, nor a family history of cardiovascular events.

Physical examination

The clinical examination on admission pinpointed, blood pressure of 120/80 mmHg, a heart rate of 85 beats/min, with cardiac and pulmonary examination showing no significant pathological findings.

Laboratory examinations

The electrocardiogram revealed sinus rhythm, ST-segment elevation in the inferior leads (Figure 1) and elevated markers of necrosis (troponin I 8.37 ng/mL, CK-MB 18.23 ng/mL). Total white blood cell count and neutrophil count were within normal ranges.

Total, low-density and high-density lipoproteins cholesterol, as well as glycated haemoglobin were within the normal cut-off values.

Imaging examinations

The subject's echocardiography showed hypokinesia in the basal segments of the inferior and infero-lateral walls of the left ventricle, with a global ejection fraction 55%. Furthermore, no significant valvular abnormalities were detected.

FINAL DIAGNOSIS

The patient was given loading dosages of aspirin and clopidogrel and was directly taken to the catheter laboratory for an angiography, with the final diagnosis of acute inferior ST-elevation MI.

The left main, anterior interventricular and circumflex artery were normal, while the right coronary artery showed a mild stenotic lesion (30%-40%) with slightly suprajacent thrombus and vasospasm (Figure 2).

TREATMENT

Medical treatment and follow-up were decided in the catheter laboratory. The patient was admitted to the coronary care unit for 3 d, where he was treated with aspirin, clopidogrel, a calcium blocker (namely verapamil), atorvastatin, isosorbide dinitrate, un-fractionated heparin and perindopril. He remained asymptomatic and was discharged with the same treatment except anticoagulants, in order to continue the chemotherapy according to the initial protocol.

OUTCOME AND FOLLOW-UP

The patient proceeded with chemotherapy without other acute cardiovascular events. At six months later he was asymptomatic from a cardiovascular point of view, with no evidence of active disease found during an abdominopelvic computer tomography. He remained under long-term surveillance for both distant and nodal relapse.

DISCUSSION

In a patient without cardiovascular risk factors, the emergence of an acute MI shortly after the first course of BEP makes chemotherapy a very likely cause of the acute ischemic event, although the exact pathophysiological mechanisms are difficult to prove conclusively. The patient was in sinus rhythm, and echocardiography ruled out intra-cardiac sources of thrombo-embolism such as intra-cardiac thrombus or tumor,

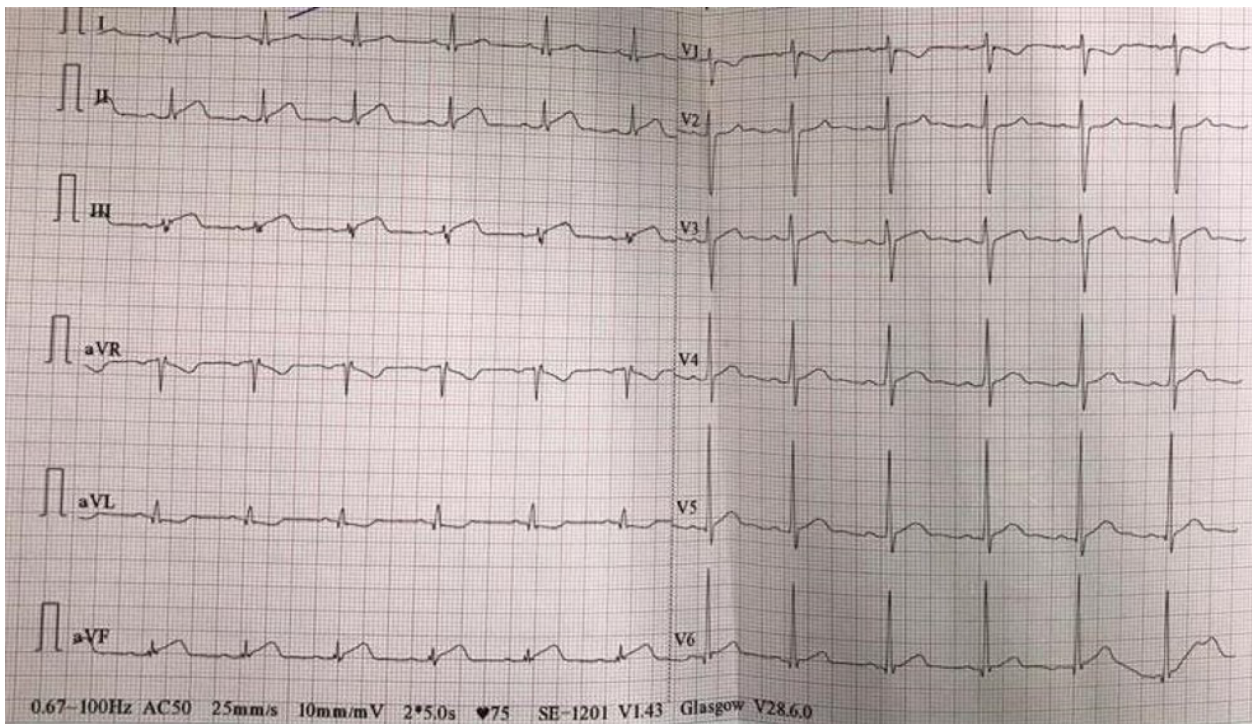


Figure 1 Electrocardiogram with sinus rhythm and ST-segment elevation in II, III and aVF leads.



Figure 2 Right coronary artery showing mild stenotic lesion with thrombus at crux cordis (arrow).

mitral stenosis or other risk factors for embolism, excluding a thromboembolism-related MI. Furthermore, emboli frequently stop at the level of stenosis, bifurcation or tortuosity, this not being the case of the thrombus identified in our patient that, with a high probability, was formed *in situ*; moreover, an atherosclerotic cause of this acute MI is less likely, as the other coronary segments showed no signs of atherosclerosis. Although rare and unpredictable, Takotsubo syndrome is associated with several chemotherapy regimens (mainly with 5-fluorouracil), reason for which we had considered this diagnosis. However, the patient did not meet the Mayo clinic criteria[7], since he did not have dyskinesia of left mid-ventricular segment with or without apical involvement as the Mayo clinic criteria requires for such a diagnosis (Video 1, Video 2, and Video 3). Furthermore, the chemotherapy undertaken by our patient is not associated with Takotsubo syndrome, but given the unpredictability of the disease this possibility should be considered even if not reported previously. Consequently, we speculate the patient suffered an endothelial lesion with *in situ* thrombus formation promoted by the anticancer drugs with suprajacent coronary

vasospasm, but in the absence of optical coherence tomography or intravascular echocardiography the exact mechanism cannot be established. For the evaluation of coronary vasospasm, the right coronary artery was injected with 200 µg nitroglycerine with the slight improvement of the acute myocardial infarction culprit lesion, but without its total disappearance. The most likely explanation for the reduced response to nitroglycerine is that the lesion encountered on angiography is the result of a complex mechanism that also includes endothelial erosion apart from coronary vasoconstriction. The erosion of the endothelial taken as one main mechanism of cisplatin vascular toxicity is supported by both experimental and clinical data regarding endothelial damage, apoptosis as well as platelet adherence, activation and aggregation[8,9]. Moreover, an increasing body of evidence points to the fact that cisplatin also induces endothelial dysfunction, affecting both the relaxation and contractile function through severe damage to blood vessel walls[10]. The mechanisms underlying the latter are mainly the reduction of endothelial nitric oxide synthetase and an increase in plasminogen activator inhibitor 1[11], but also include other mechanisms such as hydrogen sulphide availability[12]. Thrombus formation is the result of one or more of the following factors that build Virchow's triad: endothelial dysfunction, hypercoagulation and blood stagnation. All anticancer drugs our patient took can induce either one or more of the factors within Virchow's triad. Bleomycin increases interleukin-1 secretion at the pulmonary level, and further on the latter acts on the vascular endothelial promoting procoagulant activity and leukocyte adhesion[13]. Other studies have shown that cisplatin induces coronary spasms[14] and even direct damage to vascular endothelium[8]. Moreover, etoposide is associated with the increased risk of MI[15] especially if used in conjunction with other cardiotoxic agents. Another mechanism of thrombosis which could have explained the broader angiographic picture is a prolonged vasospasm with coronary occlusion and secondary thrombus formation[16].

CONCLUSION

For the case presented above we hypothesize that the particular chemotherapy regimen employed induced not only an endothelial dysfunction with vasospasm, but also a direct endothelial lesion that promoted thrombus formation in a subject predisposed to thrombosis given the neoplasia and anticancer treatment. Procoagulant activity increases mainly when multiple drugs work synergistically in combination, as in the case of our patient[6]. Furthermore, many factors contribute to the systemic overall cardiotoxicity, some related to the drugs themselves but also to the individual. Moreover, given that young individuals without any cardiovascular risk factors are predisposed to ischemic cardiovascular events, preventive measures during chemotherapy should actively be implemented; for example, one such measure could be the addition of anticoagulants or aspirin[17] to the adjuvant therapy, along with the reduction in dosages, the increase of time-intervals between treatment administration and the avoidance of certain combinations of anti-cancer drugs.

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