Cardiotossicità Acuta da Cisplatino: Uno Scenario Insolito

Dr. Andrea Chiampan
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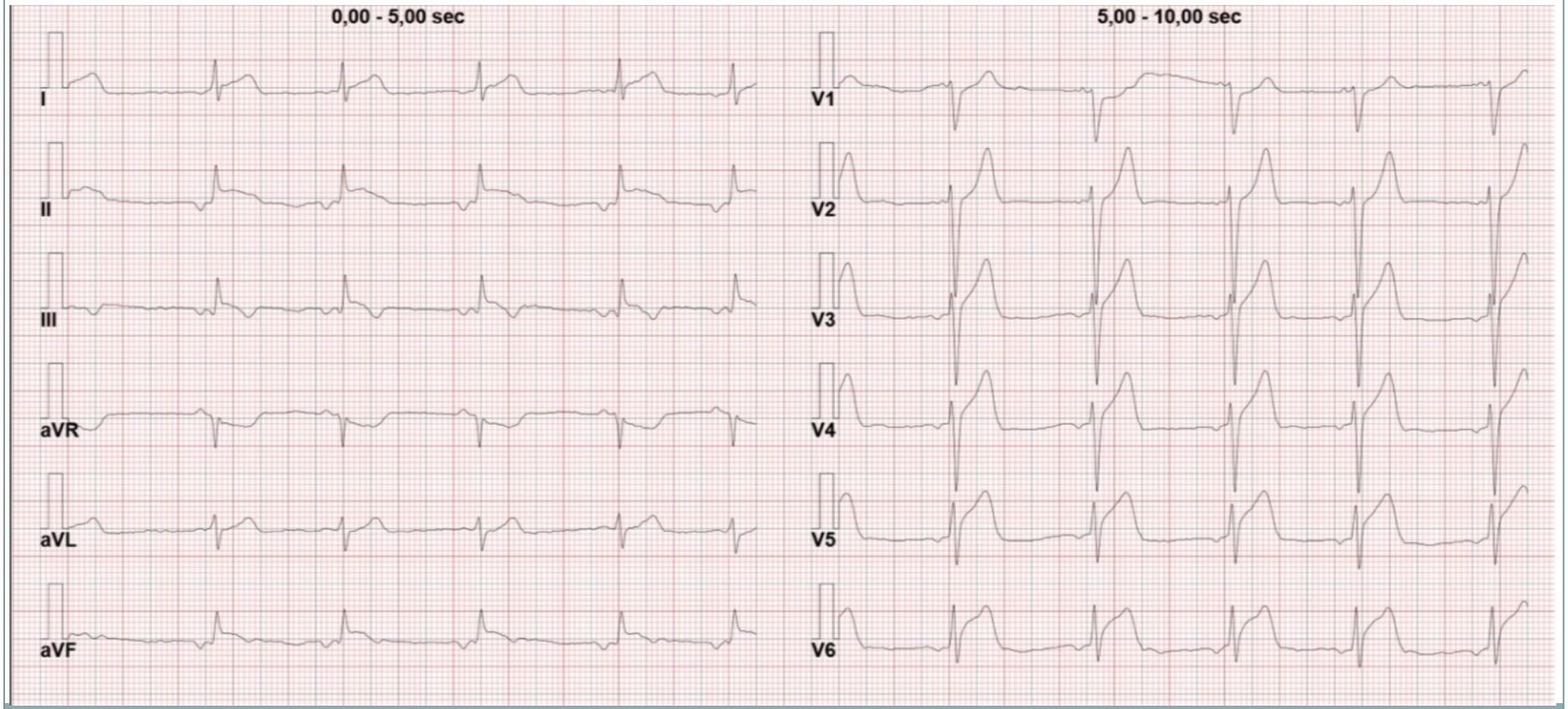
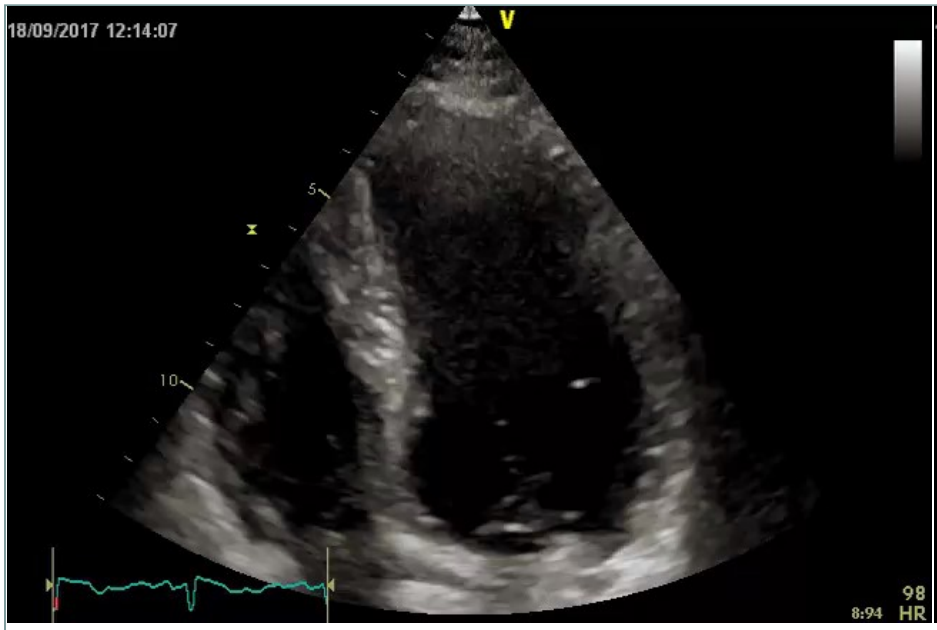
- Paziente maschio di 40 anni
- Tabagismo anamnestico blando (fino a 04/2017)
- Non altri fattori di rischio né eventi cardiovascolari anamnestici
- 21/06/2017 Intervento di Orchifunicolectomia sinistra per un Seminoma del diametro di 10 cm, con invasioni linfo-vascolari, pT3. Alla Tac stadiazione (28/6) riscontro di adenopatie addominali di natura secondaria
- Impostato trattamento chemioterapico comprendente 3 cicli di

  Cisplatino
  Etoposide
  Bleomicina

- 17/09/2017: Dopo 5 giorni dal 3° ciclo di chemioterapia accesso in PS per dolore toracico oppressivo associato a nausea e vomito

- Ecocardioscopia in PS: Ventricolo sinistro con acinesia dell'apice in toto. Contrattilità globale lieve-mediamente ridotta.

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V
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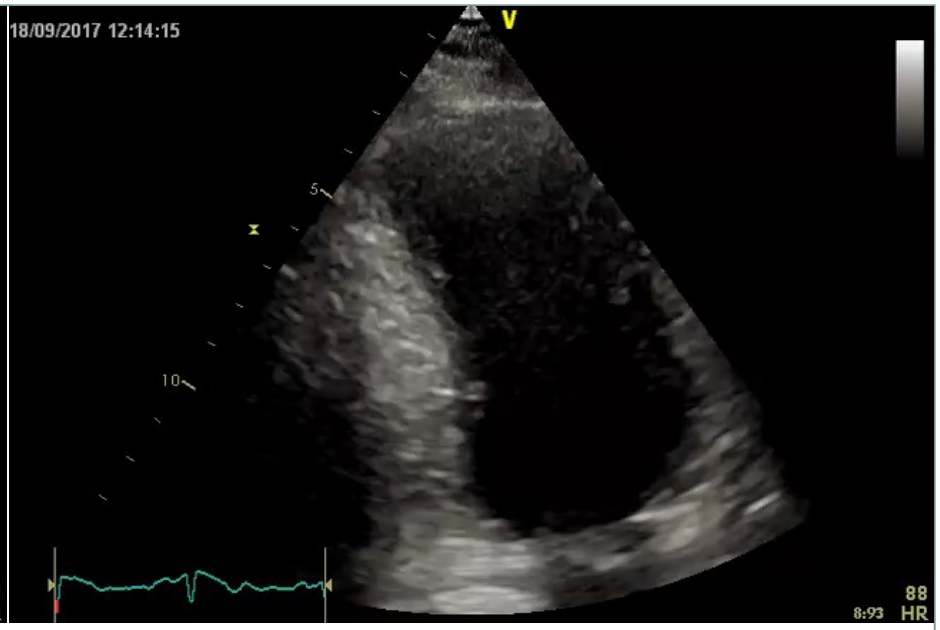
10
98
8:94
HR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V
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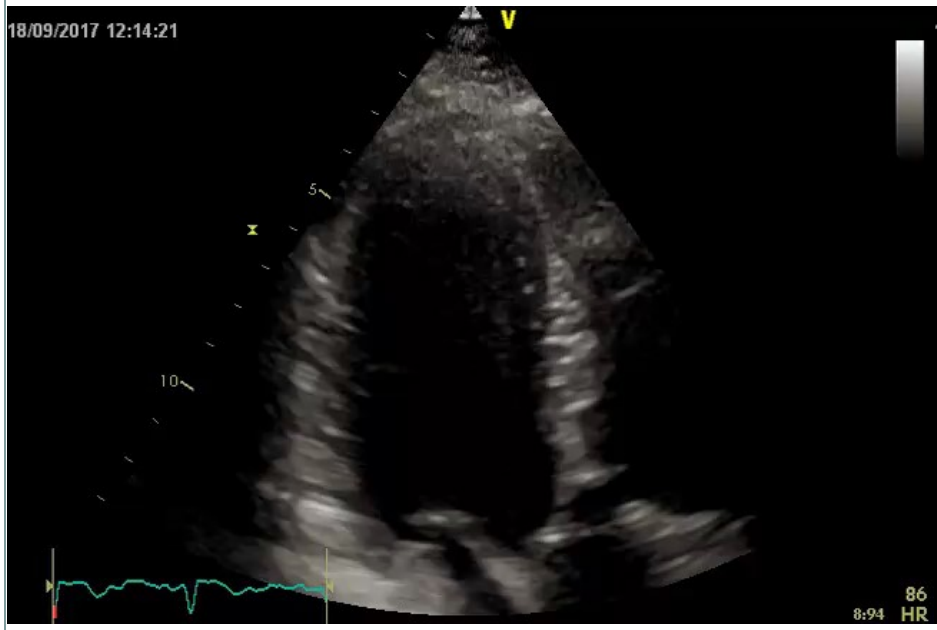
10
88
8:93
HR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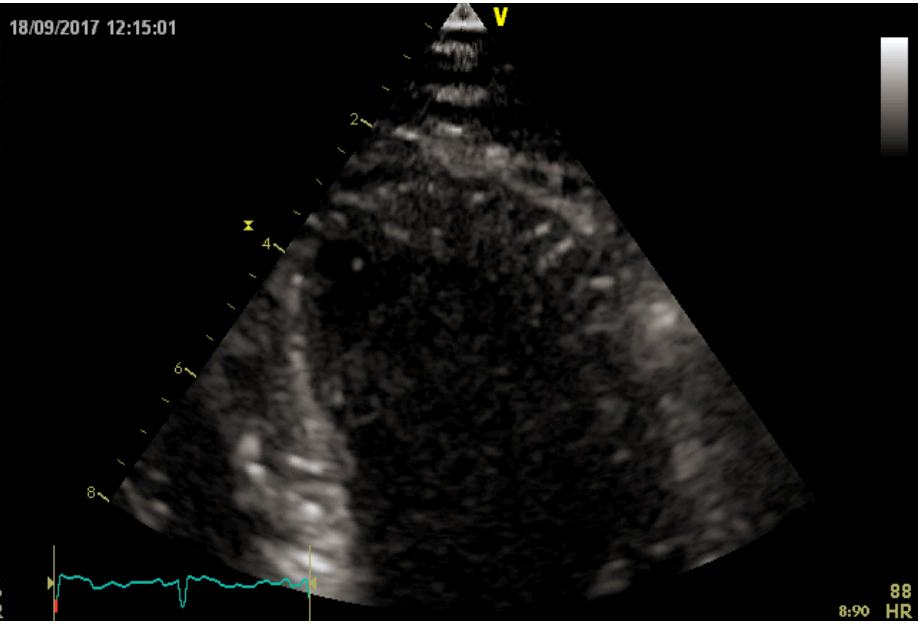
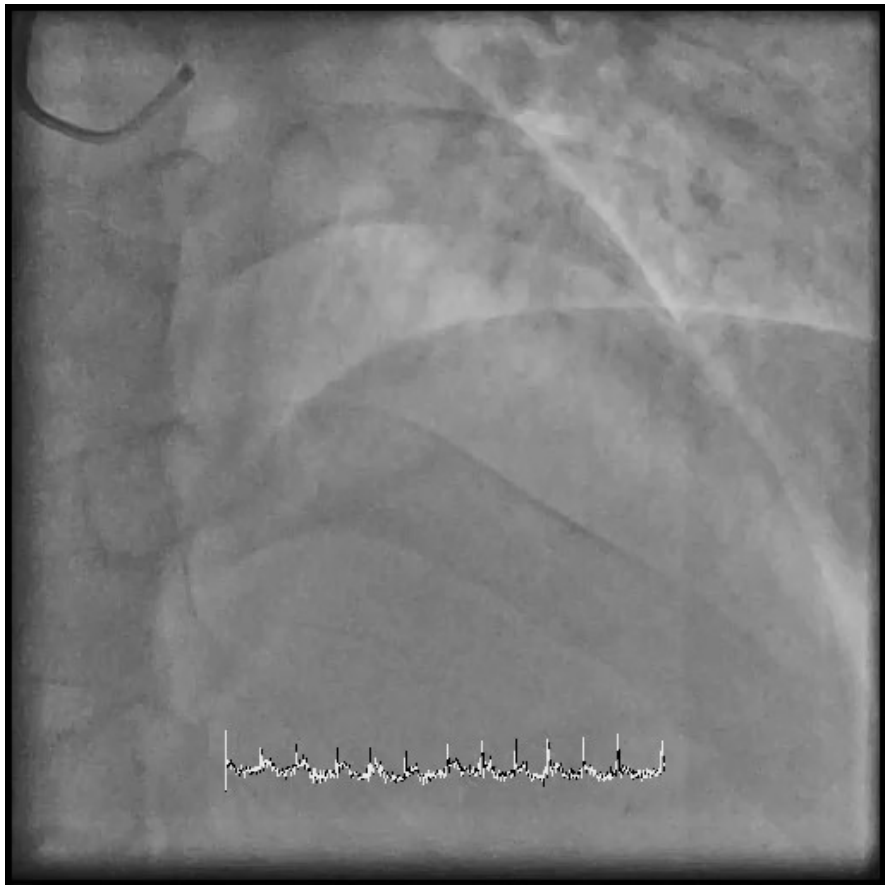
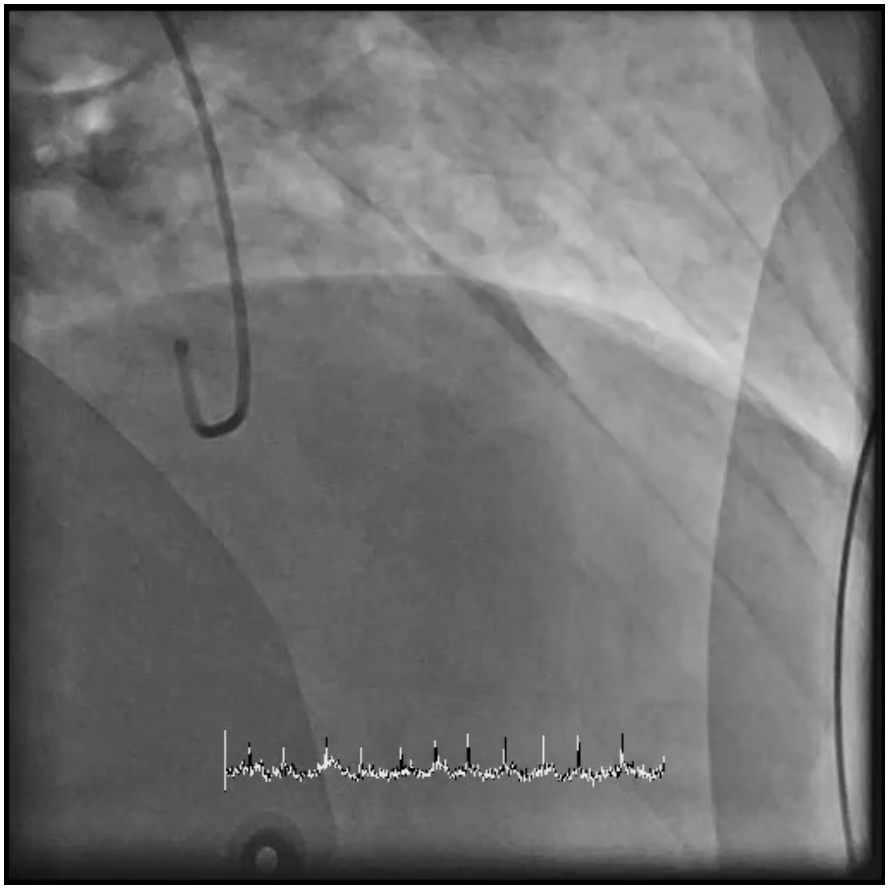
V
5
10
86
8:94
HR


18/09/2017 12:15:01
V
2
4
6
8
88
8:90
HR

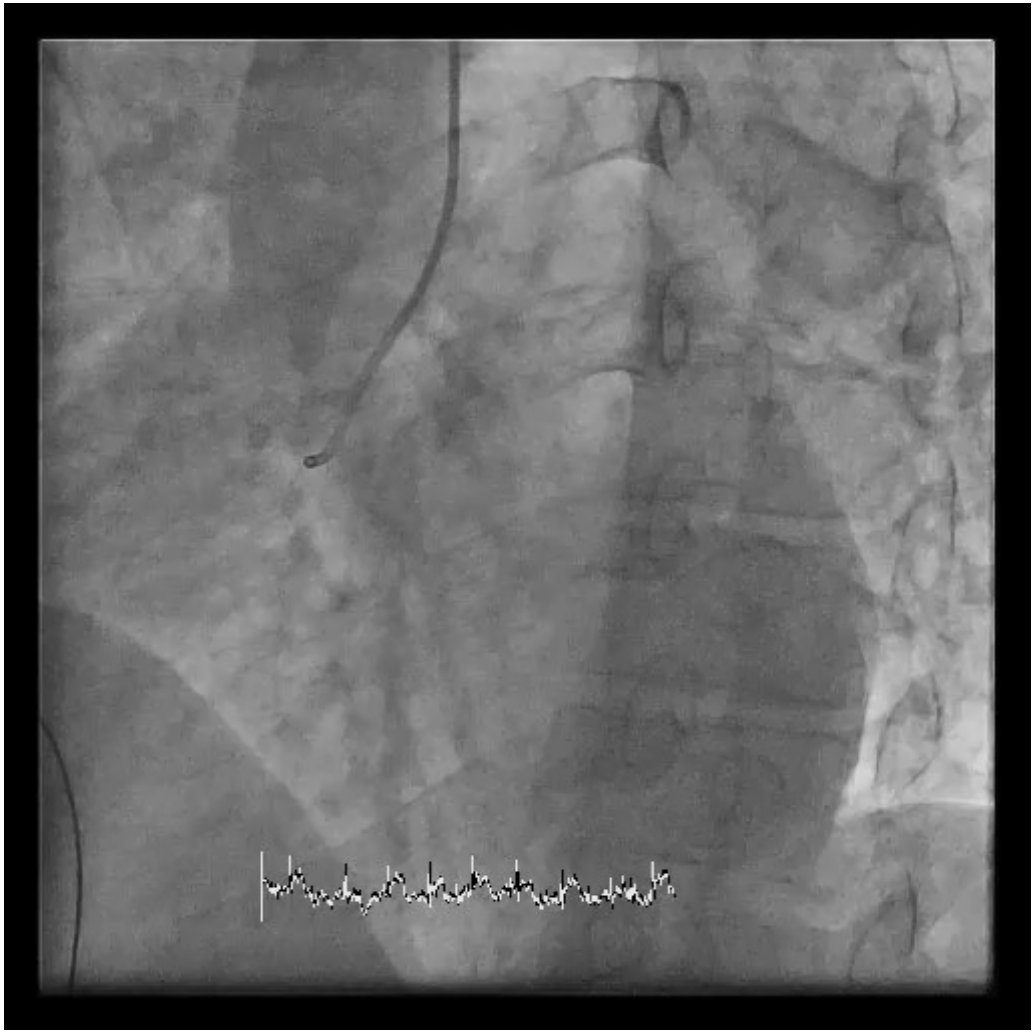
Studio coronarografico in urgenza

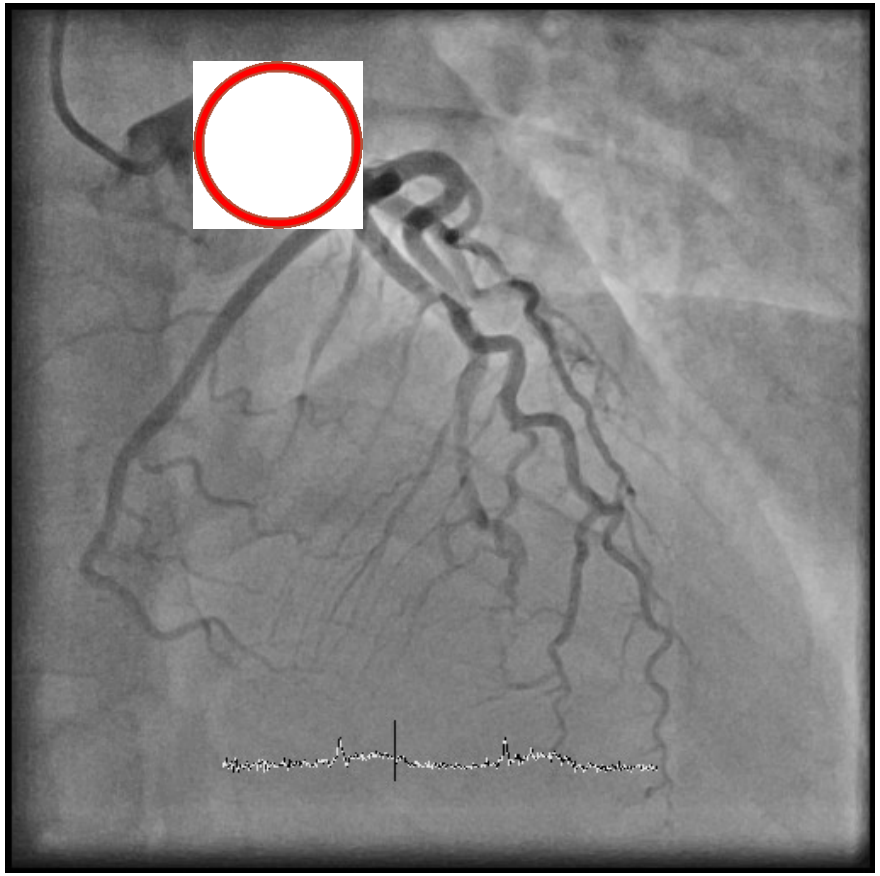


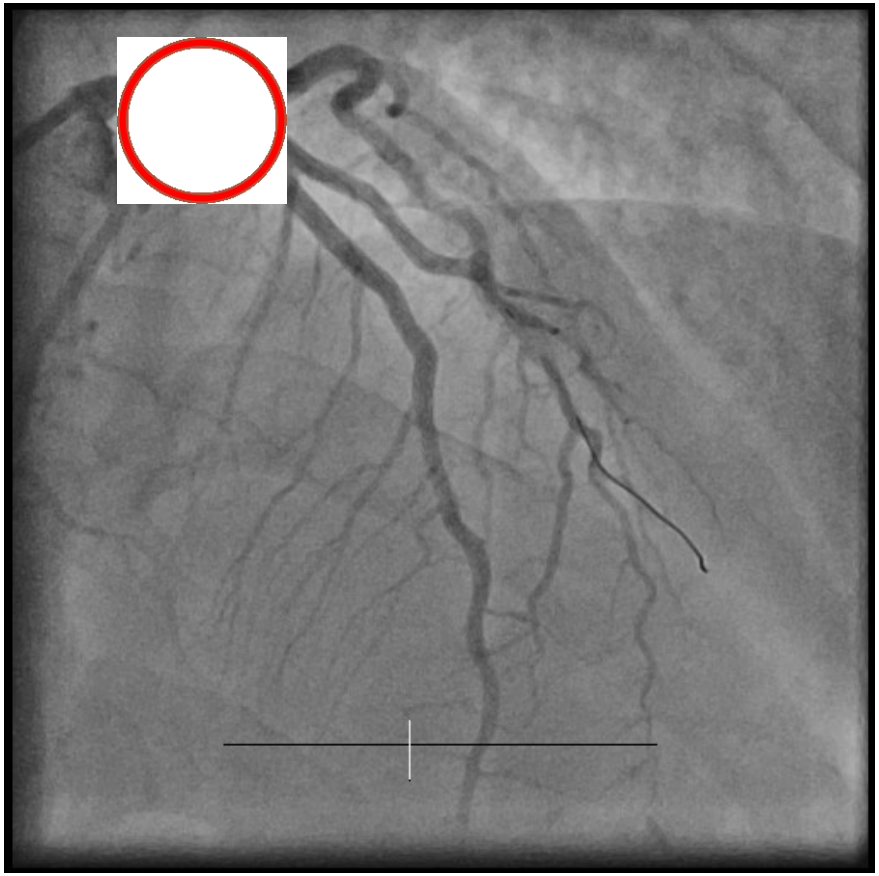


Coronaria sinistra

Coronaria destra

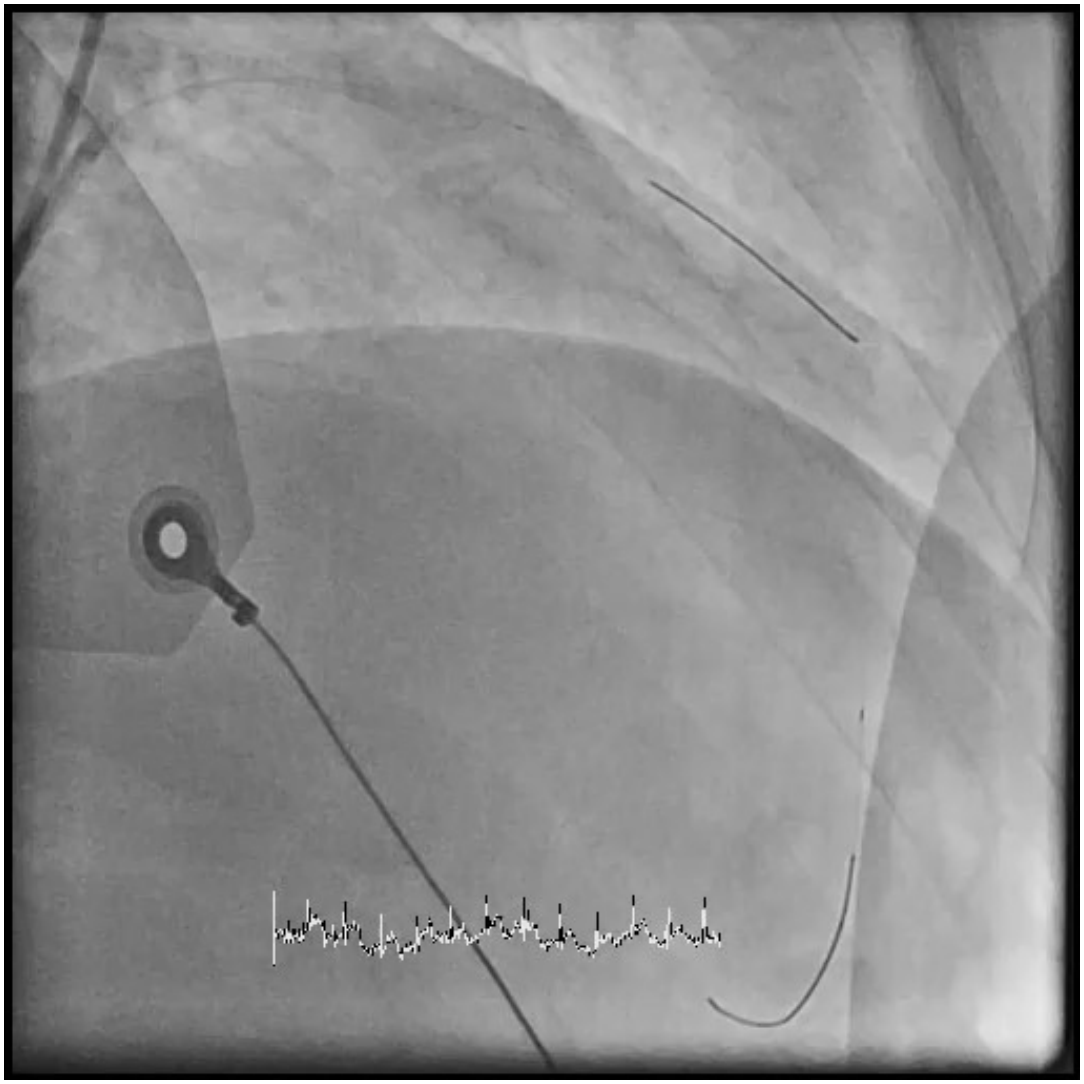






Pre e Post Tromboaspirazione

Termine Procedura



Ematologia e coagulazione

EMOCROMO

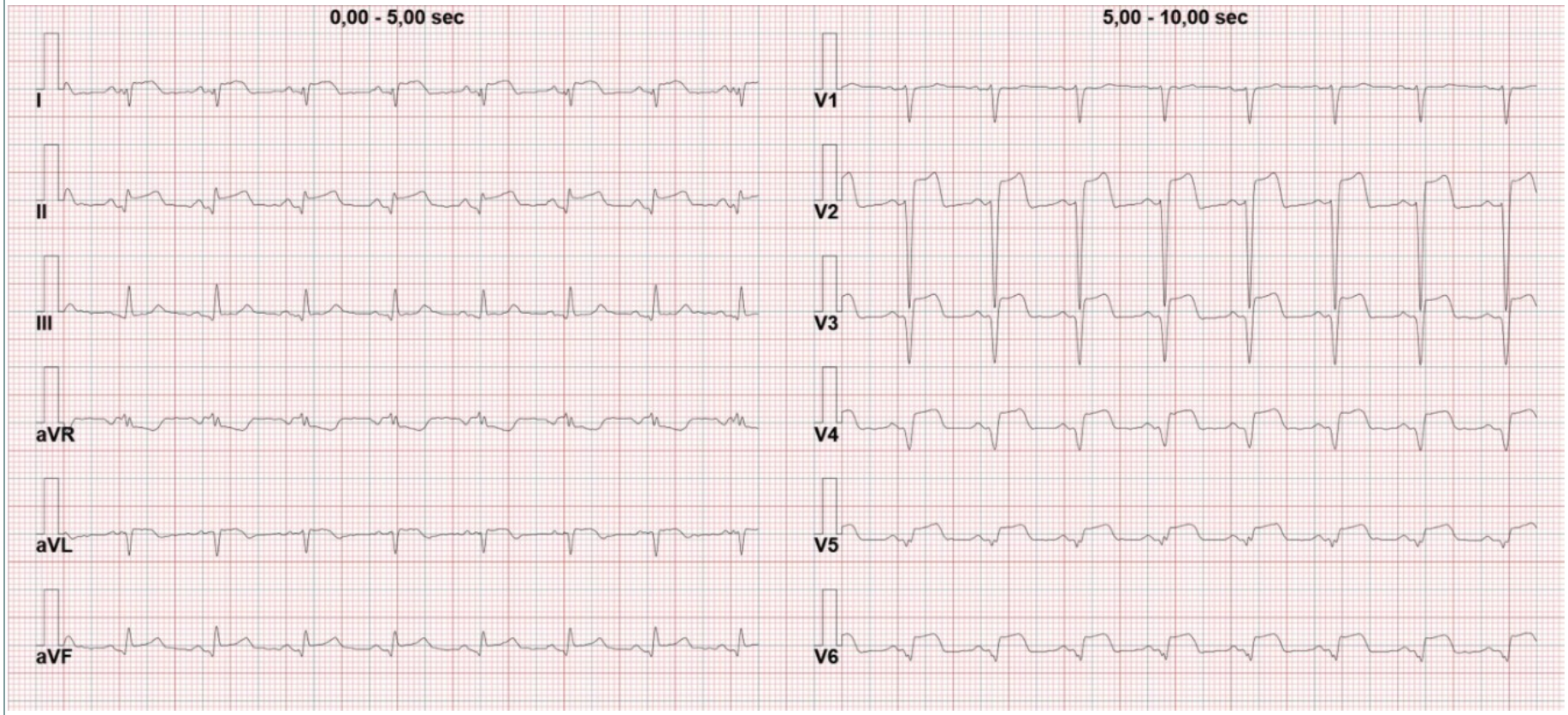
Metodo utilizzato: Citometr

SgWBC		E9/L	(5,2 - 12,4) *
SgRBC		E12/L	(4,7 - 6,1) *
SgHGB		L	(140 - 180) *
SgHCT	**25** %		(0,42 - 0,52) *
SgMCV	**95,50** fl.		(80 - 94) *
SgMCH	**33,90** pg		(27 - 31) *
SgMCHC	**355,00** g/L **35,5** g/dL		(330 - 370)
SgRDW	**14,30** %		(11,5 - 14,5)
SgPLT	**160,00** x10E9/L		(130 - 400)

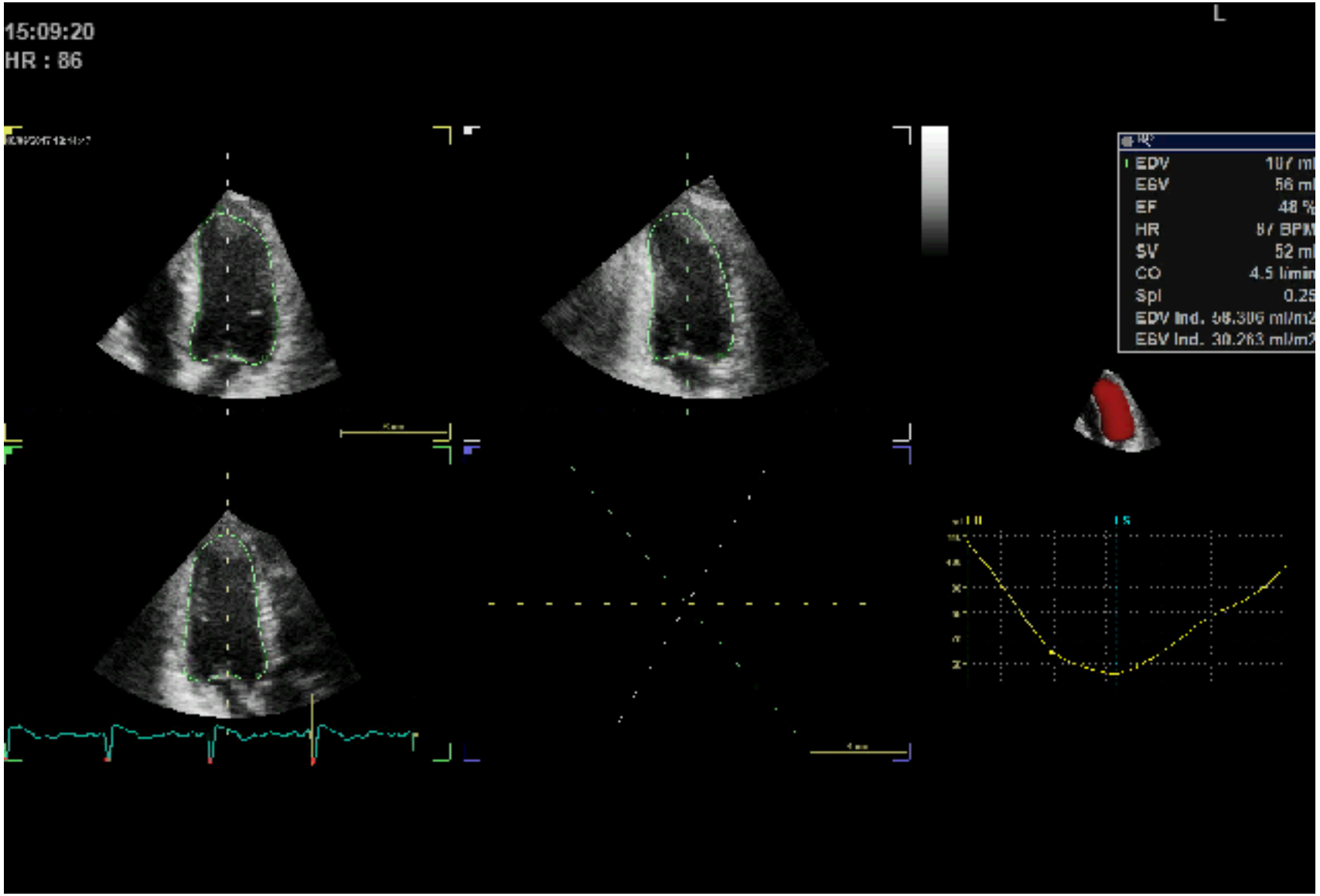
S ACIDO URICO	**0,31** mmol/L **5,21** mg/dL		(0,21 - 0,41) Metodo utilizzato: Enz.- (uricasi) con ascorbato
S SODIO	**135,00** mmol/L		(135 - 145)
S POTASSIO	**4,08** mmol/L		(3,2 - 5)
S CLORO	**104,00** mmol/L		(98 - 112)
S CREATININA	**78,00** micromol/L **0,88** mg/dL		(55 - 110) Metodo utilizzato: Jaffè cinetica - IDMS
S PROT.C REATTIVA	**65,36** mg/L		(0 - 5) * Metodo utilizzato: Immunoturbidimetrico
S TRIGLICERIDI	**0,91** mmol/L **80,6** mg/dL		(0 - 1,69) Metodo utilizzato: Enzimatico (GPO)
S COLESTEROLO totale	**4,87** mmol/L **188,32** mg/dL		(3,1 - 5,18) vedi prospetto allegato Metodo utilizzato: Colorim. (CHOD,PAP)
S COLESTEROLO HDL	**1,06** mmol/L **40,99** mg/dL		(1,03 - 2,33) Metodo utilizzato: Enzimatico omogeneo diretto
S COLESTEROLO LDL	**3,39** mmol/L **131,09** mg/dL		(0 - 3,37) * Metodo utilizzato: Calcolato sec. Friedewald
S CPK	**195,00** U/L		(46 - 171) * Metodo utilizzato: Enzim. IFCC-NAC

- Anemia: trattata con 2 sacche di emazie concentrate
- Neutropenia afebbrile trattata con isolamento, G-CFS, antibiotico-terapia con ceftriaxone, levofloxacina, difluconazolo
- Progressivo recupero dei valori ematologici

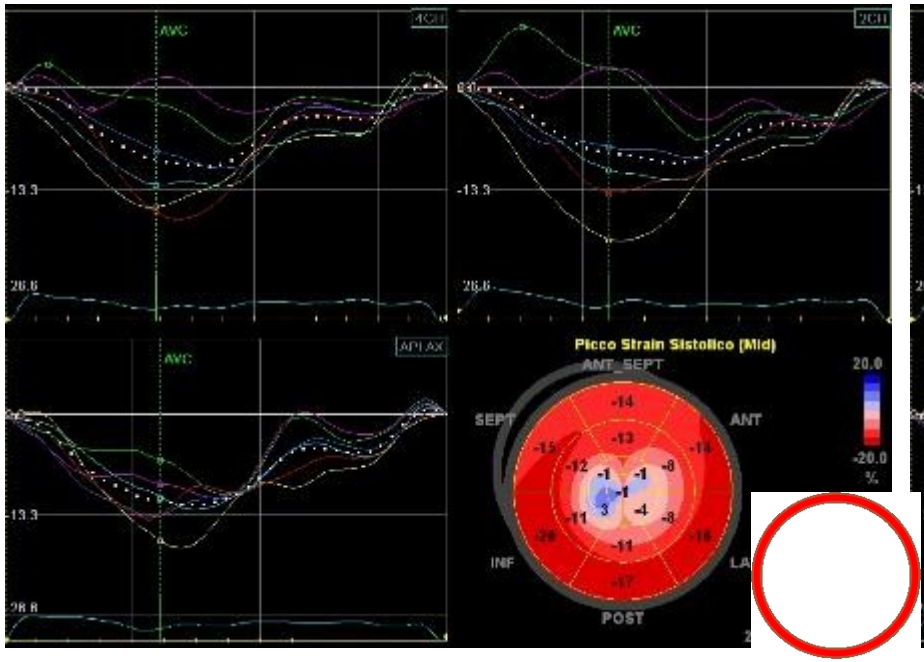
ECG in degenza



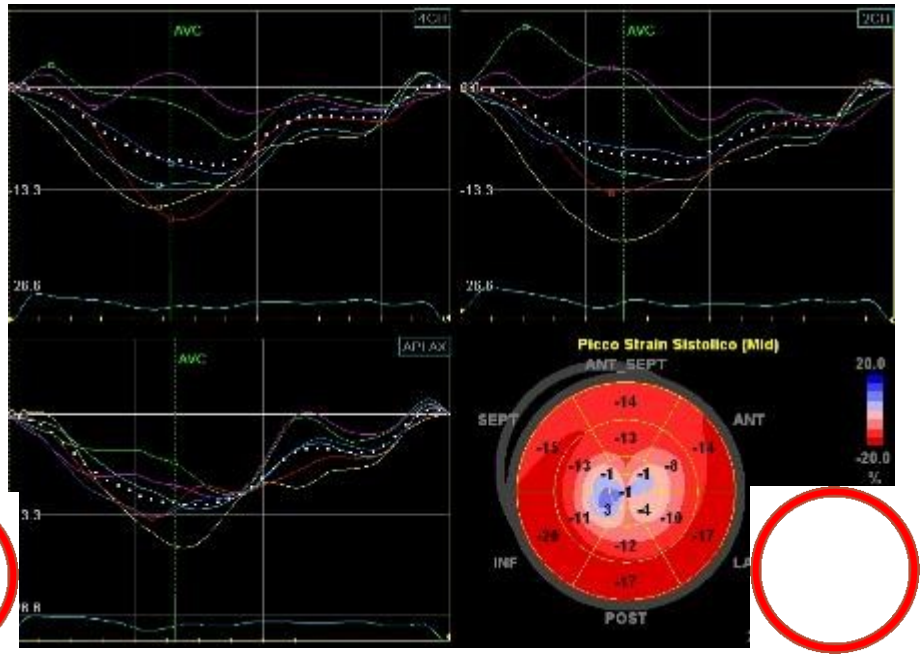
3D Eco Transtoracico



Global Longitudinal Strain (GLS)



Operatore 1

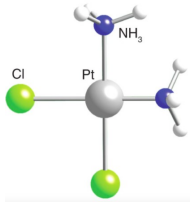


Operatore 2

Terapia alla dimissione

- Acido acetilsalicilico 100 mg 1 cp
- Ticagrelor 90 mg 1 cp BID
- Atorvastatina 80 mg 1 cp
- Bisoprololo 2.5 mg 1 cp
- Ivabradina 5 mg 1 cp BID
- Pantoprazolo 20 mg 1 cp

Attualmente asintomatico, clinicamente stabile, seminoma in remissione…



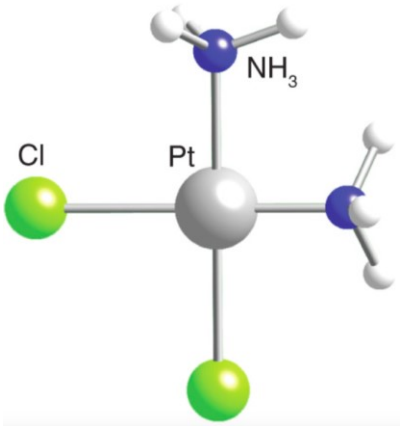
	HTN	Angina	AMI	Takotsubo	Raynaud's	Raynaud's Stroke	PAD		
Antimetabolites									
5-Fluorouracil		X	X	X	X				
Capecitabine		X	X	X	X				
Gemcitabine		X	X		X				
Antimicrotubule agents									
Paclitaxel	X	X	X						X
Alkylating agents									
Cisplatin	X	X	X		X	X	X		
Cyclophosphamide		X						X	
Antitumor antibiotics									
Bleomycin		X	X		X	X		X	

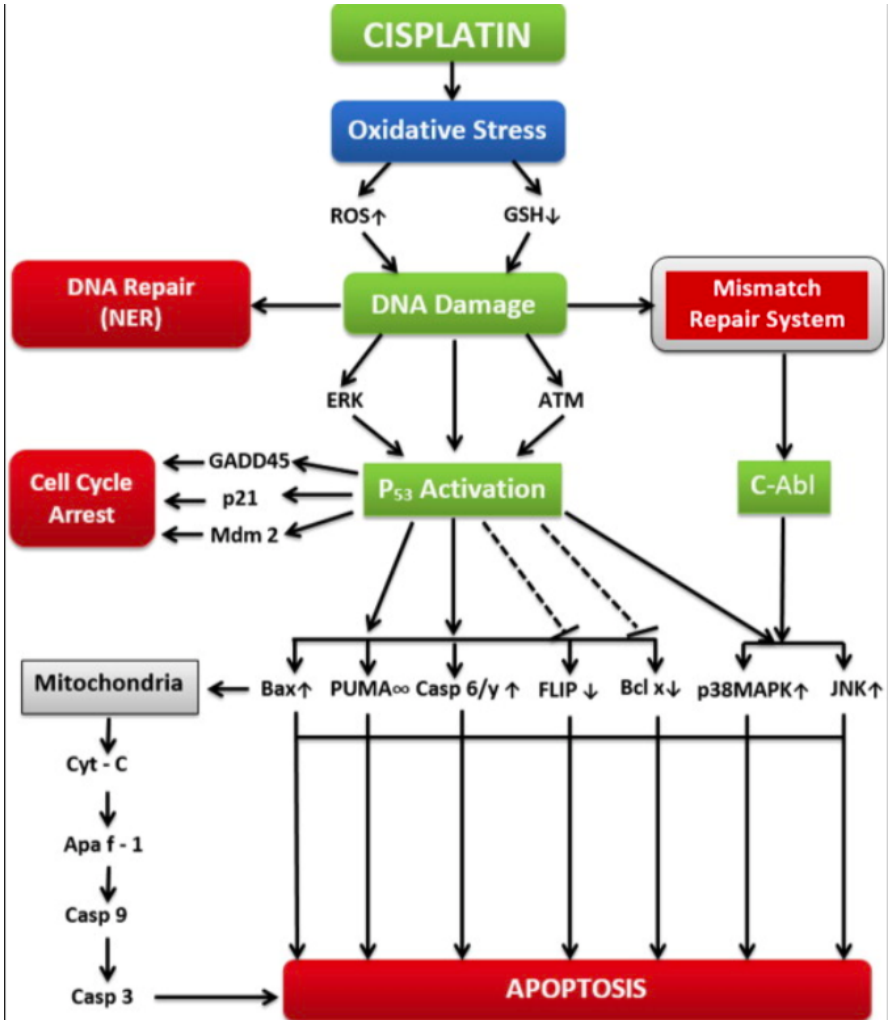
Sindromi coronariche acute	Cisplatino (12.9%) 5-fluorouracile (4%) Capecitabina (<4%) Bleomicina (<3%) Vinorelbina Vinblastina Interleuchina-2 Bevacizumab e agenti antiangiogenetici Bortezomib
Fenomeno di Raynaud	Bleomicina
Ipertensione arteriosa	Bevacizumab (23.6%) Soratinib (21%) (17-47%) Sunitinib (21.6%) Cisplatino Interferone-alfa

Table 7 Pathophysiological mechanisms of coronary artery disease in cancer treatment[7,60,81,99,117–123]

Agent	Pathophysiological mechanism	Risk of coronary artery disease and acute coronary syndrome
Fluoropyrimidines (5-FU, capecitabine, gemcitabine)	• Endothelial injury • Vasospasm	• Up to 18% manifest myocardial ischaemia • Up to 7–10%: silent myocardial ischaemia
Platinum compounds (cisplatin)	• Procoagulant status • Arterial thrombosis	• 20-year absolute risk of up to 8% after testicular cancer • 2% risk of arterial thrombosis
VEGF inhibitors (bevacizumab, sorafenib, sunitinib)	• Procoagulant status • Arterial thrombosis • Endothelial injury	• Risk of arterial thrombosis: bevacizumab 3.8%, sorafenib 1.7%, sunitinib 1.4%
Radiotherapy	• Endothelial injury • Plaque rupture • Thrombosis	• 2–7-fold increased relative risk of myocardial infarction • Cumulative 30-year coronary events incidence of 10% in Hogdkin lymphoma survivors • Risk proportional to irradiation dose

Tossicità da Cisplatino





Thrombosis Research 99 (2000) 503–509

ORIGINAL ARTICLE

Cisplatin Triggers Platelet Activation

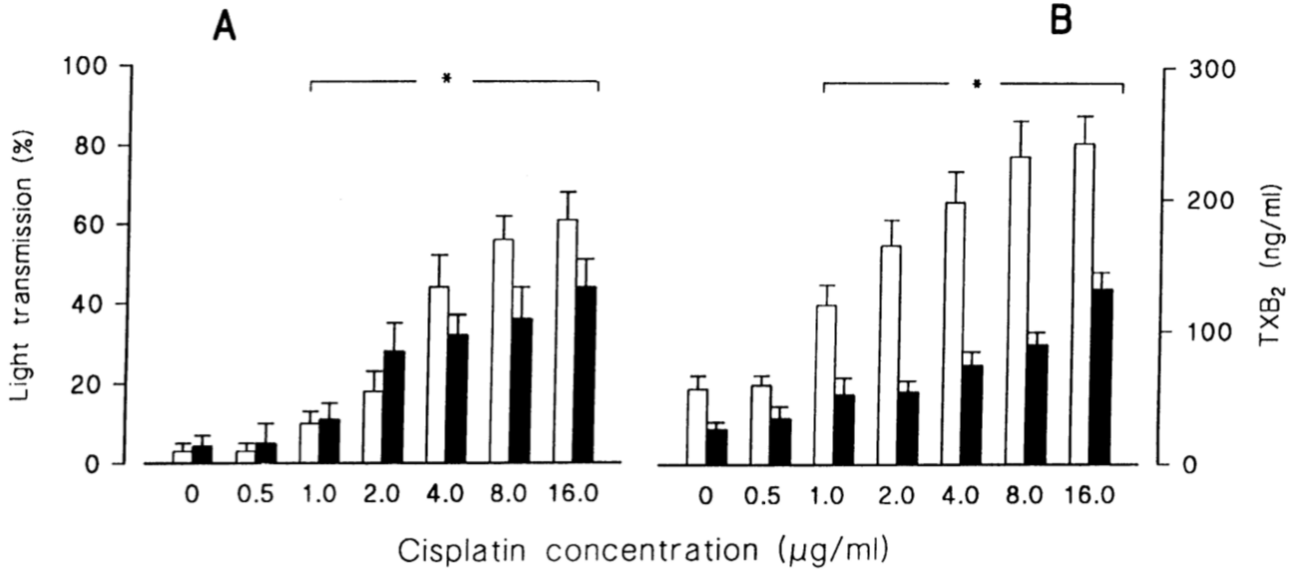
Giuseppina I. Togna, Anna Rita Togna, Matteo Franconi and Luciano Caprino
Department of Human Physiology and Pharmacology, University of Rome "La Sapienza," Rome, Italy

(Received 4 February 2000 by Editor N. Semeraro; revised/accepted 22 May 2000)

Table 1. In vitro effects of cisplatin on human platelet aggregation and thromboxane production induced by aggregating concentrations of sodium arachidonate and collagen

Treatment (μg/ml)	Sodium arachidonate, 150 μg/ml PRP[1]		Collagen, 6 μg/ml PRP[1]	
	%LT[2]	TXB_2[3] (ng/ml)	%LT[2]	TXB_2[3] (ng/ml)
Saline	73±4	87±11.0	63±4	51±6.2
Cisplatin 0.5	75±11	93±7.8	67±8	57±4.8
Cisplatin 1.0	78±9	151±13.8*	69±11	70±10.4
Cisplatin 2.0	85±10	183±19.6*	73±7	88±8.2*
Cisplatin 4.0	81±11	217±18.3*	72±5	103±10.1*
Cisplatin 8.0	84±9	253±22.2*	70±8	121±9.5*
Cisplatin 16.0	71±7	243±21.0*	58±7	161±14.6*

Data are the mean±SD for 6 independent experiments.
[1] *PRP:* Platelet-rich plasma
[2] % LT (Light Transmission)=PRP aggregation rate; Platelet-poor plasma=100% LT
[3] TXB_2, Thromboxane B_2.
* Significantly different from control, $p<0.01$



CLINICAL TRIAL

Acute Changes in Endothelial Function in Germ Cell Tumor Patients Treated With Cisplatin and Untreated Germ Cell Tumor Controls

First received on October 13, 2011. Last updated on November 1, 2018.

VOLUME 23 · NUMBER 36 · DECEMBER 20 2005

JOURNAL OF CLINICAL ONCOLOGY

ORIGINAL REPORT

Acute Chemotherapy-Induced Cardiovascular Changes in Patients With Testicular Cancer

Janine Nuver, Andries J. Smit, Jan van der Meer, Maarten P. van den Berg, Winette T.A. van der Graaf, Martin T. Meinardi, Dirk Th. Sleijfer, Harald J. Hoekstra, Anne I. van Gessel, Arie M. van Roon, and Jourik A. Gietema

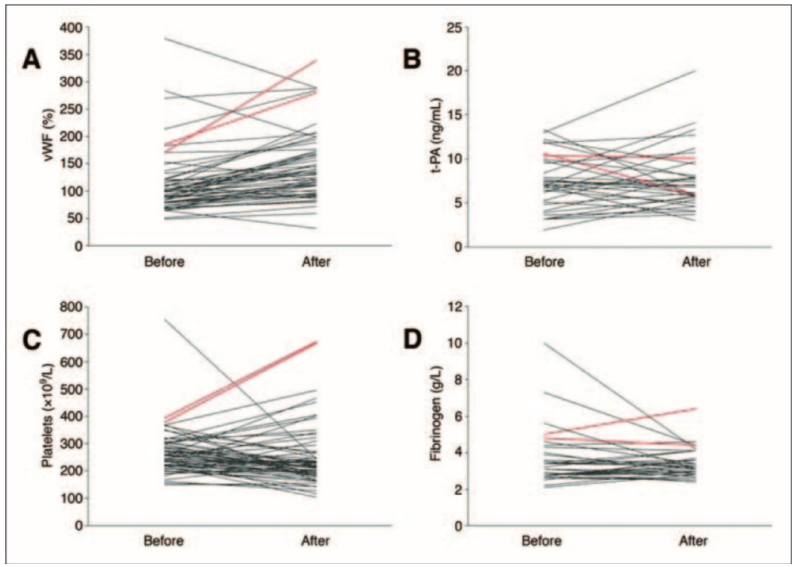


Table 4. Vascular Structure and Function Before and After Chemotherapy

Variable	No.	Before Mean	Before Median	Before Interquartile Range	After Mean	After Median	After Interquartile Range	Difference Score* Median	Difference Score* Interquartile Range	P†
CCA IMT, mm	57	0.560	0.541	0.468-0.640	0.581	0.543	0.500-0.649	0.025	−0.011-0.071	.02
Baseline BA diameter, μm	57	4,638	4,771	4,223-5,208	4,774	4,770	4,304-5,268	166	−173-392	.09
Absolute postischemic BA dilation, μm	56	355	340	149-541	409	393	249-565	20	−189-228	.67
Flow-mediated vasodilation, %	56	8.2	7.3	3.2-12.6	9.0	8.5	4.9-12.3	−0.1	−4.1-4.9	.94

Abbreviations: CCA, common carotid artery; IMT, intima-media thickness; BA, brachial artery.
*Calculated as (variable value before start of chemotherapy − variable value after chemotherapy).
†Wilcoxon test.

Conclusion

In testicular cancer patients treated with cisplatin-based chemotherapy, we found an increase in plasma von Willebrand factor levels and in the intima-media thickness of the carotid artery. These changes may indicate chemotherapy-induced vascular damage and be of prognostic significance for the development of cardiovascular complications in the long term.

THE LANCET

RESEARCH LETTERS | VOLUME 355, ISSUE 9209, P1075-1076, MARCH 25, 2000

Circulating plasma platinum more than 10 years after cisplatin treatment for testicular cancer

JA Gietema, MDc • MT Meinardi, MD • J Messerschmidt, MSc • T Gelevert • F Alt, PhD • Prof DRA Uges, PhD •

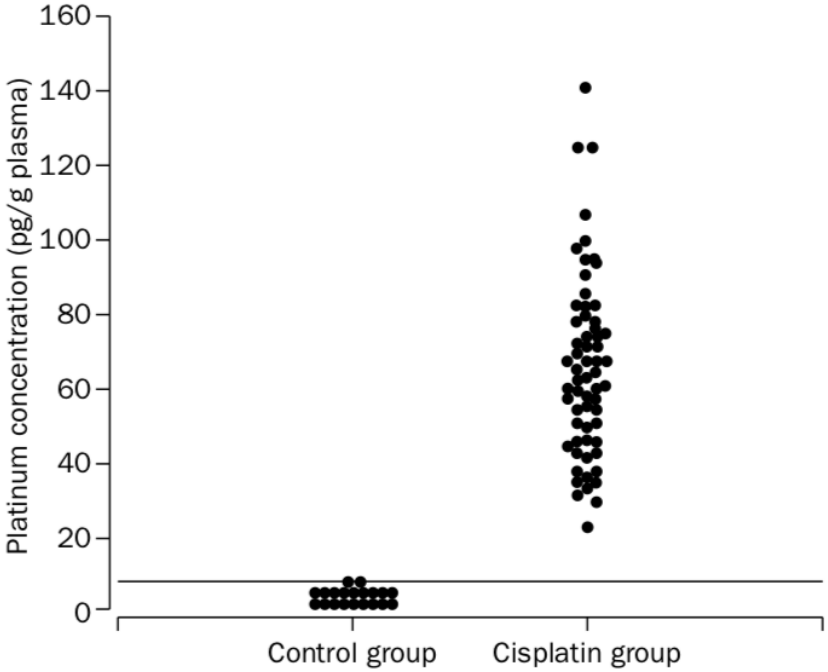


Figure 1: **Plasma platinum concentrations of 61 cured testicular cancer patients 10–20 years after cisplatin combination chemotherapy and 20 cured testicular cancer patients 10–20 years after orchidectomy**
Horizontal line=limit of quantification of platinum (6 pg/g plasma).

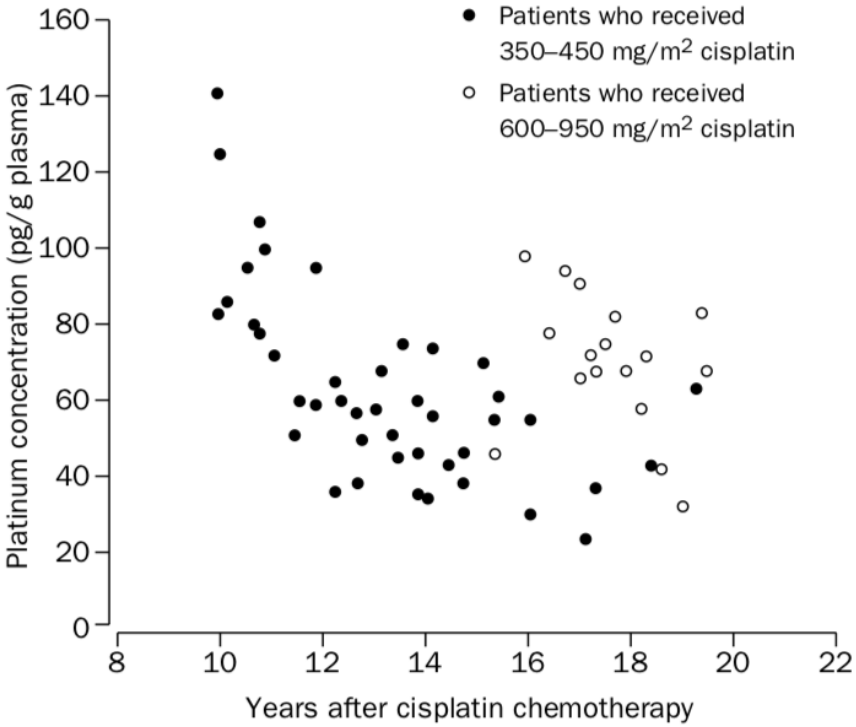


Figure 2: **Concentration-time plot of plasma platinum concentrations of 61 cured testicular-cancer patients 10–20 years after cisplatin combination chemotherapy**

Journal of Clinical Oncology®
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Cardiovascular Morbidity in Long-Term Survivors of Metastatic Testicular Cancer

2000

Table 3. Cardiovascular Risk Factors in Long-Term Survivors of Testicular Cancer After Chemotherapy (n = 62) or Orchidectomy Only (n = 40)

		Chemotherapy Group (n = 62)				Group With Stage I Disease (n = 40)				
				Patients With Abnormal Result				Patients With Abnormal Result		
Risk Factor	Normal Value	Mean ± SD	Range	No.	%	Mean ± SD	Range	No.	%	P*
Dyslipidaemia				49	79‡			23	58‡	.02
Total cholesterol	< 5.2 mmol/L	6.2 ± 1.2†	4.3-9.7	49	79‡	5.6 ± 1.2†	3.5-9.8	23	58‡	.03
HDL cholesterol	> 0.9 mmol/L	1.0 ± 0.3†	0.3-1.7	19	35‡	1.1 ± 0.3†	0.7-2.1	10	25‡	.16
LDL cholesterol	< 3.4 mmol/L	4.3 ± 1.1†	1.1-6.5	49	79‡	3.9 ± 1.1†	2.2-8.0	24	62‡	.18
Total cholesterol/HDL cholesterol ratio	< 5.0	6.8 ± 3.0†	3.2-22	44	71‡	5.5 ± 1.9†	2.7-11	21	53‡	.02
Triglycerides	< 2.3 mmol/L	2.2 ± 2.3†	0.8-14	14	25‡	1.4 ± 0.8†	0.2-4.1	4	10‡	.02
Hypertension				24	39§			5	13§	.003
Diastolic pressure	≤ 95 mmHg	90 ± 12‖	65-140	24	39¶	82 ± 11‖	65-105	3	8¶	.003
Systolic pressure	≤ 150 mmHg	137 ± 18‖	110-190	22	35¶	123 ± 14‖	95-153	1	3¶	< .001
Family history positive for cardiovascular events				23	37			10	25	.20
Smoking										
Former smoker				20	32			10	25	.43
Current smoker				19	31			9	23	.37

treatment. The high O/E ratio (7.1; 95% CI, 1.9 to 18.3) for nonfatal myocardial infarction plus angina pectoris with proven myocardial ischemia in this cohort strongly suggests that, in long-term survivors of metastatic testicular cancer cured with cisplatin-containing chemotherapy, the cardiac event rate might be elevated compared with that of the general population.

Journal of Clinical Oncology®
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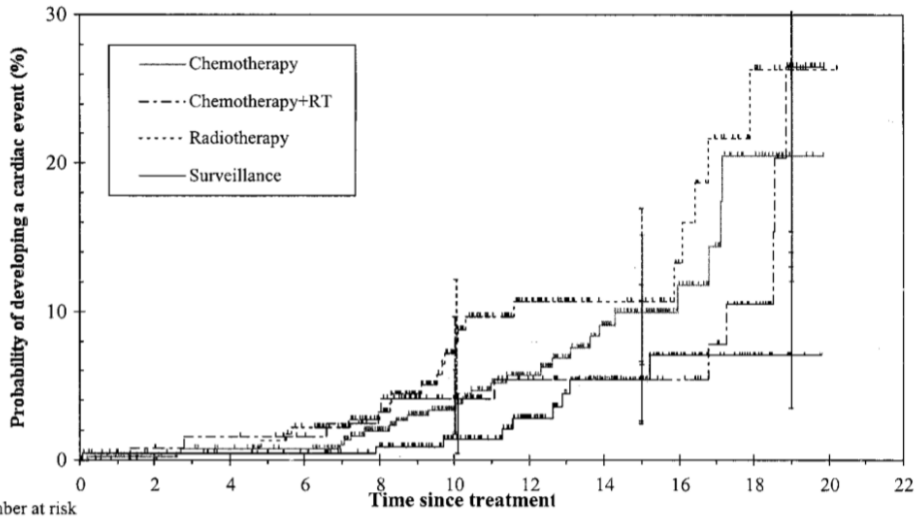
Cardiovascular Disease as a Long-Term Complication of Treatment for Testicular Cancer

R.A. Huddart , A. Norman , M. Shahidi , A. Horwich , D. Coward , J. Nicholls...



Number at risk

Chemotherapy	390	382	373	367	363	357	355	338	302	256	226	194	170	143	116	85	47	31	18	3
Chemo+RT	130	127	126	124	124	123	120	118	112	99	91	76	69	63	57	50	44	38	26	10
Radiotherapy	230	230	227	226	224	222	215	201	178	151	122	92	74	57	44	42	32	25	16	7	1
Surveillance	242	233	231	230	227	226	222	215	210	194	171	155	129	107	85	59	38	27	15	3

Conclusion: In long-term survivors of testicular cancer, we observed a two-fold or greater risk of developing cardiovascular disease. This was not due to increases in cardiac risk factors, which suggests a direct or indirect treatment effect. These data support the continued research into the minimization of treatment in good-prognosis testicular cancer.

2003

VOLUME 28 · NUMBER 30 · OCTOBER 20 2010

JOURNAL OF CLINICAL ONCOLOGY

ORIGINAL REPORT

Cardiovascular Risk Factors and Morbidity in Long-Term Survivors of Testicular Cancer: A 20-Year Follow-Up Study

Hege S. Haugnes, Torgeir Wethal, Nina Aass, Olav Dahl, Olbjørn Klepp, Carl W. Langberg, Tom Wilsgaard, Roy M. Bremnes, and Sophie D. Fosså

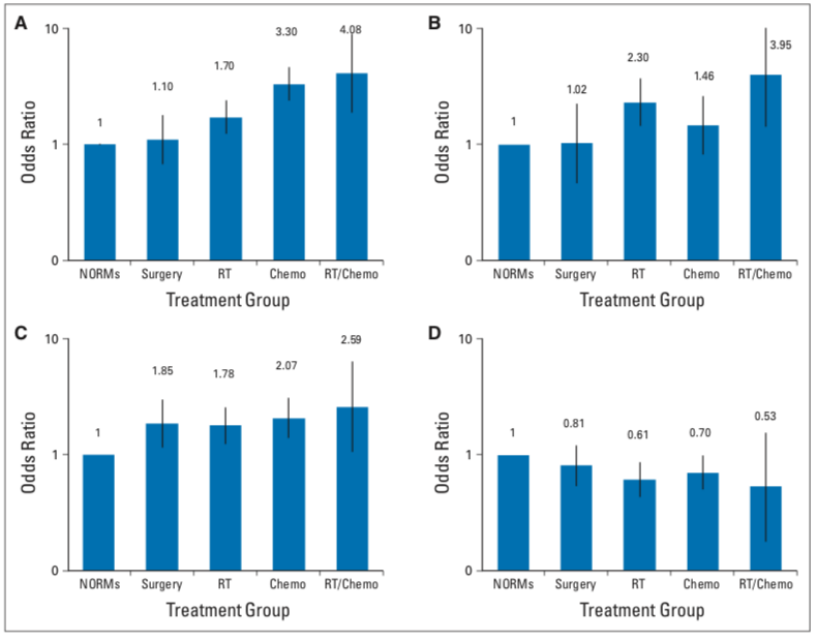
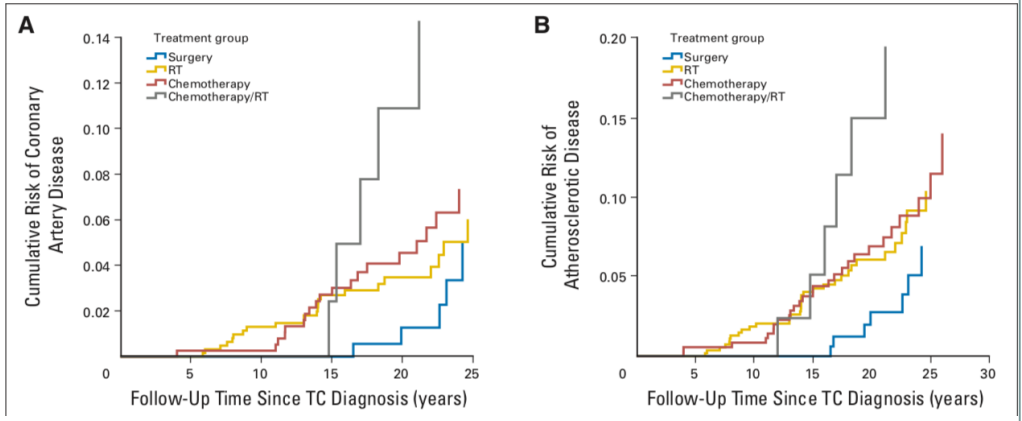


Fig 1. Odds ratios (ORs) for (A) using antihypertensive medication, (B) diabetes, (C) using lipid-lowering medication, and (D) obesity at follow-up according to treatment groups. A control group (ie, NORMs) is used as reference. Bars indicate 95% CIs for OR. RT, radiotherapy; Chemo, chemotherapy.



Conclusion

Treatment with infradiaphragmatic RT and/or cisplatin-based chemotherapy, particularly the BEP regimen, increases the long-term risk for CVD in survivors of TC.

S.#	References	Followup time	Patients Count	Hypertension	Hypercholestremia	Increased BMI
30	Meinardi et.al 2000	9-16 year	85	39%	79%	21%
31	Haugnes et.al 2010	20 years	990	22%	15%	17%
32	Dollet.al. 1986	Day 7 - 18 Months	4	None	None	None
33	Strumberg et.al. 2002	13years	32	25%	82%	48%
34	Huddart et.al. 2003	10.2 years	992	212%	1.60%	None
35	Sagstuen et.al. 2005	4-22 years	1980	50%	N/A	15%
36	Samuels et.al. 1987	1-10 years	65	None	None	None
37	Bookmeyer et.al. 1996	58 months	90	15%	30	N/A
38	Roth et.al. 1988	8.5 years	229	31%	15%	None

Acute vascular toxicity after combination chemotherapy with cisplatin, vinblastine, and bleomycin for testicular cancer

European Heart Journal. Volume 9. Issue 5. 1 May 1988. Pages 552–556.

Abstract

Twenty-one consecutive patients with testicular cancer treated with bleomycin, vinblastine, and cisplatin (PVB) were evaluated for acute vascular ischaemic events during chemotherapy. Angina pectoris occurred in 8/21 (38%) patients, a median 5·6 weeks after initiation of chemotherapy and persisted for 2·7 days. Raynaud's phenomenon was detected in seven (33%) subjects, transient ischaemia of the toes was found in six (29%) patients, one patient complained of migraine, but none had major cerebrovoscular accidents. Patientswith and without angina pectoris and/or Raynaud's phenomenon did not differ in respect of age, histology of tumor or medication. Ischaemia occurred at any time during the course of chemotherapy. No correlation was found between dosage of drugs and time of onset of ischaemic reactions. However, arterial occlusive event is a frequent and common toxicity and a result of treatment with PVB.

Non-Q-wave myocardial infarction associated with bleomycin and etoposide chemotherapy

European Heart Journal, Volume 12, Issue 6, 1 June 1991, Pages 748–750,

Abstract

During chemotherapy with bleomycin and etoposide a 28-year-old male, suffering from germ-cell cancer, developed acute myocardial infarction. Under treatment with heparin and aspirin the patient revealed no Q-waves in ECG and recovery was without complications. Four weeks after onset of infarction, thallium-201 scintigraphy showed only a small irreversible, posteroseptal perfusion defect; coronary angiography was not performed. The chemotherapy regimen was continued and modified to etoposide as well as cisplatin and ifosfamide without recurrence of cardiac symptoms or ECG changes.

Vasospastic Angina Likely Related to Cisplatin-containing Chemotherapy and Thoracic Irradiation for Lung Cancer

Masaaki FUKUDA*, **, Mikio OKA**, Naomi ITOH*, Toshifumi SAKAMOTO*, Hideki MORI*, Akira HAYAKAWA* and Shigeru KOHNO**

Vasospastic angina is rarely observed during cancer treatment. The present report describes two males with lung cancer, aged 73 and 61, who developed vasospastic angina during combination treatment of cisplatin-containing chemotherapy and thoracic irradiation. As both patients have smoked and their ages are typical for patients with coronary artery disease, such events may be incidental. However, oncologists should be aware of the possible development of myocardial ischemia during or following administration of antineoplastic agents, especially in elderly patients with pre-existing coronary risk factors or a history of thoracic radiotherapy.
(Internal Medicine 38: 436–438, 1999)

DOI: 10.7860/JCDR/2017/25546.10025

Case Report

Internal Medicine Section

Cisplatin Induced Acute Myocardial Infarction and Dyslipidemia

LALITH PRABHAKAR HANCHATE[1], SHIMPA RAKESH SHARMA[2], SADHANA MADYALKAR[3]

ABSTRACT

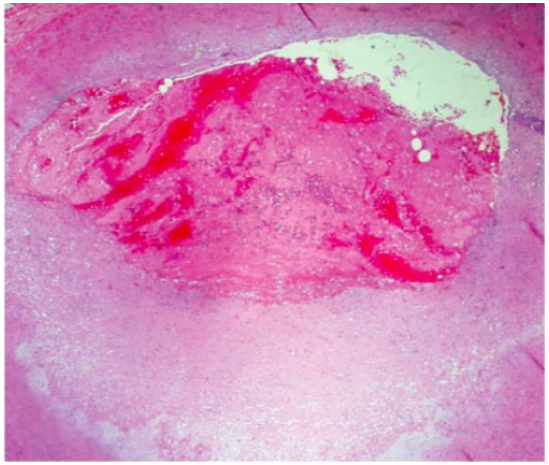
Myocardial infarction commonly occurs due to rupture of atherosclerotic plaque in the coronary arteries and is manifested on coronary angiography. Myocardial infarction with angiographically normal coronary arteries can occur due to hypercoagulable state, coronary spasm, embolism, arteritis, congenital condition and drugs. Illicit drugs such as cocaine, amphetamines and marijuana are known to cause myocardial infarction. Of the many medications that are associated with cardiovascular toxicity, chemotherapeutic agents form a large group with both acute and delayed cardiotoxicity. We present a case of acute myocardial infarction in a patient with no organic coronary artery disease on the Bleomycin, Etoposide and Cisplatin (BEP) regimen for treatment of testicular seminoma. Patient developed symptoms of Acute Coronary Syndrome (ACS) after his third cycle of the regimen. This case highlights the need for prior careful cardiovascular evaluation, close monitoring and use of strategies to prevent cardiovascular morbidity in patients on cisplatin therapy.

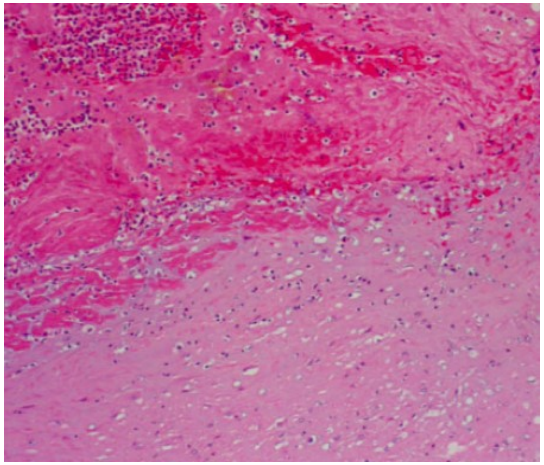
Keywords: Anterior myocardial infarction, Coronary angiography, Coronary vasospasm, Testicular seminoma

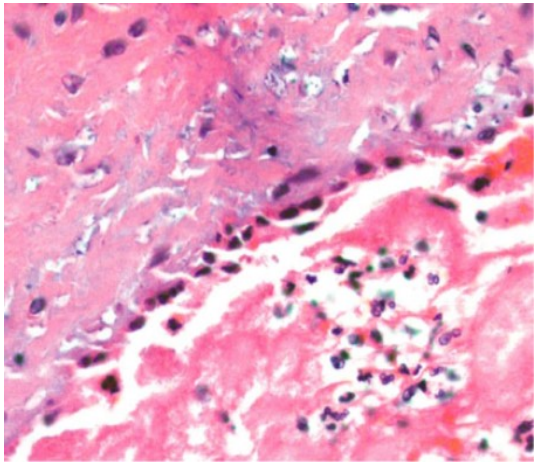
JOURNAL OF FORENSIC SCIENCES

Cardiac Sudden Death as a Result of Acute Coronary Artery Thrombosis During Chemotherapy for Testicular Carcinoma

01 September 2010







Abstract: We report the first acute coronary fibrin thrombus arising upon atherosclerosis detected at autopsy in a man receiving chemotherapy for testicular carcinoma. The decedent was a smoker with no other known atherosclerotic risk factors. Histology revealed superficial atherosclerotic plaque erosion with endothelial necrosis and no intraplaque hemorrhage. A focus of intimal lymphoid infiltrates was noted away from the plaque. These findings raise the possibility of chemotherapy-induced vascular damage as a factor in thrombogenesis. A review of Pubmed was performed which documented clinical reports of an association of chemotherapy with acute cardiac ischemia but no well described autopsy findings. Our case highlights the need for careful assessment of the coronary system in chemotherapy patients dying suddenly, particularly in the absence of significant atherosclerotic risk factors. Such postmortem examination will ensure thorough death investigation and may elucidate the pathogenesis of thrombosis with potential reduction in cardiac ischemic risks of chemotherapy patients.

Myocardial infarction and other major vascular events during chemotherapy for testicular cancer

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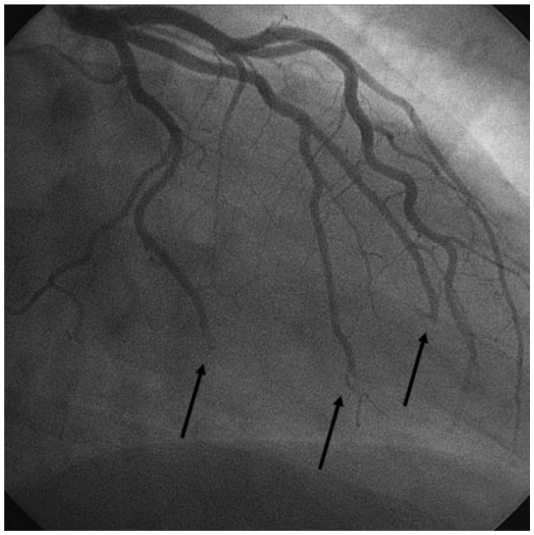
Background: Chronic vascular morbidity resulting from chemotherapy for testicular germ-cell cancer (TGCC) is recognized. Cardiovascular events (CVEs) occurring early during chemotherapy are less understood. We evaluated the incidence and clinical features of CVEs associated with chemotherapy of TGCC.

Patients and methods: A questionnaire was sent to 355 institutions in Germany to explore for early CVEs occurring during 1996–2008. To assess the relative incidence of CVEs, the number of events was put into relation to the total number of patients treated during the time span (n = 8233, calculated from national database). The response rate was 79%.

Results: Twenty cases with myocardial infarction (MI), 3 with cerebral stroke, and 2 with arterial thrombosis were recorded. The estimated incidence of MI and of all CVEs during chemotherapy is 0.24% [95% confidence intervals (CIs) 0.137% to 0.349%] and 0.30% (95% CI 0.188% to 0.423%), respectively. This estimate represents a minimum figure because the calculation is on the basis of simplifications. Six MI patients had no risk factors. Coronary angiography was indicative of thromboembolic rather than atherosclerotic origin of MI.

Conclusions: There is a small but definite risk of major early CVE associated with chemotherapy of TGCC. Physicians caring for TGCC patients must be aware of this hazard.

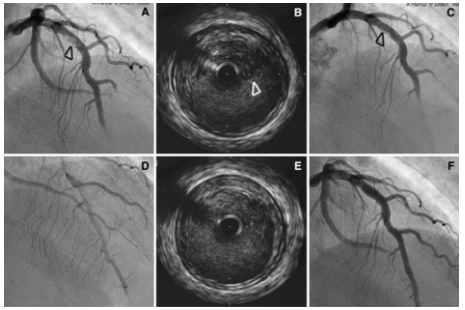


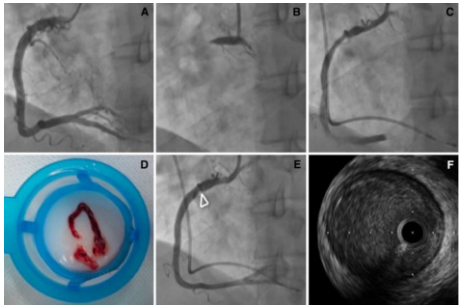
Heart Vessels (2012) 27:634–638
DOI 10.1007/s00380-011-0222-5

CASE REPORT

Primary percutaneous coronary intervention and intravascular ultrasound imaging for coronary thrombosis after cisplatin-based chemotherapy

Daisuke Ito · Jun Shiraishi · Takeshi Nakamura · Naoki Maruyama · Yumi Iwamura · Sho Hashimoto · Masayoshi Kimura · Akihiro Matsui · Hirokazu Yokoi · Masayasu Arihara · Hidekazu Irie · Masayuki Hyogo · Takatomo Shima · Yoshio Kohno · Akiyoshi Matsumuro · Takahisa Sawada · Hiroaki Matsubara





HIPPOKRATIA 2013, 17, 2:187-188

CASE REPORT

Bleomycin cardiotoxicity during chemotherapy for an ovarian germ cell tumor

Didagelos M, Boutis A, Diamantopoulos N, Sotiriadou M, Fotiou C

1st Department of Clinical Oncology-Chemotherapy, Theagenio Cancer Hospital, Thessaloniki, Greece

Abstract

Introduction: Platinum-based chemotherapeutic regimens, including BEP (bleomycin, etoposide, cisplatin) represent the standard of care, first line therapy in non-epithelial ovarian tumours. Cardiovascular toxicity is a rare adverse effect of bleomycin.

Case Report: A 41-year-old woman with ovarian granulosa tumor, treated with first line BEP chemotherapy experienced chest discomfort rapidly progressing to severe precordial pain during bleomycin infusion. The infusion was stopped and electrocardiographic changes indicative of myocardial ischemia were revealed. Anti-anginal and anti-thrombotic treatment was introduced. Cardiac enzymes were not elevated and echocardiographic findings showed no wall motion abnormalities. Twenty four hours after the episode the elctrocardiographic changes insisted and chemotherapy was decided to be continued, excluding bleomycin, with no symptom recurrence.

Discussion: Cardiovascular complications pose a rare but potential fatal adverse effect of BEP chemotherapy and should be carefully addressed, especially in patients with additional cardiovascular risk factors. Physicians dealing with bleomycin-based therapies may find this knowledge useful for a more comprehensive evaluation of chest pain syndromes in those patients. Hippokratia 2013, 17, 2: 1787-188

Keywords: BEP chemotherapy, ovarian cancer, cardiotoxicity, myocardial ischemia, chest pain

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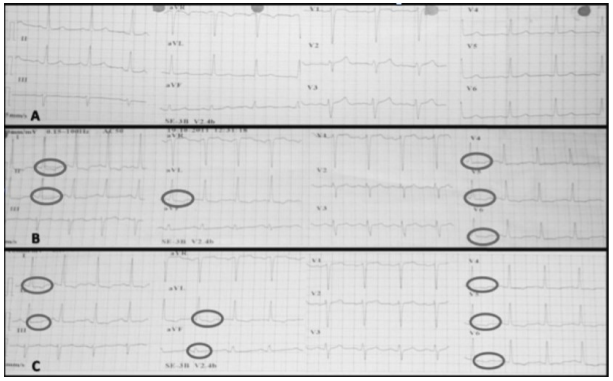

Conclusioni

- Il Cis-platino determina aumento del rischio cardiovascolare a lungo termine
- I meccanismi con cui induce alterazioni a livello dei tessuti sani non sono definitivamente compresi ma includono danno a livello endoteliale e aumento della reattività piastrinica
- Sia pur meno frequenti, vi sono numerose segnalazioni in letteratura relative a complicanze CV acute, per lo più legate a fenomeni di vasospasmo determinanti e a fenomeni trombotici
- Nel caso del nostro paziente riteniamo probabile che la terapia abbia agito destabilizzando un placca già presente