CASE REPORT

Acute disseminated intravascular coagulation following ICD lead extraction

Nasir Shariff,¹ Madhurmeet Singh,¹ Alaa Shalaby^{1,2}¹Heart and Vascular Institute, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, USA²Department of Cardiology, VA Pittsburgh Healthcare System, Pittsburgh, Pennsylvania, USA**Correspondence to**
Dr Alaa Shalaby,
alaa.shalaby@va.gov

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SUMMARY

We present an unusual case of disseminated intravascular coagulopathy (DIC) complicating percutaneous laser-assisted lead extraction. DIC has not been previously reported in association with lead extraction. It is possible to have occurred following the denudement of venous endothelium and exposure of underlying fibrous tissue. Practitioners need to be aware of this rare but potentially fatal complication of transvenous lead extraction.

BACKGROUND

Disseminated intravascular coagulation (DIC), a catastrophic phenomenon complicating certain medical conditions or surgical procedures, is characterised by concomitant bleeding and intravascular thrombosis.¹ The diagnosis rests on evidence of widespread simultaneous activation of native prothrombotic and thrombolytic systems resulting in deposition of intravascular fibrin, thrombotic occlusion of vessels, end organ damage and widespread haemorrhagic complications and reflected in laboratory values indicating overuse of clotting components with resultant excessive production of clotting by-products. The associated morbidity and mortality can be high particularly if not recognised early. Cardiovascular implantable electronic device lead extraction is associated with well-recognised complications, however DIC has not been recognised as one of them. We present a case of acute DIC following transvenous lead extraction.

CASE PRESENTATION

A 66-year-old man with ischaemic cardiomyopathy presented with inappropriate shocks due to abnormal sensing and noise detection on the ventricular lead of his implantable cardioverter defibrillator (ICD). Twelve years prior to presentation the patient sustained a myocardial infarction followed by coronary artery bypass grafting surgery. He then received an ICD for primary prophylaxis 6 years prior to current presentation on account of low ejection fraction (25%) despite optimal medical therapy. This was a single-chamber device with a dual shocking coil ventricular lead (model no. 7001 Riata ST, St Jude Medical, Sylmar, California, USA). The ICD implant was placed on the right side in view of previous extensive left-sided neck surgery and radiation therapy for treatment of squamous cell carcinoma of the tongue. Two months prior to presentation the ICD generator was changed out as the battery life had reached replacement indicator. Since the ICD lead had

become subject of a manufacturer recall, internal cable separation was ruled out at the time of generator changeout by fluoroscopic examination. Other pertinent medical history included diabetes mellitus and chronic kidney disease. Specifically, he had no prior bleeding, thrombosis or anticoagulation use history. At this point it was decided to extract the ventricular lead in preparation for replacing it with a new ICD lead.

At the time of the extraction procedure the patient was receiving the following medications: furosemide 20 mg/day, metoprolol succinate 100 mg/day, omeprazole 20 mg/day, cholecalciferol 400 units/day, finasteride 5 mg/day and aspirin 81 mg/day. In the electrophysiology lab, right femoral arterial and venous access sheaths were inserted to allow for continuous blood pressure monitoring and to provide wide bore venous access in case of emergency, respectively. Under general anaesthesia, the lead was extracted from the vascular system with the assistance of a laser sheath (Spectranetics, Inc, Colorado Springs, Colorado, USA). Brief hypotension during traction of the distal end of the lead inserted in the right ventricle rapidly reversed after extraction with no particular intervention. Apart from flattening of the lead in the pocket which had been noted on chest X-ray there was no evidence of lead breakdown or cable separation. Implantation of a new lead was uneventful. Femoral arterial and venous access sheaths were removed and local pressure was held until haemostasis was achieved. The procedure time was 90 min and fluoroscopy time 8.2 min.

Four hours after the procedure, the previously well-controlled femoral access site developed an enlarging femoral haematoma at the vascular entry site despite attempted control with ultrasound-guided manual pressure. Vascular ultrasound study ruled out a pseudo-aneurysm and demonstrated expanding thrombus in the thigh. At the same time the patient was noted to have bleeding from the peripheral intravenous access sites in the arms, followed by the development of epistaxis. An emergent complete blood count was drawn and a significant drop in haemoglobin was noted. A concomitant platelet count drop was noted and emergent coagulation studies were taken. These were significant for an elevated International Normalised Ratio of more than 19 with activated partial thromboplastin time of more than 200 s. A repeat value confirmed the above and DIC was suspected. This was confirmed with the finding of D-dimer >4000 mg/L, fibrinogen <50 mg/dL and lactate dehydrogenase 270 IU/L (table 1). These findings indicated blood loss, fibrinogen and platelet depletion in the widespread

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Table 1 Summarises the abnormal laboratory values associated with the acute illness compared with baseline

Test	Baseline	Peak/Nadir	Reference range
Activated partial thromboplastin time (s)	27.5	200.8	22.7–34.1
Prothrombin time (s)	12.9	>120	11.7–14.6
INR	1.0	>19	<1.3
Haemoglobin (g/dL)	12	8.2	14–18
Haematocrit (%)	34.8	25.2	42–52
Platelet (K/mm ³)	240	39	130–400
Fibrinogen (mg/dL)		<50	225–480
D-dimer (FEU ng/mL)		>4000	0–500
Haptoglobin (mg/dL)		85	16–200
Lactate dehydrogenase (IU/L)		270	100–190
Blood urea nitrogen (mg/dL)	14	42	6–20
Creatinine (mg/dL)	1.43	3.26	0.5–1.2
Estimated GFR (ml/min)	49.3	19.1	>60

GFR, glomerular filtration rate; INR, International Normalised Ratio.

thrombotic process and breakdown products of fibrinogen and haemoglobin due to extensive clotting and red blood cell sludging in thrombotic capillaries, respectively. End organ damage attendant with thrombosis was reflected in acute renal injury perhaps exacerbated by the brief hypotension during the case. Liver function tests however remained unchanged.

TREATMENT

Supportive therapy with blood products to replace lost blood components included 6 units packed red blood cells, 6 packs of platelets, 4 units of fresh frozen plasma and 1 unit of cryoglobulin. Over the next 48 h with the above interventions, he improved clinically with stabilisation of the thigh haematoma, haemoglobin and platelet counts. His fibrinogen level improved to 329 mg/dL.

OUTCOME AND FOLLOW-UP

Acute renal injury resolved gradually over the following weeks. CT scans ruled out thoracoabdominal malignancy or aneurysm and a prostate-specific antigen level was normal. He was discharged from the hospital 2 weeks postprocedure in stable condition. During follow-up over the following 6 months he remained clinically stable with no evidence of recurrent haematological abnormalities, residual end organ damage or recurrent malignancy.

DISCUSSION

In this case, we describe a case of acute DIC following an uneventful lead extraction for the purpose of lead management. We postulate that the lead extraction was associated with debridement of the venous intimal layer and exposure of tissue factor starting the coagulation/thrombolysis cascade. Other potential aetiology of DIC including sepsis, malignancy, medications or allergic reaction is not supported in this case.

Acute DIC is a serious complication associated with high mortality and morbidity. The reported mortality ranges from 40% to 80%.^{2 3} Early recognition of DIC, establishing and correcting the aetiology of DIC when possible, and providing supportive care are factors that are of central importance in the management of acute DIC. DIC can occur as a result of various conditions including sepsis, trauma and cancer.⁴ Major initiating factors are the release or expression of tissue factor, usually involving entry of tissue thromboplastins into the circulation,

extensive injury to vascular endothelium exposing tissue factor or enhanced expression of tissue factor by monocytes in response to endotoxin and various cytokines.^{5 6}

Cardiac implantable electronic device use has increased dramatically during the last decade with more than four million active devices and one million new leads implanted annually.⁷ Along with this increase has been a burgeoning increase in extraction of intravascular leads. Accepted indications for lead removal include infection, venous occlusion, and as part of a lead management strategy in cases of lead redundancy due to lead malfunction or fracture. Although the majority of lead extractions are uneventful, complications such as cardiac perforation, vascular injury, tricuspid regurgitation, thrombus or vegetation embolism have been reported with incidence ranging from 2% to 4%.^{8 9} To our knowledge, this is the first reported case of acute DIC occurring due to ICD lead extraction. It is not clear why this may not be more commonly encountered with the increases in lead extraction using either traditional or excimer laser tools. The low-temperature excimer laser operates in the ultraviolet spectrum at 308 nm to ablate target tissue at a depth of 50µ, theoretically limiting the extent of venous wall injury related to extraction. Either method would result in endothelial denudement and exposure of tissue activating factors inciting the initiation of the coagulation cascade. DIC resulting from dental extraction has been reported as a herald of malignancy or large vessel aneurysm and, in at least one case, with no underlying pathology other than extraction, as an inciting trigger.^{10 11} Our patient whose previously diagnosed and treated cancer was closely monitored demonstrated no evidence of recurrence or de novo malignancy. It is speculative to consider previously treated malignancy as a predisposing factor for DIC in this case.

Learning points

- Our case highlights an unusual complication of lead extraction, DIC.
- In this case uncontrolled bleeding from vascular access sites was the initial diagnostic lead.
- Prompt recognition and intervention averted a deadly outcome.

Contributors All authors contributed meaningfully to this manuscript and had full access to the source material.

Competing interests None.

Patient consent Obtained.

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