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Case Report: We describe the catastrophic hospital course in a 58-year-old male who underwent a relatively minor surgical procedure which was complicated by deep venous thrombosis, pulmonary embolism and a paradoxical cerebral embolus. He suffered numerous complications related to placement of a vena caval filter, including complete inferior vena caval thrombosis with hemodynamic collapse.

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CASE REPORT

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Keywords: Surgical complication, Paradoxical cerebral embolus, Vena caval filter, Vena caval obstruction, Autopsy

How to cite this article

Liggett A, Marik PE. Fatal acute complete inferior vena cava thrombosis after inferior vena cava filter placement: Role of autopsy in elucidating the diagnosis. Int J Case Rep Images 2016;7(1):43–47.

doi:10.5348/ijcri-201609-CR-10596

INTRODUCTION

Clinical medicine is not a perfect science and despite our best intentions patients may develop iatrogenic complications. When complications occur, we are swift to intervene and prevent further injury. However, despite our best efforts patients' may die as a result of these complications. It is important to learn from these unfortunate events and an autopsy is invaluable in revealing the underlying cause of fatal pathophysiology. Many unexpected complications can go undiscovered without post-mortem investigation [1, 2]. This case describes the discovery of an extreme complication of inferior vena caval (IVC) filter placement which lead to the death of the patient [3].

CASE REPORT

A previously healthy 58-year-old Caucasian male was transferred to our hospital for management of a large right middle cerebral artery (MCA) ischemic stroke following a complex hospital course. The patient had a history of benign prostatic hyperplasia (BPH), gastroesophageal reflux disease (GERD) and seasonal allergic rhinitis. The patient was initially treated at an outside hospital for BPH with an elective transurethral resection of the prostate (TURP), which was complicated by significant postoperative hematuria. On postoperative day-1, (POD)

Amanda Liggett¹, Paul E. Marik²

Affiliations: ¹MD, PGY-3 Resident, Combined Family/Internal Medicine Residency Program; ²MD, FCCM, FCCP, Professor and Chief, Division of Pulmonary and Critical Care Medicine, Eastern Virginia Medical School. Norfolk, VA, USA.

Corresponding Author: Paul Marik, MD, Eastern Virginia Medical School, 825 Fairfax Av, Suite 410, Norfolk VA 23507; Email: marikpe@evms.edu

Received: 06 November 2015

Accepted: 15 December 2015

Published: 01 January 2016

the patient developed cramping right calf pain and mild shortness of breath. A duplex ultrasound of the lower extremities (PVL) and computed tomography (CT) angiography of the chest revealed a proximal right lower extremity deep venous thrombosis (DVT) and bilateral submassive pulmonary emboli (PE), respectively. For fear of worsening the patient's hematuria by treating the DVT/PE with anticoagulation, the urology team consulted interventional radiology (IR) for placement of an IVC filter to prevent further venous thromboemboli (VTE). The patient underwent the procedure without any apparent complications. On POD-3, the patient developed acute onset dyspnea with associated tachycardia and hypoxemia, concerning for recurrent VTE. A bedside transthoracic echocardiogram (TTE) demonstrated right ventricular dysfunction and repeat troponins were now elevated. The patient was treated with a single dose of therapeutic low-molecular-weight-heparin (LMWH), with the intention of starting rivaroxaban that evening. However, the patient developed worsening hematuria, and per urology's recommendations, further anticoagulation was held for approximately 12 hours. A heparin infusion was started on POD-4, favored for its reversibility. The patient was tolerating therapeutic anticoagulation until POD-6, when he became minimally responsive with acute left hemiparesis and hypoxemia. A CT scan of the head revealed hyperdensity in the distribution of the right MCA consistent with a large acute ischemic stroke. The heparin infusion was discontinued to avoid hemorrhagic conversion of the ischemic stroke. The patient was emergently intubated for transfer to our hospital, where en route, he suffered a right pneumothorax during attempted placement of a subclavian central venous catheter.

When the patient arrived in our emergency department (ED), a chest tube was emergently placed and our critical care team was called for admission to the ICU.

Upon admission to ICU, a TTE demonstrated a positive bubble study indicative of an atrial shunt which supported the presumptive diagnosis of a "paradoxical cerebral embolus." CT angiography demonstrated progressive sub-massive VTE while abdominal imaging demonstrated a malpositioned infrarenal IVC filter, tilted in a manner that would allow for the passage of emboli up to 9 mm in diameter, which exceeded the size of many of the PE present on the CT angiogram. A preliminary hypercoagulable panel was negative for both the Factor V Leiden mutation and antiphospholipid syndrome. Due to malignant MCA territory edema with mass effect, a frontotemporal parietal craniectomy and duraplasty was performed on day-2 of admission to our hospital. Immediately following the craniectomy our vascular surgeons unsuccessfully attempted to remove the malpositioned infrarenal IVC filter, prompting placement of a suprarenal filter with the intention of future removal of the malpositioned filter. On hospital day (HD) 10, the patient underwent removal of both the malpositioned

infrarenal filter and suprarenal filter, followed by placement of another infrarenal filter. The procedure was, however, complicated by improper deployment of the new filter with migration to the suprarenal position. This defective filter was removed followed by the successful deployment of a correctly orientated infrarenal filter. Despite these multiple complications the patient was successfully extubated and transferred out of the ICU on HD 12.

On the day of transfer the patient was started on therapeutic LMWH as a bridge to warfarin; our neurosurgical team deemed it "safe" to start anticoagulation 7 days' status post craniectomy. The patient did well for the next two days and was being evaluated for admission to an inpatient rehabilitation facility. Then on HD 15, the patient developed sudden hypotension, tachycardia and respiratory difficulty; he was emergently transferred back to the ICU and intubated. Vasopressors were started via a peripheral intravenous catheter. While attempts were being made to insert a central venous catheter for further fluid resuscitation and vasopressor support, the patient became pulseless with asystole visible on the bedside monitor. Advanced cardiovascular life support was immediately initiated and performed for approximately 25 minutes before the team leader suspended further cardiopulmonary resuscitation and pronounced the patient dead.

Reflections on the patient's tragic outcome and the autopsy findings

The differential diagnosis for the patient's immediate cause of death included intracranial hemorrhage, massive contained bleeding, myocardial infarction or recurrent massive pulmonary embolism. Cerebral hemorrhage was thought to be most likely, with the patient being 16 days out from a massive ischemic stroke and on two forms of therapeutic anticoagulation (LMWH and warfarin), although his INR on the morning of his death was 1.4 (reference range 0.89–1.29) and his partial thromboplastin time was 14.8 seconds (reference range 9.0–13.0). Recurrent massive PE was another possibility, however unlikely, with the patient being on two forms of anticoagulation and the presence of a "well positioned" IVC filter.

This patient overcame complications from almost every intervention performed on him, and unfortunately, succumbed in a final twist of fate while nearing his anticipated discharge. Part of our job as physicians is to counsel patients on the risks of procedures and make decisions to navigate our patients through both expected and unexpected complications. We were devastated by the death of this previously healthy man who died from a series of unfortunate complications following relatively minor elective surgery. With the death of this patient, we asked ourselves, "did we start anticoagulation too early after a large ischemic stroke and neurosurgical procedure?", "were we not aggressive enough with starting

anticoagulation sooner?”, “did we miss the diagnosis of a coagulopathic disorder?” Our curiosity was assuaged with the post-mortem examination.

It is infrequent in modern medicine that clinicians order autopsies for their deceased patients, much less be witness firsthand to the gross anatomy of fatal pathophysiology. Patients die from cardiorespiratory failure, and because of old age and multiple co-morbid conditions, we do not always advocate for a post-mortem investigation. In this relatively young, previously healthy patient, about to be discharged to a rehabilitation facility after surviving multiple complications and setbacks, the critical care team and the patient’s family believed it was important to determine the immediate cause of death. Our pathologist kindly invited us to join him in the autopsy-room for the examination, where dissection revealed an acute complete obstruction of the IVC by entangled emboli at the level of the infrarenal IVC filter (Figure 1) with fresh thrombi propagating into the pelvic and iliac veins. Autopsy further revealed a probe-patent foramen ovale (Figure 2), distensible to about 8–9 mm, and a very large right hemispheric cerebral infarction (Figure 3) involving the mid-frontal to parietal to mid-occipital lobe, effecting cortical surfaces extending to the lateral ventricular surfaces with gross dimensions of 14x10x6.5 cm, with only minimal interstitial hemorrhage found within the cerebral infarct. Multiple small PE’s were present peripherally in the lungs with microscopic evidence dating them to 10–14 days ante-mortem. The immediate cause of death was determined to be “an occluded inferior vena cava by extensive thrombosis propagating distally from the site of the IVC filter with circulatory collapse”.

Figure 2: Dissection of the heart demonstrating a patent foramen ovale.

Figure 3: Section of the brain demonstrating a very large right side hemispheric cerebral infarction.

Figure 1: Thrombosed inferior vena caval filter with entangled emboli.

DISCUSSION

The autopsy is invaluable in uncovering the true, and sometimes unexpected, cause of death. It

helps to solidify our recognition of the more elusive disease processes and uncommon complications of treatment, and importantly gives families closure. The complications of IVC filter placement are many and IVC filters are not benign devices. Our patient suffered almost all of the recognized complications of IVC filter placement, including, malposition with recurrent thromboembolic complications, defective deployment and filter thrombosis [4–8]. Occlusion of the IVC is the most frequent complication following IVC filter placement, with IVC thrombosis reported in 2.7% of patients owing to new local thrombus formation/thrombogenicity of the device, trapped embolus from a more distant site, or cephalad extension of distal DVT [4–8]. IVC filter thrombosis with acute complete IVC obstruction and circulatory collapse is an extremely rare complication [4–6]. More commonly, IVC thrombosis is a chronic, insidious process, resulting in the formation of collateral circulation via the abdominal wall veins. This may present asymptotically as an incidental finding discovered on unrelated imaging, or with leg edema, stasis ulcers, and back pain [4–8]. Most symptomatic obstructions are treated with anticoagulation, catheter-directed thrombolytic therapy or thrombectomy and are rarely acutely fatal [3–8]. In our patient, who was receiving two forms of therapeutic anticoagulation, his lethal occlusive IVC filter thrombosis was a shocking and somewhat mysterious complication.

CONCLUSION

We present the case history of a previously healthy man who suffered a series of unfortunate complications following relatively minor elective surgery and who ultimately died from circulatory collapse due to a thrombosed inferior vena caval (IVC) filter.

Author Contributions

Amanda Liggett – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Paul E. Marik – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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