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Postimplantation Syndrome after Traumatic Internal Carotid Artery Pseudoaneurysm Repair with Stent

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Abstract

Endovascular repair of traumatic internal carotid artery pseudoaneurysm (TICAP) with covered stents represents a safe treatment with few complications. However, the presence of foreign material used to treat TICAP and blood clots in the excluded pseudoaneurysms can trigger an acute systemic inflammatory response syndrome called postimplantation syndrome (PIS). To the best of our knowledge, PIS is described only after abdominal aortic endovascular aneurysm repair. Here, we report the case of PIS in a young, healthy, polytraumatized female patient with TICAP treated with endovascular covered stent.

Keywords

- traumatic internal carotid artery pseudoaneurysm
- postimplantation syndrome
- nitinol

Introduction

Traumatic internal carotid artery pseudoaneurysm (TICAP) is the most insidious and lethal complication of blunt or penetrating trauma of the neck, causing acute cerebral ischemia in young patients, with a high percentage of permanent neurologic deficits.[1–3]

Endovascular repair with covered stents represents a safe treatment for TICAP, with few complications, a high success rate, and low periprocedural morbidity and mortality rates.[4]

The presence of foreign material used to treat TICAP and clotted blood in the excluded pseudoaneurysms can trigger an acute systemic inflammatory response syndrome (SIRS) called postimplantation syndrome (PIS). PIS is defined according to SIRS criteria (fever $>$ 38°C, white blood cell [WBC] $>$ 12,000/mL, and increase in C-reactive protein [CRP] without infection evidence). PIS is a known entity after aortic endovascular aneurysm repair (EVAR); however, to our knowledge, there is no literature regarding its occurrence in patients undergoing endovascular stent placement for TICAP.[5,6]

This case report describes the development of PIS in a young female patient after endovascular treatment of a TICAP.

Case Report

A healthy polytraumatized 28-year-old female (weight: 64 kg) was admitted to the intensive care unit (ICU) for the management of brain and lung contusions, jaw fractures, right kidney rupture, left femoral head dislocation, and gluteal hematoma. Two days postadmission, a tracheostomy was performed in anticipation of jaw fracture surgery. On the 8th day, an angio-computed tomography (CT) scan of the neck vessels revealed acute dissection of the right internal carotid artery, treated with clopidogrel and aspirin. Subsequently, a neck vessel angiography performed on the 16th day showed the presence of left TICAP (►**Fig. 1**), with rapid enlargement (16 mm in diameter) and longitudinal extension in further evaluations. Due to the high risk of bleeding and stroke, she was scheduled for stent-graft positioning. On the day of the procedure (23rd day), the patient was afebrile, breathing spontaneously, had no neurological deficits, and was on dexmedetomidine infusion for sedation (0.6 µg/kg/h). Laboratory tests indicated normal renal function (estimated glomerular filtration rate: 136.0 mL/min), liver enzymes (aspartate aminotransferase: 41 UI/mL, alanine aminotransferase: 71 UI/mL, bilirubin: 0.86 mg/dL), and cardiac marker

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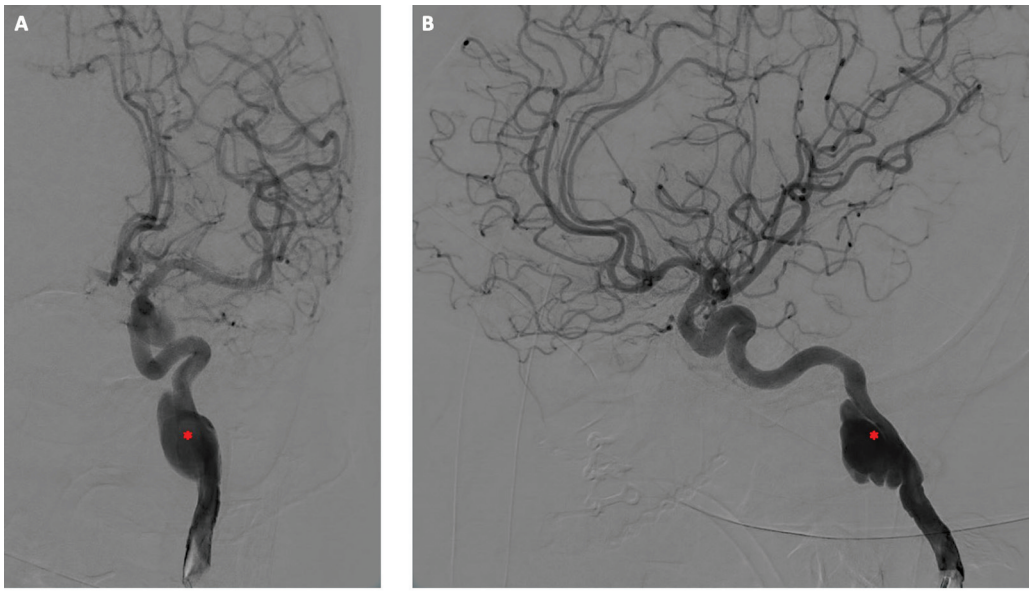

Fig. 1 The anteroposterior (**A**) and lateral (**B**) views of neck vessel angiography. Note the presence of a large left traumatic internal carotid artery pseudoaneurysm (red asterisk).

(troponin: I 2.0 ng/L), alongside normal WBC (8.38×10^3/µL), hemoglobin (11.8 g/dL), platelets (294×10^3/µL), fibrinogen (222 mg/dL), international normalized ratio (1.07), Prothrombin time (92%), activated partial thromboplastin time (21.6 seconds), CRP (4.30 mg/dL), and procalcitonin (PCT, 0.05 ng/mL), but a notable increase in D-dimer levels (3029 ng/mL).

We induced general anesthesia with propofol (1.5 mg/kg), fentanyl (1 µg/kg), and rocuronium (0.6 mg/kg). Subsequently, the patient was connected to a mechanical ventilator by the tracheostomy tube. For maintenance, total intravenous anesthesia was administered (propofol, 4–8 mg/kg/h, plus remifentanil, 0.05–0.10 µg/kg/min, plus dexmedetomidine, 0.4–0.6 µg/kg/h), adjusted based on bispectral index values (40–60).

Through an 8 French introducer in the right femoral artery, a braided nitinol stent (CASPER 7/30 mm) was successfully placed, excluding the pseudoaneurysm from the blood flow.

The procedure was completed in an hour without any complications. Postprocedure, the patient was breathing spontaneously and had stable vital parameters (heart rate: 82 bpm, blood pressure: 128/72 mm Hg, oxygen saturation (SpO_2) 100% with fraction of inspired oxygen 50%). Propofol and remifentanil infusions were discontinued; in addition to the ongoing dexmedetomidine infusion (0.4 µg/kg/h), intravenous midazolam (10 mg) and paracetamol (1000 mg) were given for sedation and pain management, respectively; the patient was transferred in ICU for further management.

Twenty minutes after arriving in the ICU, the patient developed high-rate supraventricular tachycardia (heart rate exceeding 170 bpm) and fever (38.8 °C), with normal blood pressure (115/74 mm Hg). Considering the risk of stroke, she was sedated with propofol and placed on controlled mechanical ventilation. Esmolol infusion was initiated (100 µg/kg/min) to lower her heart rate, and a single dose of methylprednisolone (40 mg) was administered to reduce fever. Her condition improved within hours, showing reduced heart rate (86 bpm) and temperature (36.8 °C), with no hypotension.

The next day, a brain CT revealed no new lesions and decreased TICAP size. Her body temperature remained normal, and after stopping sedation, neurologic evaluation showed no alteration. Laboratory tests indicated an increase in WBC (20.64×10^3/µL, neutrophils 89.8%), CRP (9.30 mg/dL), and PCT (61.71 ng/mL), negative cardiac marker injury (troponin I 9.0 ng/dL) and stable values in D-dimer (2997 ng/mL). During the next 48 hours, WBC (8.89×10^3/µL, neutrophil 73.1%), CRP (2.40 mg/dL), and PCT (4.72 ng/mL) decreased without fever or infection during the following days.

Discussion

The clinical features, laboratory investigations, and the relatively transient course of the illness are highly suggestive of the occurrence of a PIS in our patient. The diagnosis of this condition in a postsurgical ICU can be challenging, given the multiple etiologies for fever and tachycardia, such as sepsis, pulmonary embolism (PE), and anaphylactic reactions (AR).

In our patient, sepsis or ongoing infections were excluded due to low PCT levels and normal WBC despite the high preoperative level of CRP. The postprocedural increase in these markers and a decrease during the next 48 hours were interpreted as the consequence of transient inflammatory condition.[7]

PE, due to deep venous thrombosis (DVT), with or without hypotension, is a common and severe complication in ICU patients. Diagnosing in the ICU remains challenging due to elevated D-dimer levels associated with critical illness. Consequently, D-dimer levels are less specific in the ICU setting.[8] In our case, patients received clopidogrel and aspirin, drugs that offer protection against DVT. The stable values in postprocedural D-dimer and troponin and no changes in SpO_2 and arterial blood gas analysis allowed clinicians to exclude PE.

It was hypothesized that PIS might be mistaken for an AR to nitinol or other metals.[9] However, unlike AR, which typically develops minutes after exposure to an allergen, the patient's clinical condition remained stable poststenting. Tachycardia and fever only appeared several minutes after being transferred to the ICU. Additionally, the patient's medical history did not indicate allergies, and there was no hypotension, a key indicator of AR.

PIS can be considered within the spectrum of SIRS, sharing similar pathophysiological mechanisms. Understanding this relationship is crucial for accurate diagnosis and effective management.

PIS is a systemic inflammatory response that may occur shortly after EVAR,[5,6] with an incidence varying from 14 to 60%.

The PIS etiology is largely unknown, but it was hypothesized that the massive release of immune mediators (tumor necrosis factor α, interleukin-6, and other cytokines) due to the new-onset thrombus after the mechanical exclusion by the arterial blood flow could play a role.[10] Furthermore, the biomaterial composition of stent grafts to treat abdomen aortic aneurysms may promote WBC and platelets activation with a massive release of immune mediators.[9,10] Studies demonstrated that stents based on woven polyester were independently associated with a stronger inflammatory response.[11,12]

The literature has no report about PIS following TICAP treated with an endovascular nitinol-covered stent. Nitinol is a nickel-titanium alloy, and it has been widely used in coronary and peripheral arterial "bare-metal" stenting. Nitinol stents are also used in cerebral vessel endografts but not in EVAR. The chemical production of nitinol prevents breakdown, and special coating reduces nickel and titanium exposure. While pure titanium is biocompatible, pure nickel in high concentrations is known to cause systemic toxicity, cellular damage, and immune response in patients with nickel sensitivity.[13]

In conclusion, PIS occurrence is rare or probably under-reported. In patients undergoing endovascular treatment with nitinol stents, the sudden onset of fever and tachycardia should alert the clinicians of the possible onset of PIS. PIS is a transient condition, but data about outcomes in ICU patients are lacking. Furthermore, clinicians should rule out other more serious clinical conditions, such as sepsis, PE, and AR.

Conflict of Interest
None declared.

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