

## CASE REPORT

# Severe Aortic Thrombosis and Profound Hypothermia: A Case Report

Johan Schmitt<sup>1</sup>, Pierre Esnault<sup>2</sup>, Milena Sartre<sup>3</sup>, Pierre J Cungi<sup>4</sup>, Eric Meaudre<sup>5</sup>

### ABSTRACT

**Background:** Blood clot formation is a multifactorial process and has been related many times in intensive care units. Here is presented a multiple thrombosis formation in a rewarming patient.

**Case description:** A 68-year-old patient was admitted to our intensive care unit after lying on the floor for an unknown time. She presented a severe hypothermia at 26° and a severe cardiogenic shock. Because she was confused and was hypoxemic, she had been intubated at her admission. After intravascular warming, we could stop sedative medications. She presented a right hemiparesis and acute left leg ischemia. Computed tomography (CT) scan revealed a constituted left Sylvian stroke and a massive clot along the aorta. She required a surgical embolectomy and fasciotomy. She died after she presented a severe bowel ischemia on the third day after her admission.

**Conclusion:** Relevant hypothesis for blood clot formation in this patient may include prolonged lying position or blood temperature variation. Hypothermia and rewarming responsibilities may explain multiple thrombosis development.

**Keywords:** Acute limb ischemia, Hypothermia, Rewarming, Stroke, Thrombosis.

*Indian Journal of Critical Care Medicine* (2021): 10.5005/jp-journals-10071-23820

### INTRODUCTION

Ischemic complications in intensive care unit patients are frequent. Many factors have been described.<sup>1,2</sup> Both decreased cardiac output and microcirculation alterations may be responsible for arterial thrombosis.<sup>3</sup> The role of variation in body temperature in coagulopathy pathways is not fully explained, especially during rewarming. Here, we describe a case of acute limb ischemia that occurred during rewarming of a hypothermic patient.

### CASE DESCRIPTION

A 68-year-old woman with no medical history was discovered at home, lying on the floor for an unknown number of hours and her core temperature was 26°. No medication was found lying around. On admission, she presented with a severe cardiogenic shock. The mean blood pressure was 30 mm Hg with a sinus bradycardia of 31 per minute and the arterial lactate was 2.5 mmol/L. Cardiac ultrasonography showed a global hypokinesia of the left ventricle. She had acute respiratory distress, with oxygen saturation of 93% requiring high-flow nasal oxygen. Laboratory investigations revealed an increased sodium level (157 mmol/L), a KDIGO (kidney disease improving global outcomes), three acute kidney injury (uremia was at 342 mg%, creatinine 3.95 mg/dL), and a mild rhabdomyolysis (CPK at 280 U/L, myoglobin at 3880 ug/L). The initial PT was 51%, fibrinogen rate was 6.7 g/L, and blood platelets were 248 G/L. A DIC (disseminated intravascular coagulopathy) was present with raised D-dimers (4.93 ug/mL) and fibrin monomers at 24.6 ug/mL. The International Society on Thrombosis and Hemostasis (ISTH) score was 4. Initial care consisted in tracheal intubation, mechanical ventilation, external and internal rewarming (COOLGUARD 3000™ device), and continuous venovenous hemofiltration. Blood pressure quickly stabilized after initiation of norepinephrine perfusion. Complete warming occurred within 8 hours and sedation could be stopped.

<sup>1-5</sup>Department of Intensive Care Unit, HIA Sainte Anne, Toulon, Provence-Alpes-Cote d'Azur, France

**Corresponding Author:** Johan Schmitt, Department of Intensive Care Unit, HIA Sainte Anne, Toulon, Provence-Alpes-Cote d'Azur, France, Phone: +33 483162212, e-mail: Schmitt.johan83@gmail.com

**How to cite this article:** Schmitt J, Esnault P, Sartre M, Cungi PJ, Meaudre E. Severe Aortic Thrombosis and Profound Hypothermia: A Case Report. *Indian J Crit Care Med* 2021;25(5):588–589.

**Source of support:** Nil

**Conflict of interest:** None

After awakening, the patient assessment revealed acute left leg ischemia and right hemiparesis. A whole-body CT scanner showed a constituted left Sylvian stroke, multiple tiered blood clots along the aorta, and in the left iliac artery (Fig. 1). A surgical embolectomy and preemptive fasciotomy were performed, and the patient was initiated on anticoagulation. On day 3, the abdominal examination revealed a generalized contracture with a CT scan showing a diffuse hepatic and bowel portal gas, with multiple arterial thromboses. End of life decision was taken in view of the possible poor outcome and the patient died after treatment withdrawal.

### DISCUSSION

This case underlines the consequences of hypothermia and subsequent rewarming. Some hypotheses may explain clot formation. The Virchow triad has been described mainly for venous thrombosis development.<sup>4</sup> The DIC hypothesis may have led to thrombotic complications. Mahajan et al. have described DIC occurring on patient warming in hypothermia.<sup>5</sup> Hypothermia may

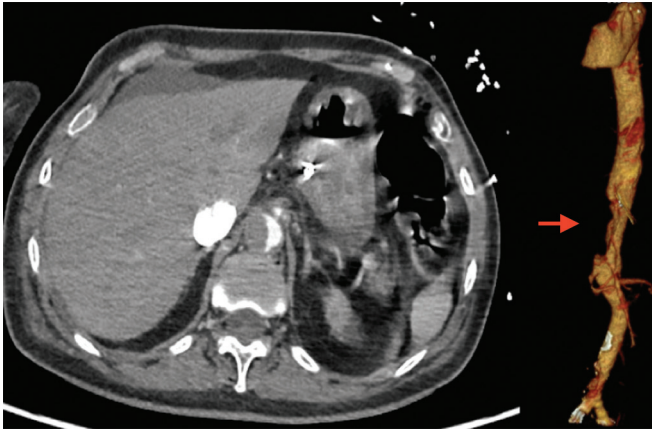


Fig. 1: CT scan view of aortic thrombosis

have caused glycocalyx alterations and a massive thromboplastin (factor III) liberation, triggering coagulation pathways.<sup>6</sup> Although we observed an increased PT and an increased partial thromboplastin time in this patient presenting with hypothermia, blood viscosity increases when cooling occurs and it increases once the patient is rewarmed.<sup>7</sup> In an experimental study, Sands et al. have found a better correlation between blood viscosity and hypothermia as against that with hematocrit. An increased hematocrit would be related to a systemic capillary leak, mostly reversible with dextran perfusion. Furthermore, initial bradycardia may have led to arterial thrombosis.<sup>8</sup> Systolic left ventricular dysfunction induced by hypothermia is related to a decreased sensitivity of the cardiac cell to calcium and a phosphorylation troponin defect.<sup>9</sup> Initial cardiogenic shock has been already described as a predictor of arterial thrombosis development in intensive care unit patients.<sup>3</sup> Sympathetic system activation modifies blood rheology and increases blood stasis.<sup>10</sup> Finally, hypothermia causes an endothelial prostacyclin synthesis inhibition, increasing platelet aggregation.<sup>11</sup> In our patient, the biological screening eliminated an acquired or constitutional thrombophilia. Prolonged immobility may have led to development of multiple arterial blood clots. Decubitus complications have been well described, but most of them concern the venous compartment.<sup>12</sup> Acute limb ischemia symptoms may be missed in patients presenting hemodynamic instability.

## CONCLUSION

This case report highlights the importance of the possible role of hypothermia in arterial clot formation, especially associated with a prolonged immobility.

## ORCID

Johan Schmitt <https://orcid.org/0000-0002-7172-7843>

Esnault Pierre <https://orcid.org/0000-0003-0789-9162>

Milena Sartre <https://orcid.org/0000-0003-1938-1598>

Pierre J Cungi <https://orcid.org/0000-0003-3928-1404>

Eric Meaudre <https://orcid.org/0000-0003-4345-9009>

## REFERENCES

1. Boddi M, Peris A. Deep vein thrombosis in intensive care. *Adv Exp Med Biol* 2017;906:167–181. DOI: 10.1007/5584\_2016\_114.
2. Leone M, Bechis C, Lefrant JY. Outcome of acute mesenteric ischemia in the intensive care unit: a retrospective, multicenter study of 780 cases. *Intensive Care Med* 2015;41(4):667–676. DOI: 10.1007/s00134-015-3690-8.
3. Warkentin TE. Microvascular thrombosis and ischaemic limb losses in critically ill patients. *Hamostaseologie* 2019;39(1):6–19. DOI: 10.1055/s-0038-1676823.
4. Kushner A, West WP, Pillarisett LS. *StatPearls*. Treasure Island: StatPearls Publishing; 2019.
5. Mahajan SL, Myers TJ, Baldini MG. Disseminated intravascular coagulation during rearming following hypothermia. *JAMA* 1981;245:2517–2518. DOI: 10.1001/jama.1981.03310490035022.
6. Spero JA, Lewis JH, Hasiba U. Disseminated intravascular coagulation: findings in 346 patients. *Thromb Haemost* 1980;43:28–33. PMID: 6773170.
7. Sands M, Mohri H, Shigekazu S, Mannik M, Hessel E, Dillard D. Hemorheology during deep hypothermia. *Cryobiology* 1979;16:229–239. DOI: 10.1016/0011-2240(79)90036-1.
8. Hynson JM, Sesslet DI, Moayeri A, McGuire J. Absence of nonshivering thermogenesis in anesthetized humans. *Anesthesiology* 1993;79:695–703. DOI: 10.1097/0000542-199310000-00010.
9. Han YS, Tveita T, Prakash YS, Sieck G. Mechanisms underlying hypothermia-induced cardiac contractile dysfunction. *Am J Physiol Heart Circ Physiol* 2009;298:890–897. DOI: 10.1152/ajpheart.00805.2009.
10. Mallet ML. Pathophysiology of accidental hypothermia. *Q J Med* 2002;95:775–785. DOI: 10.1093/qjmed/95.12.775.
11. Mikhailidis DP, Hutton RA, Jeremy JY, Dandona P. Hypothermia and pancreatitis. *J Clin Pathol* 1983;36:483–484. DOI: 10.1136/jcp.36.4.483.
12. Martin-Du Pan RC, Benoit R, Girardier L. The role of body position and gravity in the symptoms and treatment of various medical diseases. *Swiss Med Wkly* 2004;134(37-38):543–551. DOI: 10.4414/smw.2004.09765.