Blood Purification

Case Report

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Subarachnoid Hemorrhage and Extracorporeal Blood Purification with HA-380 and High-Volume Hemofiltration: A New Therapeutic Challenge at the Neurocritical Care Unit? A Case Report

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Keywords
Subarachnoid hemorrhage · Interleukin-6 · Hemadsorption

Abstract
We present the case of a patient with subarachnoid hemorrhage (SAH) secondary to a ruptured cerebral aneurysm and a refractory shock with high doses of vasopressors without a proven source of infection. This patient received therapy with high-volume hemofiltration plus adsorption, resolving the hemodynamic deterioration and with good neurological evolution. Our clinical case proposes that extracorporeal therapies may have a feasibility role in the management of complications of SAH.

Introduction

Neurocritical pathologies such as subarachnoid hemorrhage (SAH) have shown a correlation between levels of cytokines and their outcomes [1]. Early elevation of cytokines at the central and peripheral levels, mainly interleukin-6 (IL-6), with a first peak at 24–48 h after the event, along with other inflammatory events such as neutrophil activation, is associated with neurological deterioration and development of delayed cerebral ischemia [1–3]. Furthermore, severe systemic inflammatory states can lead to significant circulatory impairment with multiple organ failure and death [4]. Our group proposes that the use of extracorporeal therapies for immunomodulation could have a role in preventing complications in patients with SAH and significant elevation of cytokines, improving neurological outcomes.

Case Presentation

A 57-year-old man with aneurysmal SAH of the right posterior communicating artery and excluded with clipping was admitted to the critical care unit. Initial CT shows modified Fisher scale IV and World Federation of Neurological Surgeons grading scale IV. On postoperative day 4, fever, hypotension, and a rise of the inflammatory parameters began (Table 1) with a negative microbiological study and normal chest radiography, highlighting an inflammatory cerebrospinal fluid (CSF), without isolated mi-

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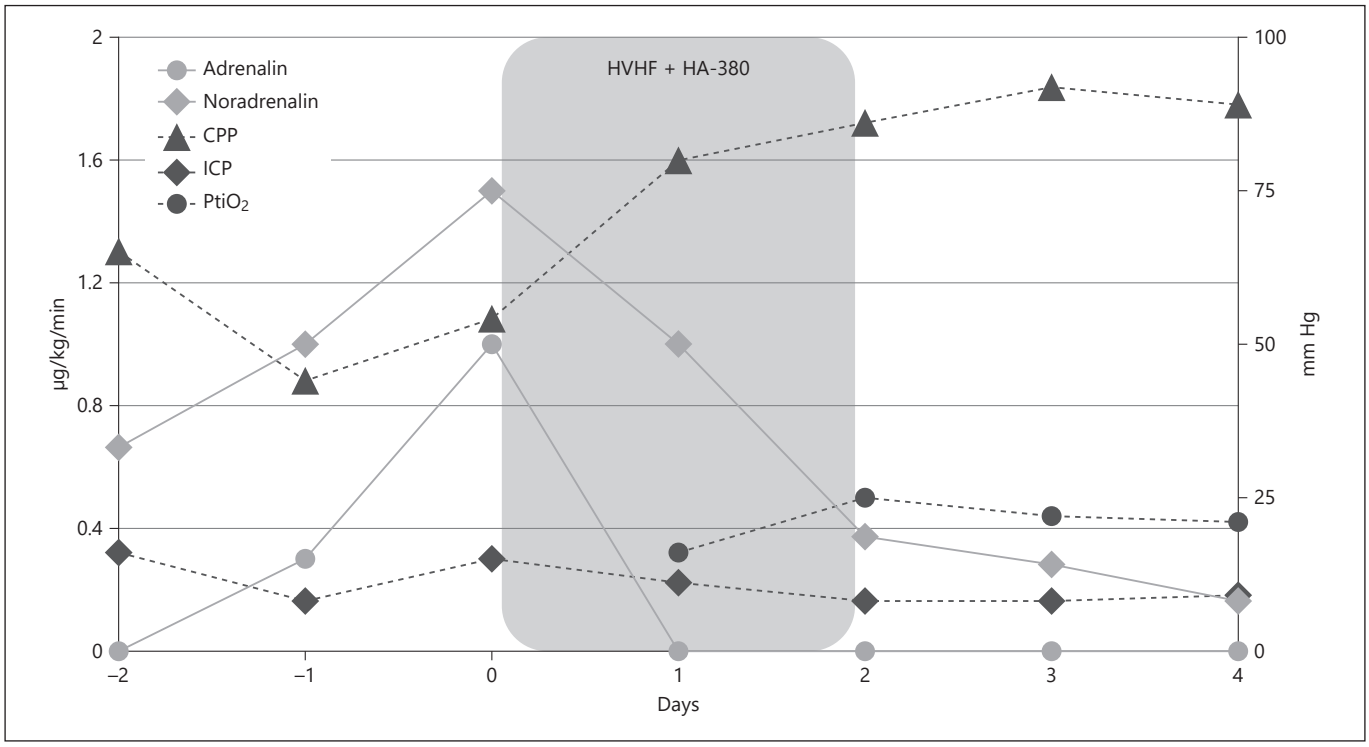

Fig. 1. Evolution of hemodynamics and neuromonitoring parameters during the HA-380 plus HVHF therapy. Hemodynamics parameters are presented with black symbols (right y-axis). Norepinephrine and epinephrine are presented with gray symbols (left y-axis). Epinephrine was stopped after the first day of HA-380 plus HVHF therapy, and norepinephrine was significantly decreased. CPP, cerebral perfusion pressure; ICP, intracranial pressure; $PtiO_2$, tissue oxygen pressure; HVHF, high-volume hemofiltration.

Table 1. Timeline values of the clinical case

Days	−3	−2	−1	0	1	2	3
Natremia, mmol/L	138	140	142	142	136	140	139
Lactate, mmol/L	2.02	1.82	1.88	3	1.23	1.49	0.77
Phosphate, U/L	2	2	nd	2.23	nd	2.55	1.64
SVRI, din-seg-m^2/cm^5	nd	nd	nd	799	1,058	1,558	1,897
Creatinine, mg/dL	0.67	0.67	0.66	1.42	0.66	0.66	0.66
C-reactive protein, mg/L	28	97	nd	268	215	153	68.6
Procalcitonin ng/mL	nd	nd	nd	2.98	nd	1.14	0.17
White blood cells $\times 10^3/\mu L$	23,500	nd	nd	19,000	15,700	13,100	13,000
Temperature, °C	36.2	37.1	37.2	35	36.1	36.8	36.6

CSF, cerebrospinal fluid; SVRI, systemic vascular resistance index; IL-6, interleukin-6; nd, no data.

croorganisms. Broad-spectrum antibiotics were started, but he evolved with greater hemodynamic deterioration (norepinephrine 1.5 µg/kg/min and adrenaline 1 µg/kg/min), requiring a guided resuscitation with advanced hemodynamic monitoring (PiCCO), not achieving adequate values (CI 4 L/min/m^2, SVRI 850 dyn s/cm^5/m^2, and MAP <70 mm Hg), despite optimizing predictors of volume response and therefore suboptimal neuromonitoring parameters, highlighting a tissue oxygen pressure of 16 mm Hg and abnormal peripheral perfusion with abnormal capillary refill time. A second microbiological study was negative. Due to hyperinflammatory phenotype, blood purification therapy was considered.

A 12-h session of hemoperfusion (HP) pulse was performed (HA-380 cartridge, Jafron Biomedical Co.) in combination with

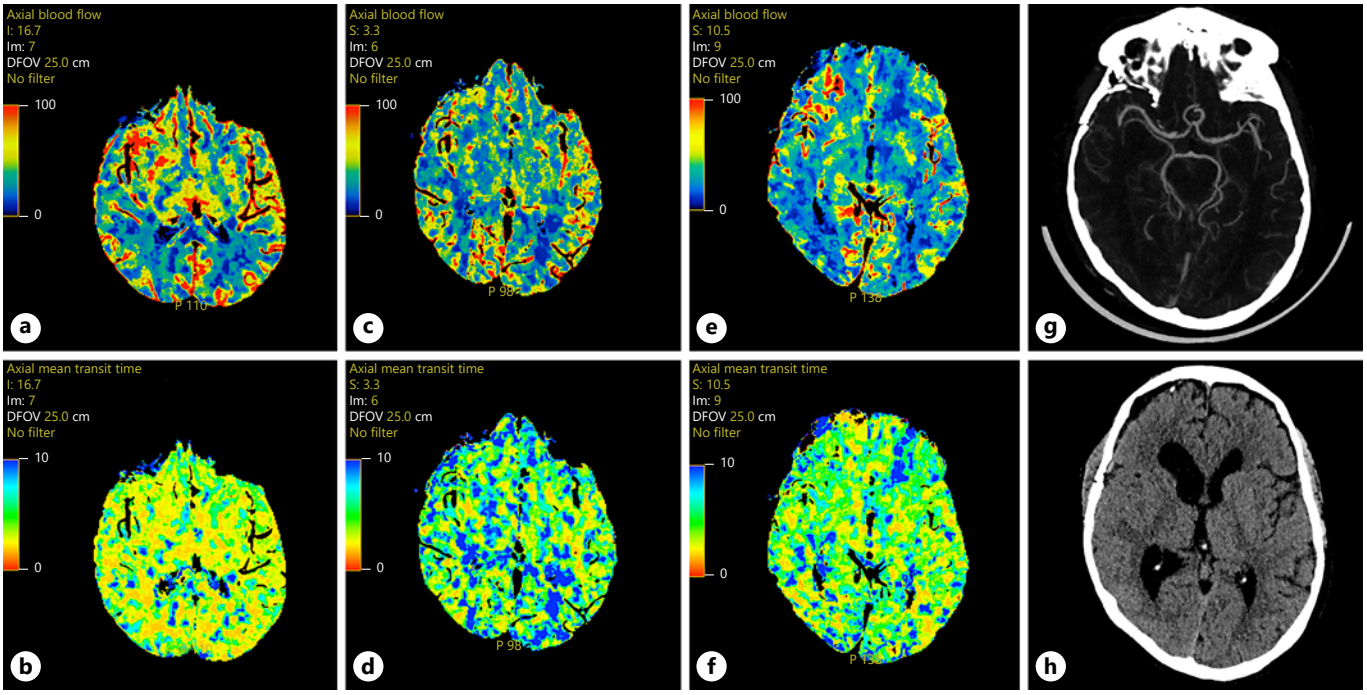

Fig. 2. Evolution of brain CT perfusion, CT angiography, and CT at discharge. **a**, **b** Blood flow map (CBF) and MTT demonstrate normal symmetric brain perfusion. **c**, **d** CBF and MMT demonstrate diffuse hypoperfusion, with mild decreased CBF at the beginning of HP + HVHF. **e**, **f** CBF and MMT show mild diffuse hypoperfusion, better than previous CT perfusion after HA-380 plus HVHF. **g** CT angiography without proximal intracranial arterial vasospasm. **h** Brain CT 1 month after SAH without new hypodense lesions in zones of previous hypoperfusion. SAH, subarachnoid hemorrhage; MTT, mean transit time; HVHF, high-volume hemofiltration; HP, hemoperfusion.

high-volume hemofiltration (HVHF) in a Prismaflex monitor (Baxter) with blood flow 250 mL/min and effluent dose 70 mL/kg/h, with prefilter replacement and heparin. The HP cartridge was installed post filter. According to local protocol, in the second and in the third day, only an 8-h isolated HVHF was performed, with same parameters. Replacement solutions were adjusted according to the protocol for target natremia [5].

A significant decrease in vasoactive drugs and improvement in inflammatory parameters were achieved (Fig. 1). Brain CT showed no vasospasm or delayed cerebral ischemia. On postoperative day 39, he was discharged from hospital without neurological sequelae.

Discussion and Conclusion

In SAH, neuroinflammation, evidenced by high levels of cytokines and a high lactate-pyruvate ratio, correlates with delayed cerebral ischemia [6]. Loss of the blood-brain barrier integrity and break down products of hemoglobin activate cellular immunity, leading to the production of pro-inflammatory cytokines and adhesion molecules at the CSF and plasma levels [7–9].

Blood purification therapies are proposed in systemic inflammatory syndrome with refractory shock [10]. We carried out HP using a macroporous resin sphere cartridge capable of adsorbing interleukins through size sieving and hydrophobic interactions, and it was combined with HVHF to enhance its effect [11–13]. Studies have shown that neutralization of peripheral IL-1 is associated with better neurological outcomes [8].

Consequently, we proposed hemadsorption plus HVHF as rescue therapy for SAH with refractory shock, achieving reduction of vasoactive drugs, plasma IL-6 from 34.4 to 4.8 pg/mL, and CSF lactate levels, maintaining adequate cerebral perfusion parameters, without evidence of delayed cerebral ischemia or cerebral infarction (Fig. 2).

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Statement of Ethics

Biochemical and clinical parameters were collected under the approval of the scientific Ethics Committee of the health service of Valparaiso – San Antonio. Written informed consent for publication was authorized by the patient of this case report and for any accompanying images. The consent document was authorized and reviewed by the local Ethics Committee.

Conflict of Interest Statement

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this article.

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Authors Contributions

G.R.G., R.O.A., and F.V.C. designed the work; G.R.G., R.O.A., and F.V.C. collected and analyzed the data; G.R.G., R.O.A., R.B.H., V.T.C., F.V.C., F.T.M., S.R.D., C.G.T., P.F.F., and O.G.C. drafted the work or substantively revised it; and all authors read and approved the final manuscript.

Availability of Data and Materials

All data generated or analyzed during this study are included in this published article.

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