

# Anesthetic Management of Patient with Varicose Veins and Undiagnosed Brain Tumor

## Varis ve Tanı Konulmamış Beyin Tümörü Olan Hastanın Anestezi Yönetimi

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**ABSTRACT** Intraoperative hemodynamic perturbations are common due to the effects of anesthetic medications and techniques, surgical manipulations, and the patient's medical comorbidities. But Intraoperative intractable hypotension is very rare and serious in young healthy patients. The case of a 36 year old American Society of Anesthesiologists (ASA) risk class I patient who developed intractable hypotension during varicose vein operation was presented and discussed in association with the relevant literature. The adverse effects of transient foam embolism and / or due to the contribution of anesthesia to autonomic disorders secondary to the brain tumor may result in resistant hypotension in the patient. The magnitude of preoperative evaluation in anesthesia practice is indisputable, fine details during anamnesis should be taken into consideration.

**ÖZET** Anestezik ilaçların ve tekniklerin, cerrahi manipülasyonların etkileri ve hastanın tıbbi komorbiditeleri nedeniyle intraoperatif hemodinamik değişiklikler yaygındır. Ancak genç sağlıklı hastalarda intraoperatif inatçı hipotansiyon çok nadir ve ciddidir. Varis ameliyatı sırasında inatçı hipotansiyon gelişen 36 yaşında ASA risk skoru I olgu, literatür eşliğinde sunuldu ve tartışıldı. Geçici foam embolisinin ve / veya anestezinin beyin tümörüne sekonder otonomik bozukluklara katkısı nedeniyle hastada dirençli hipotansiyona neden olabilir. Anestezi uygulamasında preoperatif değerlendirmenin önemli tartışılmazdır, anamnez sırasında ince ayrıntılar dikkate alınmalıdır.

**Keywords:** Foam embolism; general anaesthesia; glial tumor; hypotension

**Anahtar Kelimeler:** Köpük embolisi; genel anestezi; glial tümör; hipotansiyon

Intraoperative hypotension must be treated according to its cause, so it is critical to determine the pathophysiological process conducive to perioperative hypotension, including effect of anesthetic agents, hypovolemia, position or surgical technique, cardiac etiology, effect of mechanical ventilation, or anaphylaxis.<sup>1</sup> Rarely in young healthy individuals, surgical technique and patient factors may cause hypotension together. In this case, diagnosis and treatment may be difficult.

This report describes an intractable hypotension and visual blurring after foam injection sclerotherapy

for the treatment of varicose veins in a patient with undiagnosed brain tumor.

### CASE REPORT

The data of the patient were shared with her consent. A 36-year-old female patient presented at the Department of Cardiovascular Surgery due to swelling of the legs and vascularization for several months. After doppler ultrasonographic examination, there was no identified venous insufficiency in the patient who suffered from varicose veins in the legs. In the preoperative anesthesia visit, the patient was

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Peer review under responsibility of Türkiye Klinikleri Journal of Anesthesiology Reanimation

**Received:** 10 Apr 2020

**Received in revised form:** 02 Jul 2020

**Accepted:** 03 Jul 2020

**Available online:** 27 Oct 2020

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evaluated as ASA I after physical examinations and tests, and was found to be suitable for a low risk operation under general anesthesia.

In the operating room, 2 mg midazolam was administered as premedication and standard monitoring was performed for blood pressure: 110/80 mmHg, heart rate: 80/min, SpO<sub>2</sub>: 98%. In the induction of anesthesia, 150 mg propofol, 100 mcg fentanyl and 40 mg of rocuronium was given and the patient was intubated with a 7.0 spiral tube. Subsequently, *anesthesia was maintained* with 1.5% sevoflurane in air and oxygen and 0.1-0.2 µg/kg/min remifentanyl. The patient was then placed in the prone position.

During a 90 minute operation, stable hemodynamic measurements were monitored and there wasn't any significant change in the value of the end-tidal CO<sub>2</sub>. There was not any bleeding or allergic reaction during the operation. A sudden arrhythmia and a decrease in blood pressure (BP: 70/40 mmHg) and saturation (SpO<sub>2</sub>: 80%) were observed when the patient was taken from the prone position to the supine position. After extubating, arrhythmia, bradycardia, hypotension and desaturation continued.

After waiting for 10 minutes in the operating room, noradrenaline 0.08 µg/kg/min was administered to the patient who was conscious, oriented, cooperative but had blood pressure of 77/40 mmHg with no change in heart rate.

Since peripheral oxygen saturation did not increase (SpO<sub>2</sub>: 80%), administration of 3L/min nasal oxygen was continued and inhaler bronchodilator (ipratropium bromide salbutamol) and intravenous 2 mg/kg methylprednisolone were added to the treatment.

After the treatment, because the arterial blood gas parameters were pH: 7.29, PaO<sub>2</sub>: 47 mmHg, PaCO<sub>2</sub>: 33 mmHg, HCO<sub>3</sub><sup>-</sup>: 15.9 mmol/L, BE:-10.5 mmol/L, Lactate: 6.46 mmol/L, the patient was transferred to the intensive care unit (ICU).

In ICU, her heart rate was 55 beats/min, her blood pressure was 80/45 mmHg and respiratory rate was 18/min. Oxygen saturation was %85 of 3

l/min with nasal oxygen. Her respiratory sounds were clear bilaterally and no systolic murmur was noted over the tricuspid area. The 12 lead electrocardiogram showed sinus rhythm. Chest radiography performed at bedside was normal. Laboratory findings were normal except the D-dimer level of 3918 ng/ml. We thought that elevated D-dimer level may be associated with pulmonary embolism or deep vein thrombosis. 0.6 ml low molecular weight heparin (LMWH) was applied subcutaneous. There was no calf tenderness and Homan's sign was negative bilaterally. The echocardiography performed within the first hour of the patient's admission in the ICU was normal. Lower extremity Doppler ultrasound was normal. A computerized tomography (CT) of the chest that was immediately arranged was also normal.

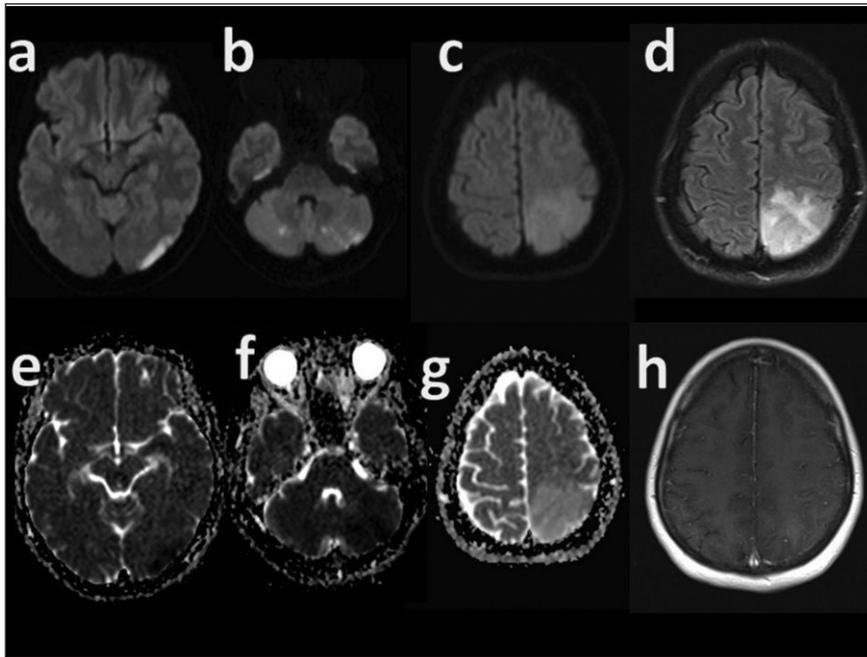
With the support of inotropic and intravenous hydration, blood pressure became 90/40 mmHg despite a constant heart rate of 62 bpm after 6 hours. Noradrenaline was decreased for entire removal. After 18 hours, the patient who was hemodynamically stable was transferred to the cardiovascular ward.

At the follow-up visit, a consultation was requested from the ophthalmology department when the patient stated that her existing visual blurring on the right side had increased after the surgery.

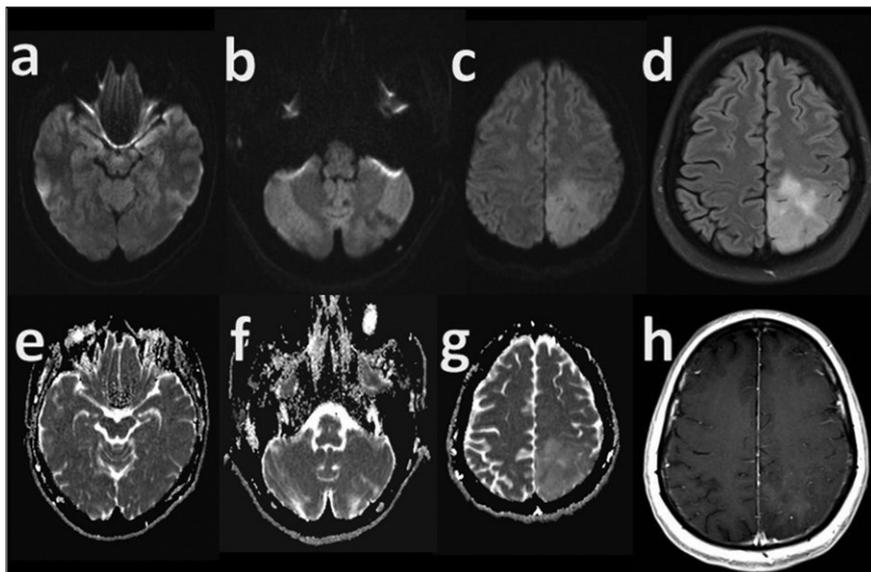
A consultation was requested from the neurology department due to homonymous hemianopsia. Cranial Magnetic Resonance Imaging (MRI), diffusion MRI, brain MR angio, bilateral carotid and vertebral artery Doppler ultrasonography (USG) imagings were performed. Contrast enhancement of cranial MR and diffusion-weighted MR showed cerebellar and occipital diffusion restriction mainly on the bilateral left and a hyperintense area (mass?) accompanied by edema in the parietooccipital region (Figure 1).

The patient, who had no hemodynamic problems and was almost free from the visual symptoms, was discharged postoperatively on the 5<sup>th</sup> day.

Contrast-enhanced brain MRI and diffusion-weighted MR were performed again after 1 month, and revealed high grade glial tumor and bilateral



**FIGURE 1:** DWIs of the case show, in both cerebellums (b), restricted diffusions compatible with acute infarct in the left occipital lobe (a) and their responses (e, f) indicating loss of signal in the ADC sequence. Apart from these, however, there is a notable increase in diffusion (c, g) in the left parietal lobe. In the left parietal pole, there is an increased intensity of signals (d) in the FLAIR sequence with a mild mass effect. There was a mild contrast involvement on this site after IV CMA (h). The mass lesion in the left parietal could not be conclusively differentiated as a subacute infarction or a mass.



**FIGURE 2:** The follow-up MRIs after one month reveal that the restricted diffusions in both cerebellums (b) and in the left occipital lobe (a) in the previous imaging now display an increase in signals in the ADC sequence (e, f) affecting the chronic infarction site positively. While other ischemic changes vary in time, the increased intensity of signals in the FLAIR sequence with a mass effect in the left parietal lobe is comparable to the results of the previous imaging and show no significant changes. The mild contrast involvement on this site after IV CMA still exists.

cerebral and left occipital subacute infarction in the left parietal lobe (Figure 2).

After two months, the patient was operated due

to intracranial mass at an external neurosurgery center. Both radiotherapy and chemotherapy are used for the ongoing treatment.

## DISCUSSION

Severe intraoperative hypotension is an anaesthetic emergency. In the event of hypotension, a rapid assessment of the patient is required. This should take into consideration: risks associated with the anaesthetic technique, risks specific to the type of surgery or approach, known or possible allergies. Systolic performance of the heart is determined by 3 factors: preload, afterload, and contractility. Hypotension may be due to a low cardiac output, restrictive volume replasmane, low systemic vascular resistance or a combination of this factors.<sup>2</sup>

When we turned the patient from prone to supine position, we determined the cause of sudden and permanent hypotension, respiratory distress as foam embolization due to foam sclerotherapy. Foam injection sclerotherapy is a minimally invasive replacement treatment for varicose veins. Adverse events associated with foam sclerotherapy occurred in 0-5.7 percent of foam injection sclerotherapy treatment.<sup>3</sup> In the literature, 1356 cases related to foam injection sclerotherapy have been reported as major and minor complications.<sup>4</sup> Major complications are anaphylaxis, stroke, myocardial infarction and thromboembolism, and minor adverse events include transient visual disturbance, cutaneous necrosis, ulceration, minor vein thrombosis, thrombophlebitis, local neurological injury and skin pigmentation. We ruled out acute coronary syndrome, atrioventricular, aortic obstruction or paradoxically embolization (due to patent foramen ovale) by using echocardiography in ICU within the first hour. In our case, which was initially considered as foam embolism, although the D-dimer value was high, we could not confirm this prerequisite with chest CT angio imaging performed at the second hour due to hemodynamic instability. It is stated in the literature that foam embolization can disappear in 50 minutes. This may explain that we are unable to detect the foam embolism.<sup>5</sup> In addition, the brain tumor diagnosed after the operation made the effect of hypotension on the patient more serious. Brain tumors may develop orthostatic hypotension due to autonomic disorders.<sup>6-8</sup> Etiologic factors of autonomic disorders with brain involvemens are due to multiple system atrophy (MSA), Wernicke

Korsakoff syndrome, posterior fossa tumors, baroreflex failure, olivopontocerebellar atrophy and Lewy body dementia. Orthostatic hypotension may be caused by autonomic disorder caused by noradrenergic neurotransmission. The mechanism of orthostatic hypotension may disrupt autonomic blood pressure regulation in human subjects, through baroreflex failure and / or neurogenic orthostatic hypotension.<sup>7,8</sup> When we question our postoperative patients, we rarely took a history of postural hypotension.

Treatment in hypotension is vital to ensure adequate organ blood flow; particularly to the brain, heart, kidneys. Uncorrected ischaemic changes may ensue, with consequences including stroke, myocardial infarction, acute tubular necrosis. In our case, the stroke areas in the brain increased as a result of hypotension lasting sixty minutes.

In an apparently healthy case, intraoperative intractable hypotension can be a sign of intravenous foam injection and an undiagnosed intracranial mass. Detailed anamnesis, monitorization and careful anesthesia management are important for maintaining intraoperative hemodynamic stability.

### Acknowledgements

*A sincere thank you to Prof. Dr. Ayda Türköz for his proofreading of the manuscript. I would like to offer my special thanks to Abdullah Karaakın for help providing language.*

### Source of Finance

*During this study, no financial or spiritual support was received neither from any pharmaceutical company that has a direct connection with the research subject, nor from a company that provides or produces medical instruments and materials which may negatively affect the evaluation process of this study.*

### Conflict of Interest

*No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.*

### Authorship Contributions

**Idea/Concept:** Deniz Kara; **Design:** Ayda Türköz; **Control/Supervision:** Deniz Kara; **Data Collection and/or**

**Processing:** Emir Cantürk, Canan Güler; **Analysis and/or Interpretation:** Canan Güler, Ayda Türköz; **Literature Review:** Tural Garayev; **Writing the Article:** Deniz Kara; **Critical Review:** Ayda Türköz.

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