Revista da Sociedade Portuguesa de Anestesiologia
http://revistas.rcaap.pt/anestesiologia
https://dx.doi.org/10.25751/rspa.21021

CASO CLÍNICO

Near-infrared Spectroscopy Oriented Anesthesia: About a Clinical Case of Subclavian Steal Syndrome

Anestesia Orientada por Espectroscopia Próximo do Infravermelho: A Propósito de Um Caso Clínico de Síndrome de Roubo da Subclávia

Sara Alves[1*], Filipa Martins[1], Tiago Faria[1]

Afiliação
[1] Serviço de Anestesiologia do Hospital de Braga, Braga, Portugal.

Keywords
Anesthesia; Hypertension; Oximetry; Spectroscopy, Near-Infrared; Subclavian Steal Syndrome
Palavras-chave
Anestesia; Espectroscopia de Luz Próxima ao Infravermelho; Hipertensão; Oximetria; Síndrome do Roubo Subclávio

ABSTRACT

Subclavian steal is a rare condition caused by steno-occlusive disease in the proximal subclavian artery and systemic hypertension can be a physiologic response to maintain adequate cerebral perfusion.

Perioperative arterial pressure targets should be individualized specially in hypertensive patients and remains challenging in most patients. Cerebral oxymetry monitor can be a powerful tool in detection and correction of cerebral ischemia associated with arterial pressure drops.

RESUMO

A síndrome de roubo da subclávia é uma patologia rara causada por doença esteno-oclusiva da artéria subclávia proximal e a hipertensão arterial sistémica pode ser a resposta fisiológica para manter a perfusão cerebral adequada.

Os objetivos de pressão arterial no peri operatório devem ser individualizados, sobretudo em doentes hipertensos, e continua a ser desafiante na maioria dos doentes. O monitor de oximetria cerebral pode ser uma ferramenta valiosa na deteção e correção de isquemia cerebral associada a descidas da pressão arterial.

Autor Correspondente/Corresponding Author*:
Sara Alves
Morada: Sete Fontes, São Victor, 4710-243 Braga, Portugal
E-mail: sara.r.alves@hb.min-saude.pt

INTRODUCTION

Subclavian steal is a phenomenon caused by steno-occlusive disease in the proximal subclavian artery.

The stenosis decreases the arterial pressure in the subclavian artery distal to the lesion that is detected by measurement of arterial pressure in the ipsilateral arm. In cases of severe stenosis, the low pressure can cause the reversion of the blood flow in the ipsilateral vertebral artery and the flow of the contralateral subclavian artery is responsible for the blood supply of the involved arm through the contralateral vertebral artery.[1]

Most of the subclavian artery stenosis are asymptomatic. In rare cases, stenosis causes symptoms related with arterial insufficiency in the upper limb and brain. Large pressure difference between arms, exercise induced harm pain, fatigue or numbness are characteristics. Syncope, disequilibrium, vertigo, dizziness, ataxia and diplopia can be explained by vertebrobasilar ischemia.[1,2]

In all patients, cardiovascular risk factors should be corrected: control of hypertension and dyslipidemia, smoking cessation, glycemic control and antithrombotic therapy.

Patients with severe symptoms can be managed with endovascular treatment or open surgical bypass.

CASE REPORT

A 74-years-old female with a subclavian steal syndrome due to occlusive disease of the left subclavian artery was admitted for open carotid-vertebral bypass. Symptoms included episodes of dizziness and syncope with exercise of the left arm.

Two years before an angioplasty of the subclavian artery was tried without relieve of the symptoms.

She had an uncontrolled hypertension, medicated with nifedipin, bisoprolol, lisinopril, furosemide, spironolactone,

clonidine, doxazosine, trimetazidine and isosorbide mononitrate. Previous attempt to control hypertension was made by renal artery angioplasty. She had an obstructive sleep apnea, using continuous positive airway pressure at night.

Pre anesthetic evaluation revealed a significant difference in arterial pressure between both arms (left arm: 130-70 mmHg; right arm: 233-82 mmHg). In pre-operative echocardiogram, a severe pulmonary hypertension was found without left ventricular dysfunction.

In the day of the procedure, monitoring included EKG and ST deviation, pulse oximetry and unilateral processed EEG (BIS™ Medtronic) on the left side and the initial value was 94. Regional brain oxygen saturation (INVOS™ Medtronic) was used to detect cerebral ischemia.

Baseline regional brain oxygen saturation before anesthesia induction was 65 on the left side and 67 on the right side. Premedication was provided with midazolam and an intra-arterial catheter was inserted on the right arm (initial value of 255/76 mmHg). Anesthetic induction was provided with intravenous fentanyl (0.2 mg), propofol (100 mg) and rocuronium (50 mg). For maintenance of anesthesia a mixture of oxygen, air and sevoflurane was used.

After induction arterial pressure dropped to 176/44 mmHg with concurrent drop of regional brain oxygen saturation ($rScO_2$) of 12% in the left cerebral hemisphere and 21% in the right cerebral hemisphere (left 57%; right 53%).

The drop was corrected with intravenous boluses of 10 mg ephedrine and $rScO_2$ recovered to 69% on the left side and 68% on the right side.

One hour after surgical incision, another drop on arterial pressure (180/50 mmHg) and $rScO_2$ (left 52%; right 58%) was detected. No significant change on BIS value was detected.

A norepinephrine perfusion was started and maintained until the end of the surgery guided by $rScO_2$ baseline value.

Multimodal analgesia was provided with intravenous 1000 mg of paracetamol, 30 mg of ketorolac and 3 mg of morphine. Extubation occurred uneventfully.

After recovery no alterations were detected on neurologic exam. In the postoperative time, the patient was transferred to intensive care unit.

No neurological symptoms were detected and arterial pressure returned to pre-operative values. Discharge occurred four days after the surgery.

DISCUSSION

This case report highlights several questions about normality, risk assessment and individualized decision making.

Taking into account the history of uncontrolled stage 3 hypertension despite all efforts to treat this condition, either by antihypertensive ambulatory medication and renal artery angioplasty we hypothesized this hypertension could be a physiologic response to the subclavian steal syndrome.[3,4]

We decided to proceed to surgery with invasive arterial pressure and regional brain oxygen saturation monitoring (INVOS™ Medtronic) anticipating an altered state of equilibrium between these two variables. INVOS™ Medtronic monitor provides an easy and simple way to obtain a continuous measurement of frontal cortex oxygen saturation, based in near-infrared spectroscopy.[5]

During surgery, a decrease in arterial pressure below 180/50 mmHg implied concurrent cerebral desaturation which was corrected by an increase in arterial pressure with ephedrine bolus.

As these observations appeared to confirm our initial hypothesis of right shift of autoregulation interval, the decision was made to initiate a norepinephrine perfusion to stabilize arterial blood pressure and consequently cerebral perfusion pressure.

Fig. 1 presents intra-operative variation of arterial pressure and regional brain oxygen saturation.

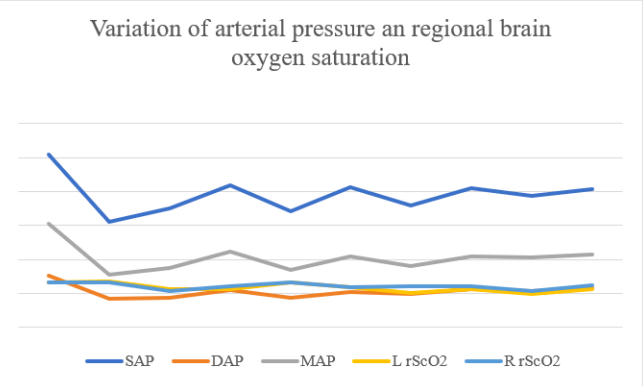

Figure 1. Intra-operative variation of arterial pressure and regional brain oxygen saturation
SAP: systolic arterial pressure; DAP: diastolic arterial pressure; MAP: medial arterial pressure; L $rScO_2$: regional brain oxygen saturation on left side; R $rScO_2$: regional brain oxygen saturation on left side.

It is uncommon, to say the least, that arterial pressure of 180/50 mmHg need to be corrected with noradrenalin. In this case, regional brain oxygen saturation helped us to establish the threshold to use vasopressors, maintaining systolic blood pressure over 190 mmHg most of the procedure.

Six months after surgery, in post-operative review, she was asymptomatic and blood pressure had dropped to 136/80 mmHg in left arm and 162/65 mmHg in right arm with the same anti-hypertensive medication. This came to reinforce our initial hypothesis. New technology allows us to characterize individual physiology parameters and therefore adapt our decision making. These may be the future of Anesthesiology in the operating room, tailor-made anesthesia.

Ethical Disclosures
Conflicts of Interest: The authors have no conflicts of interest to declare.
Financing Support: This work has not received any contribution, grant or scholarship.
Confidentiality of Data: The authors declare that they have followed the protocols of their work center on the publication of data from patients.
Patient Consent: Consent for publication was obtained.
Provenance and Peer Review: Not commissioned; externally peer reviewed.

Responsabilidades Éticas
Conflitos de Interesse: Os autores declaram a inexistência de conflitos de interesse na realização do presente trabalho.
Fontes de Financiamento: Não existiram fontes externas de financiamento para a realização deste artigo.
Confidencialidade dos Dados: Os autores declaram ter seguido os protocolos da sua instituição acerca da publicação dos dados de doentes.
Consentimento: Consentimento do doente para publicação obtido.
Proveniência e Revisão por Pares: Não comissionado; revisão externa por pares.

ORCID
Sara Alves https://orcid.org/0000-0003-0484-9223
Filipa Martins https://orcid.org/0000-0003-0381-7326
Tiago Faria https://orcid.org/0000-0001-6024-3221

Received: 26th of September, 2020 | Submissão: 26 de setembro, 2020
Accepted: 13th of January, 2021 | Aceitação: 13 de janeiro, 2021
Published: 22nd of March, 2021 | Publicado: 22 de março, 2021

REFERENCES

1. Osiro S, Zurada A, Gielecki J, Shoja M, Tubbs SR, Loukas M. A review of subclavian steal syndrome with clinical correlation. Med Sci Monit. 2012;18:57–63. doi: 10.12659/msm.882721.
2. Chafizadeh G, Rassekh E, Massih M, Behravesh M. Subclavian steal syndrome or brachiobasilar insufficiency syndrome. Dis Chest. 2013;53:649–51. doi: 10.1378/chest.53.5.649.
3. Lapage K, Wouters P. The patient with hypertension undergoing surgery. Curr Opin Anaesthesiol. 2016;29:397–402. doi: 10.1097/ACO.0000000000000343.
4. Ruland S, Aiyagari V. Cerebral autoregulation and blood pressure lowering. Hypertension. 2007;49:977–8. doi: 10.1161/HYPERTENSIONAHA.107.087502
5. Murkin JM, Arango M. Near-infrared spectroscopy as an index of brain and tissue oxygenation. Br J Anaesth. 2009;103:i3–13. doi: 10.1093/bja/aep299.