ISSN: 2377-4630

Corazzi et al. Int J Anesthetic Anesthesiol 2018, 5:066
DOI: 10.23937/2377-4630/1410066
Volume 5 | Issue 1
Open Access

International Journal of
Anesthetics and Anesthesiology

CASE REPORT

Transient Unilateral Lower Limb Palsy after Open Surgery Thoraco-Abdominal Aorta Repair

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Abstract

Paraplegia is one of the most devastating complication after Thoraco-abdominal Aorta (TAA) repair both in open surgery and in endovascular procedure, caused by critical obstruction to blood perfusion of the Spinal Cord.

Spinal Cord Ischemia (SCI) may be due to hemodynamic impairment and/or to segmental artery occlusion during aortic clamping [1,2].

We report a case of chronically dissected Thoraco-abdominal Aortic Aneurysm (TAAA) which showed unilateral transient neurological impairment of the left lower limb, postoperatively.

Keywords

Spinal cord injury, Aorta, Surgery

Case Report

A 78ys old male (ASA 3, BMI 26 kg/m^2) was submitted to open surgical TAA repair due to dissection of aneurism (Crawford 2 - Stanford B) [3,4].

Aorta was replaced by a prosthesis from the isthmus to iliac carrefour with re-implantation of celiac trunk, superior mesenteric artery and renal arteries. Two couple of intercostal and lumbar arteries were re-implanted to the prosthesis.

Anamnesis included cholecystectomy, hypertension and coronary artery by-pass graft (CABG) due to acute myocardial infarction (AMI), dislipidemia, G6PD deficit, allergy to milk proteins, liver steatosis and severe chronic obstructive pulmonary disease (COPD) with FEV1/FVC ratio 0.43 and FEV1 1.12 L (44%).

The pre-operative echocardiogram showed: Mild hypertrophy of left ventricle walls (13-14 mm), left ventricle end-diastolic volume at upper limit (LVEDVi = 74 ml/m^2), inferior wall and interventricular septum were akinetic providing a reduced ejection fraction (LVEF 0.38) that did ameliorate at dobutamine echo-stress (LVEF 0.47); furthermore it showed mild aortic valve stenosis (medium pressure gradient 20 mmHg with valve area 1.2 cm^2).

He underwent general anesthesia that was inducted by Midazolam 5 mg iv, Fentanyl 200 mcg iv. After myorelaxation with cis-Atracurium 12 mg iv a double-lumen endotracheal tube n.41 was inserted to connect the patient's airways to mechanical ventilator (Vt 560; respiratory rate 12; positive end-expiratory pressure 5 cm H_2O); he received a gas mixture of oxygen/air (FiO_2 40-60%) and Sevoflurane 1-2%, as appropriate. Myorelaxation continued with cis-Atracurium 1 mg/kg/h. Remifentanil 0.1-0.2 mcg/kg/min was administered.

During unilateral lung ventilation Vt was 420 ml and respiratory rate was adjusted according blood gas analysis (BGA).

Intraoperative monitoring included ECG (II and V), non-invasive blood pressure (NIBP), peripheral saturimetry (SpO_2) and end-tidal carbon dioxide ($EtCO_2$). After

Citation: Corazzi F, Brusa S, Giustiniano E, Civilini E, Poletto GL, et al. (2018) Transient Unilateral Lower Limb Palsy after Open Surgery Thoraco-Abdominal Aorta Repair. Int J Anesthetic Anesthesiol 5:066. doi.org/10.23937/2377-4630/1410066

Accepted: May 28, 2018: **Published:** May 30, 2018

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patient fell asleep a subarachnoid catheter (CODMAN® Johnson & Johnson 14G 40 cm) was inserted at L3-L4 level to monitor cerebro-spinal fluid pressure (P_{csf}) and lactate concentration (Lac_{csf}) and to drain the fluid aiming to assure $P_{csf} \leq 10$ mmHg.

We assured a double arterial line to monitor blood pressure invasively (ABP): Right radial artery (RRA) and right femoral artery (RFA). It permitted to monitor ABP due to heart function (RRA) and to left atrium-left femoral artery by-pass (LALFBP) bio-pump (Bio-Console® 560 - Medtronic, Inc., Minneapolis, MN, USA) during the clamping phase of the operation: Target values were systolic pressure 140-150 mmHg and blood pressure 60-70 mmHg, respectively.

Cardiac Index and Stroke Volume Variation were measured by the FloTrac/Vigileo™ (Edwards Life science, Irvine, CA, US).

During the operation, patient received trans-esophageal echocardiography (TEE) evaluation of cardiac function.

Unfractionated heparin (UFH) 5000 UI was administered 3 minutes before aortic clamping.

During supraceliac aortic clamping (98 minutes) the bio-pump assured blood delivery to splanchnic district by the right femoral artery cannulation. Renal Arteries were cannulated and kidneys perfused by cold (5 °C) Custodiol® HTK solution (Pvt) Ltd until renal circulation restored (300 ml bolus followed by 10 ml/min for 85 minutes).

After anesthesia induction hemodynamic impairment occurred and Dobutamine 2-5 mcg/kg/min needed to restore a safe cardiocirculatory setting. It was stopped after 30 minutes as cardiocirculatory assessment was satisfactory.

Five minutes after UFH administration, Activated Clotting Time (ACT) resulted 227 seconds. Then Aorta was clamped and bio-pump support started. It's blood flow ranged 1600-2100 ml/min according hemodynamics and blood loss. It provided a perfusional blood pressure of 60-85 mmHg. RRA mean blood pressure was 70-90 mmHg.

Total bleeding was 6200 ml out of which 2350 ml were collected in two red cell savers (SORIN Group Deutschland, Munich, Germany) and administered to the patient. Furthermore, 8 units (2300 ml) of concentrated red blood cells (RBC), 7 units (1793 ml) of fresh frozen plasma (FFP) and 1 unit of pool-Platelets were transfused. Total crystalloid and colloid (Uman Albumin 20%) solutions resulted 3100 ml and 300 ml, respectively.

FloTrac/Vigileo™ and TEE during operation guided the administration of fluids and vasoactive drugs. During the clamping phase Norepinephrine (NE) 0.03-0.05 mcg/kg/min needed to assure the hemodynamics goals.

Target Cerebro-Spinal Fluid pressure (P_{csf}) was ≤ 10 mmHg and when it was greater 10 ml of CSF were withdrawned. It resulted always sufficient to restore the target P_{csf}.

Table 1 reports the main intraoperative data.

Table 1: Intraoperative data.

	After induction	Aortic clamping	LALFBP pump off	Aortic unclamping	Operation end
RRA MAP (mmHg)	80	90	70	50	60
RFA MAP (mmHg)	75	85	60	43	50
pH	7.40	7.20	7.32	7.36	7.34
P/F ratio	707	351	406	253	268
pCO_2 (mmHg)	42	47	44	43	44
Lactatemia (mmol/L)	0.6	0.6	4	4.4	4.5
BE (mmol/L)	1.0	-8.9	-3.2	-1.1	-2
Hb (g/dL)	11.8	6.8	8.1	6.5	7.8
Lac_{csf} (mmol/L)	0.8	1.6	-	1.8	2.2
P_{csf} (mmHg)	12	9	12	13	8
CSF drainage (ml)	40	20	10	10	-
CI (L/min/m²)	2	2.3	2.5	3.2	3
SVV (%)	5	11	22	10	12
Blood loss (ml)	-	500	1100	2000	6200
Cristalloids (ml)	500	1500	2000	2500	3100
Colloids (ml)	-	100	100	100	300
Red Blood Cells (unit)	-	-	4	7	8
Fresh Frozen Plasma (unit)	-	-	2	5	7
Pool-PLTs (unit)	-	-	-	1	-
Diuresis (ml)	-	-	-	120	250

RRA MAP: Right Radial Artery Mean Arterial Pressure; RFA MAP: Right Femoral Artery Mean Arterial Pressure; P/F ratio: pO_2/FiO_2; BE: Base Excess; Hb: Hemoglobin; Lac_{csf}: Lactate concentration in cerebro-spinal fluid; P_{csf}: Cerebro-spinal Fluid Pressure; CSF: Cerebro-Spinal Fluid.

Table 2: Postoperative data.

Postoperative hours	0	6*	16**	24	48	72
pH	7.11	7.15	7.49	7.51	7.56	7.40
Lactatemia (mmol/L)	9.6	11.2	4.2	1.3	1.0	1.0
Lac_{csf} (mmol/L)	4.6	6.9	4.6	1.7	1.6	1.7
P_{csf} (mmHg)	16	18	15	18	14	15
PP_{csf} (mmHg)	44	45	68	72	79	78
NE (mcg/kg/min)	0.09	0.10	0.15	0.05	-	-
Ep (mcg/kg/min)	0.15	0.15	0.05	-	-	-
CSF Drainage (ml)	-	20	10	5##	5##	5##
CI (L/min/m^2)	3.2	3.3	3.4	4.1	2.8	3.3
SVV (%)	15	18	10	8	7	8
MAP (mmHg)	60	63	83	90	93	95
HR (bpm)	72	110	90	82	55	55
Diuresis (ml/h)	40	32	60	80	100	100
Hb (g/dL)	9.4	7.7	11.5	11.5	11.3	10.2
Platelets count (/mm^3)	51000	92000	72000	71000	62000	47000
Serum Fibrinogen (mg/dL)	71	133	213	202	233	365
Bleeding (ml/h)	300	150	10	-	-	-
Inferior limbs motility	not evaluable§	Left limb deficit#	Left limb deficit	Recovered motility	Recovered motility	Recovered motility

*Patient re-entered operative room for surgical hemostasis; **Patient returned to ICU after surgical hemostasis; §Patient sedation; #After sedation stopped for 1 h; ##To measure Lac_{csf}; Lac_{csf}: Lactate concentration in cerebrospinal fluid; P_{csf}: Cerebro-spinal Fluid Pressure; CI: Cardiac Index; SVV: Stroke Volume variation; MAP: Mean Arterial Pressure; HR: Heart Rate; Hb: Hemoglobin.

Postoperative analgesia was performed by a double supra-fascial multi-holed catheter 22.5 cm 19-gauge (Painfusor Catheter© 2017 Plan 1 Health, Amaro-Udine, Italy) through which Ropivacaine 0.4% was administered at a rate of 10 ml/h.

At the end of the operation, patient continued sedation by Propofol 2% 10 ml/h and Remifentanil 0.05 mcg/kg/min. The two-lumen endotracheal tube was substituted by a single lumen oro-tracheal tube n.8 and patient was admitted to our ICU for postoperative surveillance and mechanical ventilation support. Table 2 shows the post-operative data.

At the ICU admission, BGA showed compensated lactic acidosis and a significant bleeding that continued for 6 hs. A first "sedation window" (1 h stopped Propofol and Remifentanil) showed a motor deficit of the left lower limb.

Blood loss was thought to be due to coagulation system impairment because serum Fibrinogen was 71 mg/dL and Platelet counts lowered to 51000/mm^3, International Normalized Ratio (INR) was 1.79. Since surgeons did not indicate operative revision, medical treatment was chosen.

After Fibrinogen administration 3 g i.v. serum concentration reached 133 mg/dL. Furthermore 10 units of RBC, 5 units of FFP and 1 unit of pool-PLTs were administered to maintain a sufficient serum Hb and aiming to recover the coagulation system alteration. Hemodynamics needed to be supported by Norepinephrine 0.09 mcg/kg/min and Epinephrine 0.15 mcg/kg/min (after cardiac ultrasounds examination).

Since therapy resulted unsuccessful, patient re-entered the operative room for surgical hemostasis that was decisive. After the re-operation he returned to ICU. A second longer "sedation window" confirmed the left lower limb paresis. Sensitive function was not adequately evaluable as the patient showed psycho-motor agitation (upper limbs and right lower limb) and was not collaborative. P_{csf} was < 10 mmHg and Lac_{csf} resulted 4.6 mmol/L. Mean arterial pressure (MAP) was 83 mmHg and the difference MAP - P_{csf} (i.e. the perfusion pressure of spinal cord, PP_{csf}) was 68 mmHg. Then we modified the vasoactive drug (NE) administration rate to reach a better perfusion pressure of the spinal cord (> 70 mmHg).

At 48 post-operative hours PP_{csf} was 79 mmHg and Lac_{csf} lowered to 1.6 mmo/L. During the preceding 12 hours sedation was completely stopped and we could observe a recovering of the neurological deficit of the left lower limb.

During the following ICU-staying patients experienced a worsening of respiratory function due to pneumonia and atelectasis of the left lower lung lobe, that was treated as appropriate (antibacterial drugs, long-lasting mechanical ventilation even in prone position).

Discussion

We reported a case of transient unilateral plegia of the left lower limb in a subject who underwent open surgical repair of chronically dissected TAAA.

We consider that the peculiarity of such a case was the one-side neurological deficit subsequent to the operation and its complete recovering.

Post-operative SCI is a devastating complication following TAA surgery. Literature reports about cases of

paraplegia after this kind of surgery is plentiful, but a one-side lower palsy lasting only few hours post-operatively has not been published as yet, at our knowledge.

During the operation we monitored intratecal pressure (P_{csf}) and the spinal cord perfusion pressure (PP_{csf}) along with Lactate concentration into CSF (Lac_{csf}) because it may be a marker of local ischemic suffering [5-8]. Lac_{csf} and P_{csf} did not showed significant alterations, intraoperatively. On the contrary, P_{csf} lowered just after surgery consequently to hemodynamic impairment due to severe bleeding and Lac_{csf} increased. Then we may infer that SCI occurred during hemorrhage despite blood transfusions and vasoactive drugs administration.

After surgical hemostasis, intrathecal pressure recovered, but lactate concentration into cerebrospinal fluid, even lowered, remained above normal values [5].

Unexpectedly, after 72 hours post-operation Lac_{csf} recovered and lower left limb sensitive-motor function restored.

The unique publication we found about unilateral lower limb paralysis reports a case about abdominal aortic aneurysm repair. It is a rare complication after aorto-iliac surgery: The diagnosis was ischemic lumbar plexopathy (Magnetic Resonance Imaging of the spine was negative 3 weeks later). The patient showed a partial deficit when discharged [9].

Nowadays we know the vascularization of the spine cord better than when the arteria radicularis magna (ARM) was described for the first time [10]. Nevertheless, we are not able to explain all post-surgical spine cord damage due to ischemia with certainty, as yet.

Our case was not investigated by Magnetic Resonance Imaging because of severe respiratory impairment requiring mechanical ventilation and then we considered very dangerous moving the patient to Radiology Department for the exam. Furthermore, the neurologist consultant did not indicate further exams because the patient recovered from unilateral palsy completely.

We are aware it is a great limitation to this case report. But, despite this limitation, we consider it may be worthy of report due to the rarity of the case and also to overcome the anecdotal passing on the knowledge about such an issue.

Conflict of Interest

Authors declare they have not any conflict of interest and they did not received any fund for such a report.

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