Intensive Care Med (2011) 37:357–359
DOI 10.1007/s00134-010-2063-6

Armin Seibel
Andreas Müller
Samir G. Sakka

Indocyanine green plasma disappearance rate for monitoring hepatosplanchnic blood flow

Accepted: 24 September 2010
Published online: 28 October 2010

Dear Editor,

Increased intra-abdominal pressure (IAP) leading to reduced organ blood flow may cause abdominal compartment syndrome (ACS) [1]. However, measurement of IAP in the urinary bladder, as is most widely practiced, may be misleading—especially after surgical procedures, when different compartments may exist [2, 3]. Furthermore, assessment of tissue oxygenation by global parameters, i.e., central venous oxygen saturation ($ScvO_2$), may be limited. Recently, indocyanine green plasma disappearance rate (ICG-PDR) has been suggested for bedside assessment of hepatosplanchnic blood flow [4]. Here, we describe a patient with normal systemic oxygen transport (i.e., $ScvO_2$) and normal IAP but markedly reduced ICG-PDR who was finally confirmed by relaparotomy to be suffering from gut hypoperfusion.

A 64-year-old male (75 kg, 1.78 m) was transferred from a district hospital after complaining of diarrhea and abdominal pain. On admission, angiography revealed mesenteric artery occlusion, and explorative laparotomy was performed. Intraoperatively, extended ischemia of small intestine and colon due to thrombotic occlusion of the arteria mesenterica superior was found. The arteria mesenterica inferior was not detected, since the patient had received an aorto-bifemoral prosthesis several years ago. The artery was reopened, and a bypass from the arteria lienalis to the arteria mesenterica superior was placed. After surgery, the patient was brought to the intensive care unit (ICU) while still on vasopressor support. Extended hemodynamic monitoring (PiCCO™; Pulsion Medical Systems AG, Munich) and IAP monitoring (urinary bladder technique, using 20 ml 0.9% NaCl) were established. ICG-PDR (LiMON™; Pulsion Medical Systems AG, Munich) measured by 0.25 mg/kg ICG was 10.9%/min (IAP 14 mmHg, $ScvO_2$ 74%), and dobutamine was added (Table 1).

Two days later, IAP was 24 mmHg and relaparotomy was indicated. At this time, ICG-PDR was 8.2%/min and $ScvO_2$ 65%. Since gut ischemia in the distal duodenum/proximal jejunum and colon was found, these

Table 1 Global hemodynamics, regional and systemic oxygen transport variables, and vasoactive drugs

	Emergency surgery			Relaparotomy		Final surgery				
	Day 1	Day 2	Day 3 6 a.m.	Day 3 6 p.m.	Day 4	Day 5	Day 6	Day 7	Day 8	Day 15
MAP (mmHg)	64	57	96	91	80	65	91	71	71	77
CVP (mmHg)	11	15	27	16	16	16	19	9	18	15
CI (l/min/m^2)	2.7	3.4	3.9	3.5	4.1	4.2	3.0	3.7	4.5	×
$dpdt_{max}$ (mmHg/s)	1,328	1,360	1,720	1,725	1,778	1,190	1,940	2,386	1,465	×
ITBVI (ml/m^2)	827	948	887	884	850	959	937	919	1,005	×
EVLWI (ml/kg)	8.7	7.1	10.6	9.6	7.8	7.9	9.0	10.6	9.9	×
SVRI (dyn s cm m^2)	1,510	1,030	1,420	1,700	1,230	960	1,950	1,370	940	×
SVV (%)	28	22	17	7	16	12	15	15	16	×
$ScvO_2$ (%)	74	75	65	78	78	88	85	80	83	×
Lactate (mmol/l)	1.6	1.6	2.7	1.8	1.6	1.8	1.8	1.2	1.2	1.6
ICG-PDR (%/min)	10.9	8.5	8.2	8.5	8.6	7.6	7.2	6.6	6.4	×
IAP (mmHg)	14	18	24	2	13	7	10	8	2	×
NEPI (µg/kg/min)	0.13	0.13	0.27	0.27	0.15	0.15	0.24	0.04	0.01	0.13
DOB (µg/kg/min)	0	4.44	4.44	4.44	4.44	4.44	4.44	2.22	2.22	2.22
Fluid balance (ml)	7,000	10,150		1,600	770	110	1,550	−750	−640	×
PCT (mg/dl)	2.33	1.55	4.76		4.06	2.79	2.31	1.83	1.38	1.44
CRP (ng/ml)	26.8	17.7	21.5		21.1	15.5	23.4	25.5	28.2	31.3

MAP mean arterial pressure, *CVP* central venous pressure, *CI* cardiac index, *$dpdt_{max}$* maximum rate of pressure increase in systole, *ITBVI* intrathoracic blood volume index, *EVLWI* extravascular lung water index, *SVRI* systemic-vascular resistance index, *SVV* stroke volume variation, *$ScvO_2$* central venous oxygen saturation, *ICG-PDR* indocyanine green plasma disappearance rate, *IAP* intra-abdominal pressure, *NEPI* norepinephrine, *DOB* dobutamine

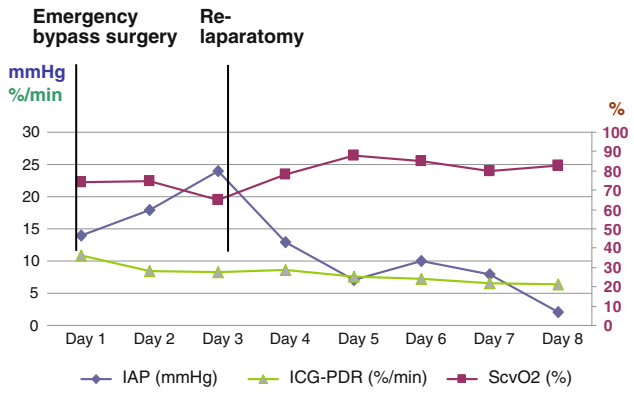

Fig. 1 Intra-abdominal pressure (*IAP*), indocyanine green plasma disappearance rate (*ICG-PDR*), and central venous oxygen saturation ($ScvO_2$) during the course. In contrast to ICG-PDR, neither $ScvO_2$ nor IAP as parameters of tissue hypoxia and intra-abdominal hypertension indicated the severity of disease with existing mesenteric ischemia

parts were removed. An ileostoma was placed, and the abdomen was definitively closed.

Postoperatively (IAP 2 mmHg, $ScvO_2$ 78%), ICG-PDR was still unchanged (8.5%/min) and further dropped, while global and other laboratory variables did not change or even stabilized (creatinine: 2.8 mg/dl on admission, <1.2 mg/dl on day 3). Also, leukocyte count, procalcitonin, and serum transaminases dropped, while C-reactive protein slightly increased. However, conjugated bilirubin increased (1.7 mg/dl after decompression, 3.8 mg/dl on day 5), while unconjugated bilirubin was unchanged (0.1 mg/dl). Over the following days, the patient was hemodynamically stable under reduced dosages of vasoactive drugs and was mobilized while breathing spontaneously in assisted mode. Secretion over abdominal drainages was always serous, and the amount decreased continuously. In the light of this clinical course, no specific interventions were undertaken despite the low ICG-PDR. On day 15, feces came over the abdominal drainages and operative revision confirmed incurable situs due to multiple gut perforations. Unfortunately, the patient died a few hours later (Fig. 1).

In general, extravasation of fluids may lead to ACS, and treatment should focus on recognition and reduction of IAP. However, the instruments for assessing organ function (anuria, lactate, bilirubin) may only be possible with limitations and time delay. Liver venous catheterization and angiography are invasive techniques and not screening instruments, for which ICG-PDR has been suggested. In our case, no chronic liver disease was evident, and low ICG-PDR was related to poor liver function/perfusion. Notably, hepatic protein synthesis improved (cholinesterase from 933 to 1,642 U/l), which may be interpreted as adequate liver function during inadequate gut perfusion. Also, $ScvO_2$, lactate, and IAP did not help to identify the problem, while ICG-PDR has been found to react adequately to changes in IAP [5]. Previous data indicate that anastomosis insufficiency in the gut is related to compromised microcirculation in the intramural micro-arcades [6], which may be aggravated by insufficient cardiac output or increased IAP. In our case, normal IAP was associated with markedly reduced ICG-PDR while global parameters were not indicative for gut hypoperfusion, which has been addressed previously [7]. Only $ScvO_2$ increased temporarily to supranormal values (88%), as can be observed in sepsis. However, other clinical signs and markers such as hemodynamics, procalcitonin, and blood cultures did not indicate systemic infection. Unfortunately, though gut hypoperfusion was not ruled out, low ICG-PDR in a recovering patient was not regarded sufficient to indicate surgical revision.

In conclusion, ICG-PDR seems to be an important marker for detecting persistent hypoperfusion in hepatosplanchnic circulation, especially in case of intra-abdominal volume reduction or abdomen left open after surgery [8]. However, the findings presented here should be regarded as a trigger for future investigations assessing ICG-PDR for monitoring organ perfusion.

Conflict of interest S. Sakka is a member of the Medical Advisory Board of Pulsion Medical Systems AG, Munich and has received honoraria for giving lectures from this company and from MSD Sharp Dohme, Munich. A. Seibel has received honoraria from Pulsion Medical Systems AG, Munich, for giving lectures.

References

1. Sieh KM, Chu KM, Wong J (2001) Intraabdominal hypertension and abdominal compartment syndrome. Langenbeck Arch Surg 386:53–61
2. Malbrain ML, Wilmer A (2007) The polycompartment syndrome: towards an understanding of the interactions between different compartments. Intensive Care Med 33:1869–1872
3. Malbrain ML, De Laet IE, Willems A, Van Regenmortel N, Schoonheydt K, Dits H (2010) Localised abdominal compartment syndrome: bladder-over-gastric pressure ratio (B/G ratio) as a clue to diagnosis. Acta Clin Belg 65:98–106
4. Thümer O, Hüttemann E, Sakka SG (2007) Indocyanine green plasma disappearance rate. Marker of partial hepato-splanchnic ischemia. Anaesthesist 56:339–344
5. Sakka SG (2007) Indocyanine green plasma disappearance rate as an indicator of hepato-splanchnic ischemia during abdominal compartment syndrome. Anesth Analg 104:1003–1004
6. Shekarriz H, Wedel T, Eckmann C, Nolde J, Bruch HP (2004) Ursachen der Anastomoseninsuffizienz am Darm: anatomische und physiologische Untersuchungen mit klinischen Konsequenzen. German Medical Science; DOC 04 dgch 1392

7. Poeze M, Solberg BC, Greve JW, Ramsay G (2005) Monitoring global volume-related hemodynamic or regional variables after initial resuscitation: What is a better predictor of outcome in critically ill septic patients? Crit Care Med 33:2494–2500
8. Michelet P, Roch A, Gainnier M, Sainty JM, Auffray JP, Papazian L (2005) Influence of support on intra-abdominal pressure, hepatic kinetics of Indocyanine green and extravascular lung water during prone positioning in patients with ARDS: a randomized crossover study. Crit Care 9:R251–R257

A. Seibel
Department of Anesthesiology, Intensive Care Medicine, and Emergency Medicine, Evangelisches Jung-Stilling-Krankenhaus Siegen, Siegen, Germany

A. Müller
Department of Visceral, Chest, and Vascular Surgery, Evangelisches Jung-Stilling-Krankenhaus Siegen, Siegen, Germany

S. G. Sakka (✉)
Department of Anesthesiology and Operative Intensive Care Medicine, Medical Centre Cologne-Merheim, University of Witten/Herdecke, Ostmerheimerstr. 200, 51109 Cologne, Germany
e-mail: SakkaS@kliniken-koeln.de
Tel.: +49-221-89073863
Fax: +49-221-89078666