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Indocyanine green plasma disappearance rate during relief of increased abdominal pressure

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Sir: Indocyanine green plasma disappearance rate (ICG-PDR) has been suggested [1, 2] and is currently increasingly used for assessment of liver perfusion and function. Although the details regarding this parameter are still not fully elucidated and its usefulness has been questioned recently [3], elimination of indocyanine green, which physiologically is exclusively removed from the blood by the liver, is reliably assessed by ICG-PDR [4]. In general, ICG-PDR has been described to react very quickly following interventions, i.e., drug administration [5]. Here, I present the case of a 46-year-old man with chronic heart failure due to dilatative cardiomyopathy and clinical diagnosis of liver congestion with an increased intra-abdominal pressure due to several liters of ascites. The intra-abdominal pressure as measured by the urinary bladder catheter technique using

50 ml of normal saline for filling was 18 mmHg. Laboratory tests taken during abdominal sonography and immediately before ascites puncture were: serum protein 49 g/l, albumin 20 g/l, hemoglobin 5.1 mmol/l, Quick 79%, INR 1.2, aPTT 47 s, central venous O₂-saturation (ScvO₂) 56.6%, lactate 1.0 mmol/l, ASAT < 0.2 μmol/l*s, ALAT 0.21 μmol/l*s, alkaline phosphatase 1.56 μmol/l*s, cholinesterase 25 μmol/l*s (normal 75–190), γGT 0.59 μmol/l*s (normal < 0.92 μmol/l*s) and GLDH < 50 nmol/l*s (normal < 120 nmol/l*s). In this patient, ICG-PDR as measured transcutaneously by a commercially available system (LiMon; Pulsion Medical Systems, Munich, Germany) was 11.6%/min and 15-min residual rate 17.6%. After local anesthesia and test puncture, 3,000 ml of clear ascites (protein 20.8 g/l, albumin 11.4 g/l) was then removed without complications over about 30 min. Repeated measurement of the intra-abdominal pressure revealed 12 mmHg. ICG-PDR measured 30 min after end of paracentesis and thus 1 h after initial measurement was 15.6%/min (15-min residual rate 9.6%). Central venous blood gas analysis was repeated: ScvO₂ 55.2%, lactate 1.1 mmol/l, hemoglobin 5.1 mmol/l. For substitution of protein loss, 200 ml of 20% albumin was infused after decompression over the next 4 h. The patient did

not receive vasoactive drugs during the study period, and central venous pressure as a marker of fluid status was not different between the two time points (13 mmHg). Notably, the changes in regional blood flow (ICG-PDR) were not visible on the global level (ScvO₂). In detail, ScvO₂ was low due to the underlying cardiac disease but slightly decreased, lactate levels remained unchanged (but were normal), and hemoglobin was unchanged (Table 1). These findings suggest an absence of hemoconcentration, and slight decrease in cardiac output, that may have happened in the context of massive ascites puncture without concomitant fluid administration.

In conclusion, since liver cell function is unlikely to change within this short time period, the increase in ICG-PDR may indicate an increase in hepatic blood flow that resulted from decompression of the abdomen by paracentesis. ICG-PDR may be regarded as an attractive bedside tool to assess short-time changes in hepatic blood flow, especially since measurable reliably by means of a transcutaneous system.

References

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Table 1 Global and regional perfusion and oxygen transport parameters before abdominal decompression by ascites puncture and 1 h later after decompression

	Before decompression	After decompression
ICG-PDR (%/min)	11.6	15.6
ICG 15-min residual rate (%)	17.6	9.6
Lactate (mmol/l)	1.0	1.1
ScvO ₂ (%)	56.6	55.2
Hemoglobin (mmol/l)	5.1	5.1

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