Annals Oncology & Cancer Case Reports

Mini Review

Volume 1 | Issue 5

Serum Lactate in Liver Resection Surgery. A Mini-Review

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Received: 12 Apr 2021
Accepted: 03 May 2021
Published: 08 May 2021

Citation:

Nisi F. Serum Lactate in Liver Resection Surgery. A Mini-Review . Annals Onco & Cancer Case Rep. 2021; V1(5): 1-4.

1. Abstract

Intermittent Pringle maneuver (PM), i.e. temporary clamping of the hepatic hilum, has been pointed out as safe and useful for reducing blood loss during liver resection, but it exposes the patient the risk of ischemia-reperfusion liver injury. Serum lactate (sLac) concentration depends on the balance between production and clearance from the blood stream, and it has been reported to be a predictor of outcome in critically ill patients, including those with liver failure, sepsis and trauma.The patients undergoing to liver resection differ from critically ill patients, but this type of hepatic surgery may be somehow compared with critical illness, as major surgery causes a certain degree of Systemic Inflammatory Reaction Syndrome (SIRS). In such operations, postoperative acidosis may be mainly due both to high level of serum chloride (in case of large amount of NaCl 0.9% saline solution administered intra-operatively) and to hyperlactatemia. We reviewed the most recent Literature about this issue, in particular into the field of hepatic resection surgery.

2. Text

Intermittent Pringle maneuver (PM), i.e. temporary clamping of the hepatic hilum, has been pointed out as safe and useful for reducing blood loss during liver resection [1], but it exposes the patient the risk of ischemia-reperfusion liver injury [2]. Serum lactate (sLac) concentration depends on the balance between production and clearance from the blood stream, and it has been reported to be a predictor of outcome in critically ill patients, including those with liver failure, sepsis and trauma [3–10]. Although the peak serum concentration of Lactate may correlate with outcome [11, 12], its clearance (cLac) seems to be a better predictor [13].

The sLac metabolism follows a complex pathway, particularly during and after liver surgery. In liver surgery, high sLac levels can be related to several clinical factors:

- impairment in lactate metabolism (i.e. extraction and utilization by the liver) or overproduction by splanchnic organs.
- the fluid regimen administration: according the ERAS (Enhanced Recovery After Surgery) protocols, intraoperative fluid management should be limited, but an excessively restrictive fluid regimen (also in order to limit the intraoperative backflow bleeding), may cause a low-flow state.
- ischemia-reperfusion syndrome due to the hepatic blood flow clamping itself; in this case, Lactate clearance will depend also on the residual amount of hepatic tissue after the resection, [14, 15]. In critical patients sLac is an epiphenomenon of low blood flow state, hypoxia and other mechanisms that make the cellular metabolism switch on anaerobic setting [3,4].

The patients undergoing to liver resection differ from critically ill patients, but this type of hepatic surgery may be somehow compared with critical illness, as major surgery causes a certain degree of Systemic Inflammatory Reaction Syndrome (SIRS), [16,17]. In

such operations, postoperative acidosis may be mainly due both to high level of serum chloride (in case of large amount of NaCl 0.9% saline solution administered intra-operatively) and to hyperlactatemia [18].

The PM induces indeed a significant increase in sLac levels during liver resection, particularly in the event of compromised liver function such as in cirrhosis and steatosis or when its duration is prolonged. High intraoperative sLac concentration has been reported to have prognostic significance after hepatectomy [18-22].

Investigations about lactatemia and its correlation with postoperative liver function provided contrasting results, [23,24]. Data regarding the clearance of lactate in patients undergoing liver resection under intermittent PM are still lacking [19,20]. Several studies on trauma patients showed that prolonged cLac was associated with increased mortality, and this was confirmed in critical surgical patients too [25].

reported that the Lactic acid accumulation seems to depend on the capability of the liver to clear Lactate from the blood, and such a function is related to the duration of the hepatic blood inflow limitation [26]. The correlation between Lactate and PM seems to exists both in terms of serum concentration and in terms of Lactate Clearance (cLac). When the hepatic hilum clamping lasts more than 76 min, the cLac during the first postoperative period has been reported to be poor, but it improves as the hours go by. In our experience, whatever the PM duration, more or less than 76 minutes, the post-operative clearance of serum Lactate showed a similar trend with that we named as the "square-root" shape, as it draws a curve which reminds us of the mathematical sign of the square root, (Figure 1). The clearance of Lactic Acid generally falls immediately after surgery (causing lactatemia increasing), but improves during the following hours whilst serum Lactate concentration reduces. The peculiar significant difference in lactate clearance associated with different duration of PM (i.e. longer or shorter than 76 minutes) was observed after the first two postoperative hours [27].

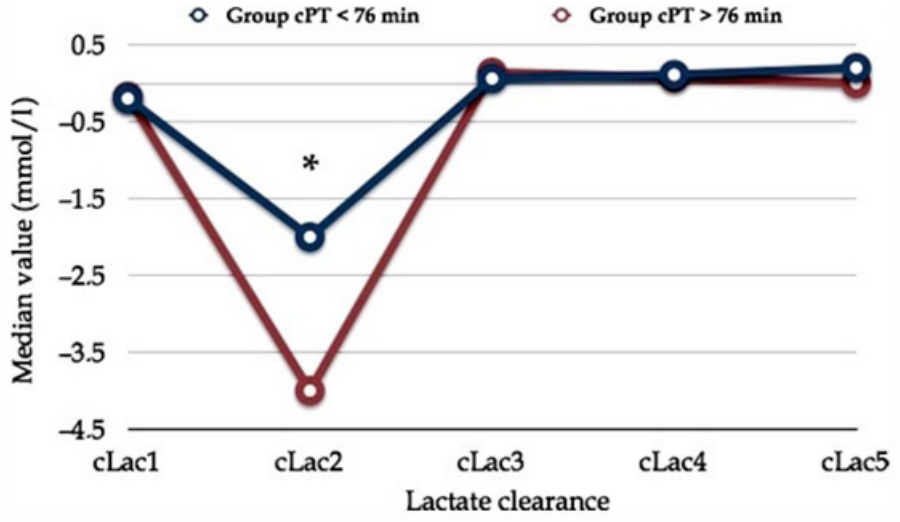

Figure 1: The "Square-root" shape

Furthermore, to date, the correlation between the length of the operation and postoperative sLac has not been clearly investigated. In an observational trial carried out at our Institution, we found that a very long surgical time (>300 minutes) was associated with a high postoperative serum Lactate concentration, perhaps because even the Pringle Maneuver duration was prolonged during a long lasting surgery. Moreover, in the same study we observed post-resection hyperlactatemia (>4 mmol/L) was also associated with an increased heart rate (and consequently a higher Cardiac Output): this could occur to compensate for anemia due to the blood loss [27].

Some hours after hepatic resection, hyperlactatemia reduction may be due to a less restrictive fluid management, but it surely depends on the postoperative residual liver volume and function as well. In other words, sLac trend may be a marker of the ability of the liver to clear Lactic acid [19-21].

reported that a Goal-Directed Fluid Therapy (GDFT), guided by a semi-invasive hemodynamic monitoring device, in open abdominal surgery, induced a more rapid normalization of serum Lacate level [28]. Despite being aware that our patients were different from those enrolled by, although we used the same hemodynamic monitoring, we did not find any correlation between Cardiac Output and fluid regimen with the blood Lactate concentration and its clearance, at least intraoperatively. Our protocol of fluid management was a safe compromise between maintaining a sufficient circulating volume and avoiding fluid load, aimed at avoidance of any negative effect on outcome [29-31].

In several trials it has been demonstrated that Cardiac Output

monitoring during major surgery reduces the postoperative complications rate, [32]. Accordingly, we improved the outcome of our patients undergoing liver resection using intraoperative semi-invasive hemodynamic monitoring as this ameliorated the fluid management, [33].

Finally, the background condition of the liver tissue showed to be related with serum Lactate concentration. In particular, cirrhotic/chronic hepatitis patients had higher levels of sLac, even before the operation started. Conversely, we found cLac was never different when compared to normal and steatotic subjects. May this mean that the hepatic tissue health does not always affect the clearance of Lactate? Furthermore, normal liver unexpectedly resulted a risk factor for hyperlactatemia even more than steatosis. Hence, we speculated that the preoperative chemotherapy might be a relevant factor. Indded, only 30% of patients with normal liver received chemotherapy, compared to 80% of steatotic patients, and this difference was statistically significant, [27]. According to previous Literature reports and based on our finding, we dare to say that chemotherapy may exert a sort of "preconditioning" effect on ischemia-reperfusion tolerance because of the hepatic microcirculation changes provoked by chemotherapy itself, [35, 36]. Such a supposed mechanism would be only speculative since Literature does not report significant data about this issue, to the best of our knowledge. Certainly, it deserves more specific investigation trials.

3. Conclusion

the serum Lactate concentration may be considered the "core-marker" of the whole clinical postoperative assessment of the hemodynamic and metabolic setting of the patients who undergo hepatic resection. After such a major operation, patients often need postoperative Intensive Care Unit (ICU) surveillance. To date, the ICU-bed availability and the indication for ICU admission remains a worldwide debated matter, reflecting patient factors and resources availability, [37]. In this context, the clinical relevance of serum Lactate concentration may be used as one of the criteria to decide whether or not the patient deserves postoperative ICU admission.

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