Qualitative and Quantitative Changes in Thoracic Duct Lymph during Canine Experimental Shock

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Summary

Changes of the body fluid exchange and of the composition of metabolites in the hepatosplanchnic area in canine hemorrhagic and endotoxin or septic shock models were studied by investigating the qualitative and quantitative changes in thoracic duct lymph draining from abdominal organs. In the present study, it might be summarized that the changes in the flow rate and composition of thoracic duct lymph were put forward to much more directly and apparently indicate the degree of hepatosplanchnic cellular impairment in canine experimental shock than in the circulating blood.

It may be assumable that thoracic duct lymph in the resting animal is primarily a derivative of hepatosplanchnic interstitial fluid which, in turn, can probably be influenced by hemodynamic or metabolic alterations occurring across the hepatosplanchnic capillary bed in shock state.

Experiments were designed to study the changes in the flow rate and composition of thoracic duct lymph, probably resulting from the changes in the fluid exchange and tissue metabolism in the hepatoplanchnic area of anesthetized dogs in shock state, and also to evaluate an effect of the adrenocorticosteroid preparation on the impaired metabolism in experimental shock models.

Materials and Methods

Mongrel dogs were anesthetized with sodium pentobarbital, and the respiration was assisted by a mechanical respirator. Flow rate of lymph was determined by cannulating the thoracic duct and collecting lymph by gravidity drainage into appropriate calibrated test tubes. Arte-

rial blood pressure, portal and central venous pressure were recorded continuously throughout the course of experiments, and specimens of lymph, arterial and portal venous blood were undergone analyzing of protein, albumin, osmotic pressure, lactate, pyruvate, 5-HT, lysozyme, acid phosphatase, electrolytes and so forth. Experimental shock models were consisted of hemorrhagic shock induced by either the exsanguination at a rate of 30 ml/kg or the reservoir bottle procedure, and septic shock by i. v. injection of either E. coli endotoxin (2 mg/kg) or live E. coli organisms (1 x 10 to the 5, 7, 8, 9 or 10th organisms/kg).

Results

- 1. A prompt rise in thoracic duct lymph was observed when bleeding was initiated in hemorrhagic shock group. Once the bleeding was controlled with the stable blood pressure at a lower level, lymph flow tended to be below the control value. However, in irreversible shock state, lymph flow frequently rose above the normal levels, associated with an increase in the output of lactate or lysosomal enzymes via the thoracic duct (1).
- 2. Immediately after the i. v. injection of endotoxin, there was a prompt and definite increase in thoracic duct lymph flow which paralleled a rise in portal vein pressure at the early stage after the endotoxin injection (Fig. 1). Even when the portal vein pressure returned toward normal 10 to 15 minutes after the administration of endotoxin, lymph flow was still maintained at a high level. These phenomenon may implicate that the initial rise in lymph flow may probably be due to the increased hepatic lymph flow,

followed by the subsequent response induced by the increased intestinal lymph flow.

- In endotoxin shock group, thoracic duct lymph appeared hemorrhagic as the shock progressed, and the increased lymph flow was associated with an enhanced output of protein, albumin, potassium, lactate, 5-HT and lysosomal enzymes.
- 4. Comparing to the changes of the concentrations of compositions in blood, changes in the output of lactate, lysozyme or 5-HT via the thoracic duct were more evidently observed in endotoxin shock group.
- 5. The similar response in lymph was, also, induced by i. v. injection of a large doses (1 x 10 to the 9 or 10th organisms/kg) of live E. coli organisms.
- 6. Comparing to that in the control group, a lesser degree of lymphatic response in pretreatment with the portacaval shunt formation or total resection of small intestine was observed after the injection of endotoxin (Fig. 2).
- Pretreatment with 10 mg/kg of hydrocortisone suppressed the increase in the output of lactate via the thoracic duct and, also, the rise of excess lactate in blood and lymph after the injection of endotoxin.

Discussion

It might be assumable that, with dogs immobile under anesthesia, lymph in the thoracic duct drains from abdominal organs only.

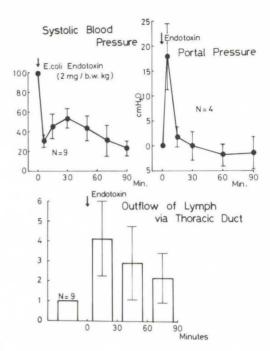
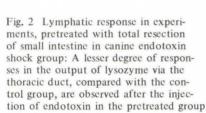
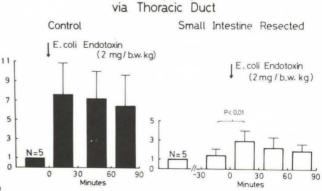


Fig. 1 Hemodynamic and lymphatic response in canine endotoxin shock: Increased lymphatic flow is still maintained even when the portal vein pressure returns toward normal

The present study was planned to study thoracic duct lymph as the reflection of the well-known hemodynamic and metabolic events during shock from abdominal organs. Similar responses of the lymphatic system in the present study, as shown in the previous investigations (2, 3, 4, 5), indicated that changes of the fluid exchange and metabolism





Output of Lysozyme

in the splanchnic area might be more accurately reflected in thoracic duct lymph than in the circulating blood during shock in dogs. It has been supposed that, in endotoxin shock, the vasculature of liver might be an initial target area and next of the intestine, as mentioned by *Alican* and *Hardy* (2). It is, also, noted that even a smaller dose of adrenocortical hormone, compared with the dose described by *Williams* and *Clermont* (5), might be effective for eliminating the metabolic impairment in the endotoxin group, if given prior to the injection of endotoxin.

Conclusion

The changes in the flow rate and composition of thoracic duct lymph was put forward to indicate the degree of hepatoplanchnic cellular impairment in canine experimental shock models.

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