

# Terlipressin treatment for gastrointestinal bleeding reduces serum sodium

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A new study published in the October issue of the journal *Hepatology* found that patients with severe portal-hypertensive bleeding who are treated with terlipressin may experience an acute reduction of sodium in their blood. This reduction in serum sodium, known as hyponatremia, can cause adverse reactions such as neurological complications, and is rapidly reversible upon terlipressin withdrawal. Researchers suggest that serum sodium should be closely monitored in these patients and caution that use of solutions with high sodium content to treat this condition may cause a too rapid recovery of sodium leading to adverse events.

Cirrhosis and portal vein [thrombosis](#) are two of the primary causes of severe portal hypertension—an increase in blood pressure of the (portal) vein between digestive organs and the liver. This increase in pressure contributes to the development of varices, or large veins, that can weaken over time and lead to [gastrointestinal bleeding](#). Terlipressin is commonly used to treat acute variceal bleeding, however its effect on serum sodium is largely unknown and the focus of the current retrospective study, led by Pere Ginčs, MD, from the Hospital Clínic in Barcelona, Spain.

The study included 58 consecutive patients treated with terlipressin for gastrointestinal bleeding due to portal-hypertension. Median age of participants was 54 years, with 77% of the cohort being male and 22% female. Initially patients were treated with somatostatin, but switched to terlipressin due to uncontrolled bleeding or rebleeding in 57% and 43% of cases, respectively.

Researchers noted a significant reduction in serum sodium concentration during terlipressin treatment (from 134.9 at baseline to 130.5 mEq/L at day 5 of treatment). A reduction of sodium in the blood was found in 67% of patients with 31% having a moderate decrease (5-10 mEq/L) and 36% experiencing a marked decrease in serum sodium (greater than 10mEq/L). Only 19 patients were determined to have no change in serum sodium levels.

Further results indicate that patients with a low model for end-stage liver disease (MELD) score and normal or near-normal baseline serum sodium had the highest risk of hyponatremia. "The reduction in serum sodium concentration, common in terlipressin therapy, develops rapidly after onset of treatment, said Dr. Gines. "In some patients hyponatremia can be severe, leading to neurological complications which usually resolve upon withdrawal from treatment."

The research team found that 3 of the 21 patients who had a marked reduction in serum sodium developed neurological manifestations. Two of the patients with neurological complications improved after withdrawal of therapy and administration of hypertonic saline. One patient who developed a progressive impairment in neurological status leading to coma, did not improve after terlipressin withdrawal and treatment with hypertonic saline, and later died due to multiorgan failure.

"In patients with high risk of reduction in serum sodium levels (low MELD scores and normal or near-normal baseline serum sodium concentration), serum sodium concentration should be monitored closely during treatment and terlipressin stopped if hyponatremia develops. Hypotonic fluids should probably be avoided to prevent a further reduction in serum sodium concentration," concluded Dr. Ginčs. "Physicians should be aware of our findings in order to prevent the possible [neurological complications](#) related to acute hyponatremia or

rapid recovery of serum sodium levels."

**More information:** "Hyponatremia in Patients Treated with Terlipressin for Severe Gastrointestinal Bleeding due to Portal Hypertension." Elsa Solà, Sabela Lens, Mónica Guevara, Marta Martín-Llahí, Claudia Fagundes, Gustavo Pereira, Marco Pavesi, Javier Fernández, Juan González-Abraldes, Angels Escorsell, Antoni Mas, Jaume Bosch, Vicente Arroyo, and Pere Ginçs. *Hepatology*; Published Online: August 5, 2010 [DOI: 10.1002/hep.23893](https://doi.org/10.1002/hep.23893)

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