Blood transfusion during haemodialysis improves systemic tissue oxygenation: A case report

La transfusión de sangre durante la hemodiálisis mejora la oxigenación sistémica del tejido: un reporte de un caso

Dear Editor,

Tissue oxygenation is maintained through various mechanisms, including blood pressure, circulating blood volume, and hemoglobin (Hb) concentration. In particular, Hb itself is considered an important factor because of its oxygen transportation function to systemic organs. Therefore, it is evident that oxygen supply would decrease in a severe anemic state.[1] Recent reports have demonstrated the measurement of regional oxygen saturation (rSO_2), a real-time marker of tissue oxygenation using near-infrared spectroscopy (NIRS).[2,3] Measurement of rSO_2 by using NIRS is a straightforward non-invasive procedure, which can be performed continuously. Nevertheless, only a few studies have investigated the association between systemic tissue oxygenation and the increase in Hb levels after blood transfusion in hemodialysis (HD) patients. In the current case study, we were able to monitor changes in the rSO_2 of brain, liver, and lower-limb muscles during HD with and without blood transfusion.

A 79-year-old woman undergoing HD was admitted to our hospital with acute obstructive suppurative cholangitis. Upon admission, we administered intravenous antibiotics and performed endoscopic biliary drainage, which resulted in a gradual improvement of her symptoms. Her anemia had been previously managed with an erythropoietin-beta (3000 IU/session, 3 times/week); however, her Hb levels decreased to 6.8 g/dL. Therefore, blood transfusion during HD was performed. She provided written informed consent to participate in monitoring of her systemic rSO_2 during HD with or without blood transfusion. The rSO_2 levels were monitored at the forehead, right hypochondriac region above the liver, and lower leg above the gastrocnemii muscles by using the INVOS 5100c (Covidien Japan). She received a transfusion of 560 mL of concentrated red blood cells during HD and her Hb levels increased from 6.8 to 10.0 g/dL after HD (Table 1). Furthermore, we compared the time course of rSO_2 ratio in each organ, with and without blood transfusion. The rSO_2 ratio was defined as the ratio of rSO_2 values at t (min) during HD and initial rSO_2 value before HD. As shown in Fig. 1, the changes in rSO_2 ratio during HD without blood transfusion were modest in each organ, whereas the rSO_2 ratio at each organ increased with blood transfusion, particularly in cerebral and hepatic regions.

In the world including United States, Europe, and Japan in around 1990, the use of erythropoiesis stimulating agents (ESA) in clinical settings was approved, which dramatically improved the Hb levels in patients with HD. However, even in the present day, blood transfusion during HD is still necessary for improving Hb levels in HD patients with severe anemia. Indeed, in comparison of blood transfusion frequency between ESA responsive and hyporesponsive HD patients, hyporesponsive patients had approximately 5 to 7-fold higher burden of blood transfusion than those with responsiveness.[4] In addition, changes in systemic oxygenation induced by blood transfusion during HD have not been investigated extensively.

The brain has an auto-regulatory mechanism to maintain cerebral oxygenation. In HD patients, normalization of hematocrit by ESA did not increase cerebral oxygenation.[5]

Table 1 – Vital signs and laboratory findings under HD with or without blood transfusion.

	HD without blood transfusion	HD with blood transfusion
Body weight, kg		
Before	59.0	58.9
After	57.8	57.9
BP, mmHg		
Before	147/71	118/93
After	149/74	173/75
Pulse, beats/min		
Before	78	85
After	77	75
Hb, g/dL		
Before	7.1	6.8
After	7.2	10.0
BUN, mg/dL		
Before	38	26
After	7	5
Cr, mg/dL		
Before	4.3	3.7
After	1.0	0.9
Albumin, g/dL		
Before	2.9	2.8
After	3.0	3.0

BP, blood pressure; BUN, blood urea nitrogen; Cr, creatinine; Hb, hemoglobin; HD, hemodialysis.

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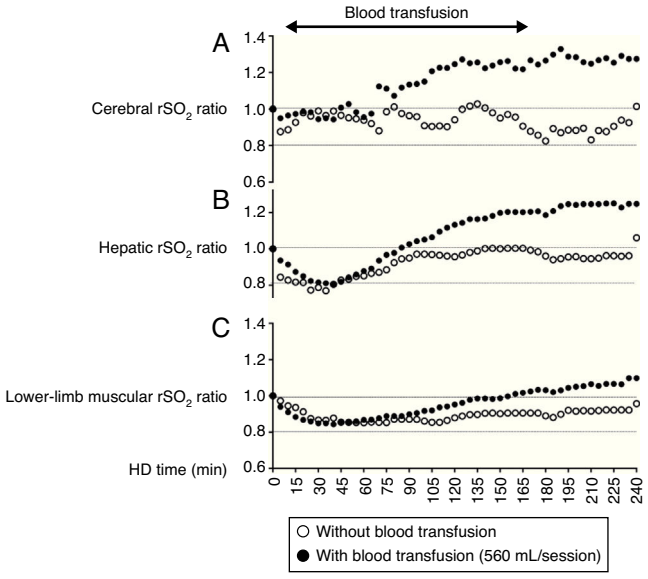

Fig. 1 – Changes in regional oxygen saturation (rSO_2) of the forehead (A), liver (B), and lower leg (C) as per the oxygenation values of cerebral, hepatic, and muscle tissue, respectively, under hemodialysis (HD) with or without blood transfusion. rSO_2 ratio is defined as the ratio of rSO_2 value at *t* (min) during HD and the initial rSO_2 value before HD (rSO_2 at *t* (min) during HD/initial rSO_2 before HD).

Moreover, cerebral oxygenation in these patients was well-maintained when compared to pre-dialysis patients,[6] and cerebral rSO_2 values did not change by ultrafiltration under well-managed Hb levels.[7] In the present case, rSO_2 values did not change in any organ during HD without blood transfusion, which is similar to a previous report.[7] On the other hand, each rSO_2 values improved with an increase in Hb levels after blood transfusion. Particularly in the brain, oxygen supply decreases in patients with severe anemia[1]; furthermore, acute anemia by phlebotomy induces the deterioration of intracellular oxygen reactions in mice.[8] Thus, presence of severe anemia may lead to a decrease in cerebral oxygenation despite the auto-regulatory mechanism of the brain. Therefore, the improvement of cerebral oxygenation after blood transfusion could be explained by the increase of oxygen-carrying capacity, which is associated with the increase in Hb levels.

Regarding the changes in rSO_2 of each organ after blood transfusion, the improvement of lower-limb muscular rSO_2 was relatively lower than that of cerebral and hepatic rSO_2 even in this case without peripheral artery disease. In HD patients, the prevalence of subclinical peripheral artery disease reached around 20–25%,[9] and its presence may directly influence the lower-limb muscular rSO_2 via a decrease in oxygen supply induced by the dysfunction of macro- and micro-circulation. Furthermore, the skeletal muscle index has been reported to be lower in HD patients than in healthy subjects.[10] Therefore, the changes in lower-limb muscular rSO_2 value during HD might be influenced by the circulatory impairment and skeletal muscle weakness. However, the mechanism responsible for the differences in rSO_2 improvement between different organs, induced by blood transfusion during HD, remains unclear and requires further investigation.

Our study suggests that blood transfusion during HD could be an effective method to improve tissue oxygenation, particularly cerebral and hepatic oxygenation in HD patients with severe anemia.

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Conflict of interest statement

The authors have declared that no conflict of interest exists.

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Glomerular involvement in patient with sickle cell disease☆

Afectación glomerular en paciente con enfermedad falciforme

Dear Editor,

Sickle cell nephropathy is one of the complications of sickle cell disease (SCD); this is due to the polymerization of deoxygenated hemoglobin S in the renal medulla, a place of special physiological conditions (hyperosmolarity, hypoxia and acidosis)[1,2] which contributes to the typical obstruction of vessels.[3]

Chronic renal failure and proteinuria are the risk factors associated with increased mortality in these patients.[4] Albuminuria is the initial marker of SCD associated glomerulopathy, the most frequent expression of which is focal and segmental glomerulosclerosis (FSG).[5,6] Renal biopsy is indicated if glomerulopathy is suspected.

We present the case of a 30-year-old white male, with a history of homozygous sickle cell disease that was treated with hydroxyurea and deferasirox, he develops 1–2 annual crises, with no renal repercussions until present. Smoker of 3 cigarettes per day.

The patient was sent for consultation in relation of proteinuria of 2.75 g in 24 h (albuminuria: 1.5 g/24 h). The urine density was 1006, pH: 5.5, proteinuria 100 mg/dl and a normal sediment. Renal function was normal (Cr: 0.7 mg/dl and MDRD > 60 ml/min); Hb: 10.8 g/dl; Hto: 30% (TSI: 80%, folic acid: 8.2 ng/ml; VitB: 570 pg/ml) and CRP: 6.6 mg/l. The expanded study with autoimmunity and hepatitis C and B viruses as well as HIV is negative. In abdominal ultrasound, the kidneys do not have morphological abnormality and the spleen was small with diffuse increase of echogenicity suggesting fibrosis after repeated infarctions.

Once proteinuria was confirmed, a renal biopsy was performed that showed glomerular hypertrophy without sclerosis, enlargement of the glomerular capillaries and the presence of sickle cells within the capillaries (Fig. 1). The tubules have cells with haemosiderin by Perls staining and occasional areas of atrophy associated with fibrosis seen with trichrome staining (Fig. 2A and B). Interstitial vessels do not reveal abnormalities and direct immunofluorescence shows absence of deposits.

The patient is diagnosed of sickle cell glomerulopathy. After one year with valsartan, 80 mg/day, proteinuria decreased to 1.6 g/24 h (albuminuria: 1.1 g/24 h) and the mean blood pressure was 111/63 mmHg by ABPM, values did not allow an increase in doses of ARA-II or double blockage of the renin–angiotensin system due to risk of symptomatic hypotension. At present, renal function is maintained normal

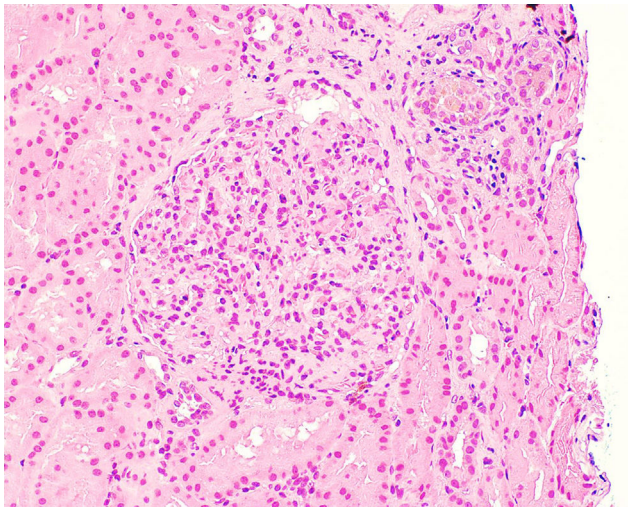

Fig. 1 – Hematoxylin–eosin. Glomerular hypertrophy.

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