TURKISH JOURNAL OF EMERGENCY MEDICINE

Case Report/Case Series

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Quick Response Code:

Website: https://turkjemergmed.com/

DOI: 10.4103/tjem.tjem_30_24

Posterior Reversible Encephalopathy Syndrome (PRES) following blood transfusion in a polytrauma victim, an atypical occurrence

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Abstract:

Posterior reversible encephalopathy syndrome (PRES) is a neuroradiological syndrome, clinically present by impaired consciousness, headache, visual disturbances, and seizures, and radiologically brain edema. Cases of PRES induced by blood transfusion are rarely documented. We report this case to increase the awareness of treating physicians for the possible complications of rapid blood transfusion. A 29-year-old man presented with polytrauma and was in hemorrhagic shock. He was transfused with multiple transfusions. Later, he was found to have quadriplegia with minimal movement of fingers in the left hand. His computed tomography showed cerebral edema in multiple cerebral regions. We propose that the etiology in this case is that rapid blood transfusion induced acute rise in hemoglobin which led to PRES. The influences of blood transfusion on blood flow, blood viscosity, and endothelial dysfunction lead to blood–brain barrier dysfunction, which can result in PRES.

Keywords:

Anemia, blood transfusion, posterior reversible encephalopathy syndrome

Introduction

Several neurological complications are described in polytrauma and can be categorized by etiology or clinical presentations. Neurovascular complications include strokes, intracerebral hemorrhages, spinal cord ischemia, or development of traumatic arteriovenous fistulae.[1] Posterior reversible encephalopathy syndrome (PRES) has not been reported in polytrauma cases as a direct complication. Prompt identification and initiation of management of the underlying cause are crucial. In polytrauma, the neurologic examination is often confounded by discomfort, sedative medication use, mechanical ventilation, and distracting injuries.[1]

Regular sedation interruptions for examination and keeping a high index of suspicion for neurologic complications are necessary, principally since prompt identification and management can improve the functional outcomes. PRES was described first time as a clinicoradiological entity in 1996.[2] PRES commonly presents by acute neurological manifestations, such as headache, impaired consciousness, visual disturbances, and seizure.[3] It is characterized by vasogenic edema affecting the posterior cerebral areas, predominantly

Submitted: 13-02-2024
Revised: 02-05-2024
Accepted: 06-05-2024
Published: 01-07-2024

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How to cite this article: Shariff E, Soltan NM. Posterior Reversible Encephalopathy Syndrome (PRES) following blood transfusion in a polytrauma victim, an atypical occurrence. Turk J Emerg Med 2024;24:180-4.

parietal and occipital lobes. However, infratentorial as well as anterior cerebral region involvement is described in the literature.[4] The outcome of PRES is good in the form of amelioration and resolution of clinical and radiological findings.[5] However, atypical cases have been reported with cytotoxic edema and permanent neurological sequelae, without hypertension.[5,6] PRES commonly happens with hypertensive crisis, autoimmune disorders, exposure to immunosuppressive agents, eclampsia, and renal failure.[2] PRES following red blood cell transfusion has been reported in few numbers of patients.[7] Failure of the blood–brain barrier (BBB) and endothelial dysfunction are the underlying mechanisms common to all eliciting events; however, the exact pathogenesis is still controversial.[8]

We report a case of a patient who developed PRES in the context of rapid packed red blood cell (pRBC) transfusion needed for hemorrhagic shock. This patient showed cerebral edema in the frontoparietal and occipital regions on computed tomography (CT) head and developed quadriplegia. There is a need to emphasize the carefulness of massively transfusing blood and to involve the patients and their caregivers in the counseling of possible neurological sequelae.

Case Report

Patient information

We had a 29-year-old male patient, previously healthy with no neurological, autoimmune disease and no use of any particular medications.

Clinical findings

The patient was brought to the emergency department (ED) by Red Crescent as a case of gunshot injury to the thorax and abdomen. Polytrauma teams were involved immediately upon arrival to the ED. He had signs of hypotensive shock in the form of pale skin, sweating, tachycardia, and severe hypotension. His heart rate was 112 BPM, blood pressure (BP) 61/40 mmHg, mean arterial pressure (MAP) 47 mmHg, temperature 35.6°C, and respiratory rate was 30/min. His body was covered with the fresh blood with gunshot entry point over the right side of the thorax and abdomen. His neurological examination showed the Glasgow Coma Scale of 4/15 (verbal 2, motor 1, and eye 1), pupils reactive bilaterally with no facial asymmetry, and bilateral mute planters.

Diagnosis and management

He was intubated and attached with mechanical ventilator. Two intravenous (IV) lines were inserted immediately, and he was started on IV fluid. He had decreased air entry on the right side of the chest. After chest tube insertion, he had a cardiac arrest, cardiopulmonary resuscitation was started, and he was revived in 2 min. The patient underwent thoracotomy with repair of thoracic injury, exploratory laparotomy, and small bowel resection. Upon arrival, he received 9 pRBCs, 3 pooled platelets, 8 fresh frozen plasma, and 10 cryoprecipitate within 24 hours to secure the homeostasis. BP started to improve following surgical repair with a reading of 93/63. He was kept on vasopressors: norepinephrine and vasopressin to keep the target MAP above 65 mmHg. Initial hemoglobin (Hb) was 4 g/dL, which improved to 9.5 g/dL after transfusion. He had elevated C-reactive protein (CRP), procalcitonin, and coagulation profile which gradually improved to normal level. His level of consciousness improved in next 2 days after stopping the sedation. However, he was found to have quadriplegia with minimum movement of fingers in the left hand. CT head without contrast [Figure 1a] was performed after the stabilization, and it showed multiple cortical and subcortical hypodensities, edema, and loss of gray-white matter differentiation involving bilateral frontal lobes, bilateral superior parietal lobes, and bilateral occipitoparietal regions and initially interpreted as acute ischemic infarctions; however, his CT angiogram showed normal cerebral vessels [Figure 1b]. Electroencephalogram (EEG) was performed, and it showed diffuse slowing and was consistent with nonspecific encephalopathy [Figure 2].

Follow-up and outcomes

His neurological status improved gradually. After 18 days, he had mild dysarthria, mild left-sided ataxia, and mild right-sided hemiparesis with brisk deep tendon reflexes, sustained clonus, and extensor planter response on the right side. MRI brain could not be performed due to the bullet injury. Follow-up CT head [Figure 3] which was done 18 days after the first CT head shows significant interval regression of the previously seen cerebral edema involving bilateral frontoparietal region. The patient was discharged home after 33 days with minimal right-sided weakness and appendicular ataxia.

Patient perspective

The patient was critically ill upon presentation and had not been aware of the management. After the recovery, he was informed about the medical/neurological complications he had and possible etiologies as well. He was counseled about the nature of neurological insult and possible recovery in due time; he showed a good understanding and attended all his follow-ups.

Discussion

PRES is an acute neuroradiological entity, and the syndrome can involve or extend beyond the posterior brain regions.[9] The well-known etiologies of PRES are drugs,

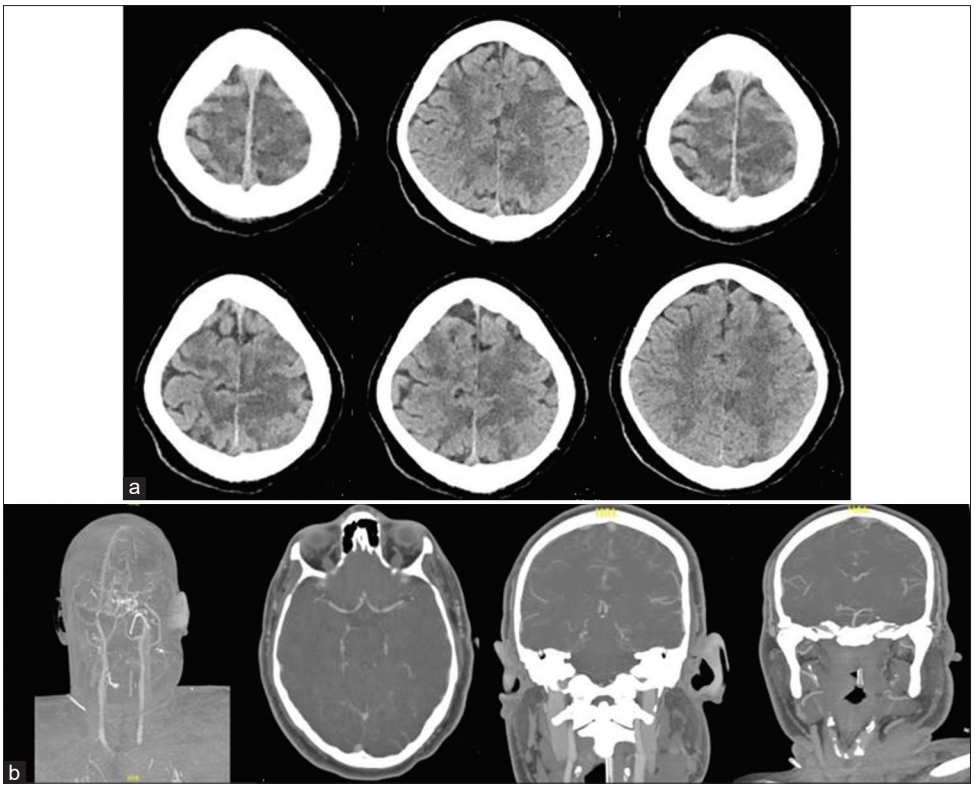

Figure 1: (a) Axial computed tomography head without contrast shows multiple cortical and subcortical hypodensities, edema, and loss of gray-white matter differentiation involving bilateral frontal lobes, bilateral superior parietal lobes, and bilateral occipitoparietal regions. (b) Computed tomography angiogram with contrast shows patent cerebral vessels

Figure 2: Electroencephalogram epochs (longitudinal bipolar montage and average reference montage, sensitivity; 7 mic V/mm, low-frequency filter; 1 Hz, high-frequency filter; 70 Hz, time base; 30 mm/s); diffuse slow-wave activity

hypertension, preeclampsia, renal failure, autoimmune disorders, and, in our case, blood transfusion.[10] Although recovery from PRES is common in most cases when managed appropriately, some patients ended up with permanent brain damage and are left with residual neurological sequelae.[11] Some reports have described poor prognostic factors for PRES such as advanced age, higher CRP level, cause of PRES, altered coagulation profile, altered mental status at onset, and presence of subarachnoid hemorrhage.[11] In our report, the patient had very high CRP, procalcitonin, and altered coagulation profile. These abnormal results in our case were most likely related to acute phase reactivity in the phase of acute injury and massive blood loss. PRES commonly presents with headache, seizures, and visual disturbances; however, the presentation can be quite diverse and may include ataxia, focal neurological deficits, or vertigo.[12] The patient had relatively uncommon manifestations of PRES in the form of corticospinal tract involvement and ataxia. The patient was comatose during the CT head examination; hence, we were unable to ascertain the presence of other manifestations of PRES, such as headache or visual disturbances. However, the EEG revealed no epileptiform abnormalities, and there were no clinical seizures observed. When his level of consciousness improved, he was found quadriplegic with no visual field defects and no sensory impairment. He remained normotensive after initial hemorrhagic shock was treated by huge blood transfusions, and blood transfusion was the trigger after an extensive search for other possible etiologies. Previously, transfusion-related PRES showed similar cases where the Hb level was

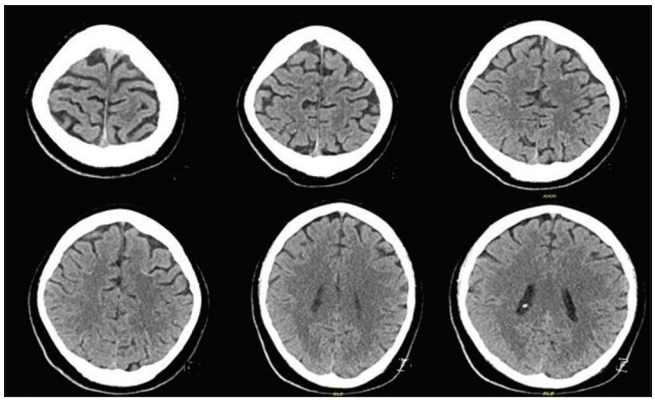

Figure 3: Axial computed tomography head without contrast shows resolution of previously shown lesions

increased by at least 5 g/dl.[13] The patient showed an increase in Hb by 6 g/dl over few hours. Follow-up CT head showed almost complete resolution of cerebral lesions in both hemispheres. The patient was discharged from the hospital requiring minimal assistance. There are few case reports in the literature that reported PRES following blood transfusion;[7] however, all of them were related to correction of chronic anemia by blood transfusion. Blood transfusion may give rise to a quick increase in the circulatory blood volume, which can cause cerebral blood flow overload. Sudden or acute cerebral hyperperfusion surpasses the ability of autoregulation of cerebral capillary perfusion pressure. This could result in vasogenic cerebral edema which presents as PRES. In addition, the likelihood of severe anemia as the predisposing factor for inducing the PRES cannot be excluded. Anemia-related insufficient oxygen supply might result in endothelial cell dysfunction, further causing a functional loss or damage to the integrity of the BBB in capillary circulation.[7] The patient did not have a high reading of BP throughout his hospital course; therefore, we hypothesize that only transfusion-induced mechanism is responsible for PRES in the present case. The literature has reported several cases of transfusion-induced PRES without evidence of hypertension,[14] suggesting that an acute increase in blood volume, irrespective of BP, may contribute to the induction of PRES in the context of significant blood transfusion. Our patient was quite different; he had acute blood loss due to the gunshot injury and received huge transfusions to secure the hemostasis.

Conclusion

We propose that it is crucial to recognize the potential for rapid blood transfusion-induced PRES. This awareness enables medical practitioners to either mitigate the rapid increase in Hb levels or to inform the patient and their families about the potential risks associated with rapid transfusion, particularly in cases of life-threatening blood loss, as exemplified in our scenario.

Acknowledgments
This study made use of the computational resources and technical services of the Scientific and High-Performance Computing Center at Imam Abdulrahman Bin Faisal University, Dammam, Kingdom of Saudi Arabia.

Author contribution statement
ES contributed to provide the main idea of reporting the case, and revising the manuscript. NS wrote the initial manuscript.

Conflicts of interest
None Declared.

Informed consent
Written informed consent was obtained from the patient to participate in this study.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

Funding
None.

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