

A Rare Case Report of Subdural Hematoma with Aplastic Anemia

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Abstract

This article illustrates the development of a rare case of spontaneous subdural hematoma (SDH) secondary to aplastic anemia and conservative treatment of SDH. Clinical Presentation: a 43-year-old male complained of severe progressive headaches that starting from one month ago. His laboratory values showed pancytopenia and his peripheral blood smear showed no abnormalities except for the lack of number of erythrocytes, leukocyte, and thrombocyte. We could not find any malignancy in the smear. The patient experienced progressive headache, disorder of balance, and decrease of consciousness. CT imaging of the head showed a 7.0 cm (2 cm thickness) left frontal-parietal subdural hematoma. Patient were managed conservatively. This is a rare case of aplastic anemia with subdural hematoma and the patient was discharged in good condition.

Keywords: anemia, aplastic, subdural hematoma

Studi Kasus: Pasien dengan Subdural Hematoma dan Anemia Aplastik

Abstrak

Pasien dengan subdural hematoma spontan yang disebabkan oleh anemia aplastik dan ditatalaksana non-operatif tidak banyak dilaporkan. Studi kasus ini membahas seorang laki-laki, 43 tahun, dengan nyeri kepala yang bertambah berat sejak 1 bulan yang lalu serta mengeluhkan lemah dan lesu. Pemeriksaan laboratorium menunjukkan pansitopenia dan pemeriksaan pembuluh darah perifer menunjukkan kekurangan jumlah eritrosit, leukosit, dan trombosit serta tidak ditemukan adanya tanda keganasan dalam sediaan. Pasien mengeluhkan nyeri kepala yang bertambah, gangguan keseimbangan dan penurunan kesadaran. CT scan kepala menunjukkan 7,0 cm (ketebalan 2 cm) perdarahan subdural di bagian fronto-parietal kiri. Tatalaksana non operatif dilakukan pada pasien yang membaik dan pulang dengan kondisi perbaikan. Studi kasus ini menggambarkan kasus yang jarang yaitu anemia aplastik dengan subdural hematoma di kepala dan dilakukan tata laksana non operatif.

Kata Kunci: anemia, aplasia, perdarahan subdural

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Introduction

Anemia aplastic (AA) is a condition that bone marrow has been damaged because of toxic agents or idiopathic in origin. This could be regarded as deficits in the numbers of all three cell lineages and lack of cells in the bone marrow and no increase in reticulin. The presenting symptoms of aplastic anemia can be seriously fatal.

Anemia, bleeding, and infection are usually presenting symptoms. Some patients developed signs of intracranial hypertension due to pseudotumor cerebri (PTC).¹⁻³ PTC or is known as Idiopathic intracranial hypertension, is delineated by normal size cerebral ventricles and high pressure inside the skull with accompanied papilledema.³

Another rare intracranial presentation was intracranial hemorrhage. There are 19.2% of patients developed intracranial bleeding and this caused mortality in up to 27.3% of patients with AA.⁴ Yamasaki (1989) encountered four patients who experienced six episodes of intracranial hemorrhage (1 subdural hematoma, 3 intracerebral hemorrhages, 2 subarachnoid hemorrhages).⁵ There was a case reported by Menger that administration of trimethoprim-sulfamethoxazole resulted in aplastic anemia and simultaneously spontaneous subdural hematoma.⁴

A study showed that among 9,627 patients there were 106 (1.1%) of which diagnosed with hematologic disorders and ICH. There was a

significant higher rate of ICH in aplastic anemia than any other benign malignancies.⁶

We present the subdural hemorrhage in an aplastic anemia patient, but the etiology is still unknown.

Case Presentation

A 43-year-old Asian male with a history of progressive headache presented to the emergency department. He stated that his headache started one month ago. He felt pain all over his head, but sometimes the left side was more prominent than the right side.

He complained of frequent tiredness when he works. He could not stand straight because of dizziness. He denied any history of taking antibiotics or having a fever before. He denied any history of high blood pressure and diabetes mellitus. He did not have any history of trauma and stroke. He did not smoke or drink alcohol. He was a school teacher with a lot of sedentary activities.

Physical examination showed that He was pale and alert. His physical diagnosis BP=100/80 mmHg, HR= 82 beats/min. CT head showed a 7.0 × 2 cm left frontoparietal subdural hematoma with shift to the right. (Figure 1). Because the patient was awake and without any neurological deficits, the neurosurgeon on duty observed the condition, and postpone the burr-hole evacuation. Laboratory values haemoglobin 5 gr%, leucocyte 5,000/mm³, Hematocrit 10 volume%, platelet count 20,000/mm³. Lymphocyte absolut 1,600, Netrophil lymphocyte ratio 1.24.

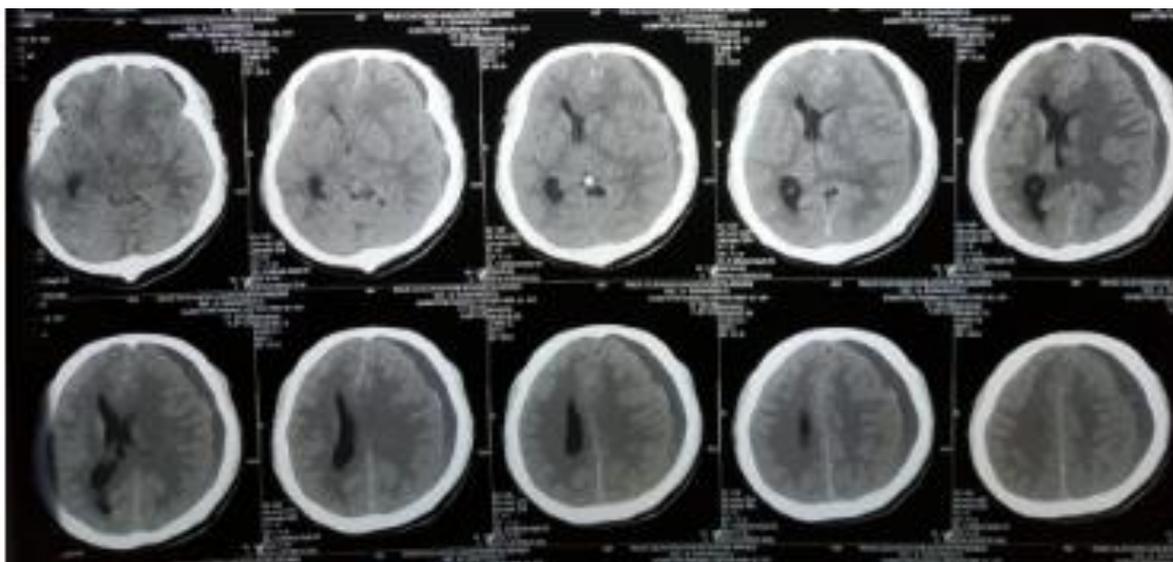


Figure 1. Head CT Scan Non Contrast of Anemia Aplastic Patient with Subdural Hematoma. There was a Midline Shift and Hipodense Crescent Appearance at Left Frontoparietal with Thickness Maximum was 2

Neutrophyl showed shift to the right. The laboratory values showed normal liver function test and normal kidney function test, random blood glucose showed 95 mg/dL and , LED >140

mm/hour. Peripheral Blood Smear of Aplastic Anemia with Subdural Hematoma showed normal small erythrocyte, no sign of malignancy cells (Figure 2).

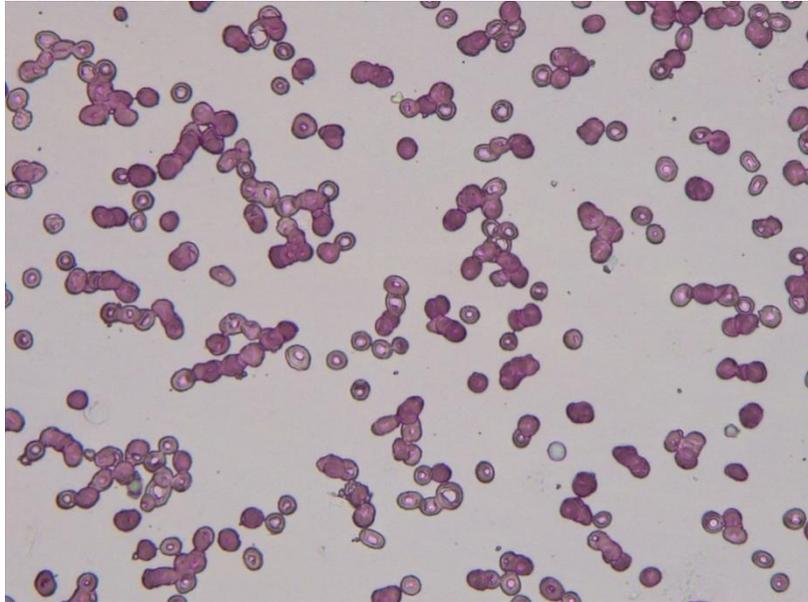


Figure 2. Peripheral Blood Smear of Anemia Aplastic with Subdural Hematoma Showed Normal Small Erythrocyte, No Sign of Malignancy Cells cm.

The patient was monitored in ICU ward, and packed red cells were administered. Screening for iron deficiency was negative, and dexamethason was given to trigger the cell lines demargination. Vitamin B12 was also given as supportive treatment.

His platelets count raised the next day, but the number was below 100,000. The other cell lines improved remarkably. He was transferred to the ward, and sent home after he was stabil for a few days. When he was discharge, hemoglobin of 12.0, hematocrit of 36, a white count of 6000 , and a platelet count of 60000.

The next two days, he visited out-patient department polyclinic and his laboratory values improved, such hemoglobin of 12 , hematocrit of 36, and white count of 10000, and a platelet count of 120000. He was alert, and complained no headache as before. However, he was not sent for a follow-up CT scan. The aetiology work up such as HIV, Hepatitis A, B or C, infection of bacteria was negative, still the cause of anemia aplastic was unknown.

Discussion

Our case discusses a conservative treatment of spontaneous subdural hematoma with accompanied intracranial hypertension in aplastic anemia patient.

Even there is no consensus regarding the treatment of spontaneous chronic subdural hematoma, many still choose burrhole procedures and drain placement as treatment options. The advantage of implementation of small tube to drainage has been shown in a randomized controlled trial and meta analyses.^{7-9,10} There are various of operation techniques ranging from twist-drill burrhole, and craniotomy.¹¹

Regarding the conservative treatment, the background theory was the administration of hyperosmolar substances would decrease the osmolarity and resulted in reduction of volume of hematoma.¹²⁻¹⁴ There was a case that reported resolved spontaneous subdural hematoma with tranexamic acid treatment.¹⁵ Tranexamic acid is a medication to minimize bleeding events in many types of surgeries, and its effectiveness has been

demonstrated. It was reported rarely in the management of chronic subdural hematoma.¹⁶

The literature of conservative treatment of chronic subdural hematoma was scarce and tranexamic acid had increased risk of thromboembolic events in patients with anti thrombotic or anticoagulant drugs.⁽¹⁷⁾ Some studies have disclosed that corticosteroids, angiotensin converting enzyme inhibitors, tranexamic acid may be co-treatment with surgery or even single treatment for CSDH.¹⁸⁻²⁰ However, the literature for conservative treatment was very rare.

Thrombocytopenia in aplastic anemia patients have presenting symptoms such as gingival bleeding, dermal petechiae, and echymoses. Bleeding that cause mortality is corticosteroid-gastritis related or fungal infection of the lungs.^{21,22} Intracranial presentation is the most feared because of its sequelae or disability.⁵

Formation of spontaneous SDH was linked to thrombocytopenia in hematological disorder patients. There were series of a conservative treatments of SDH that were found incidental during chemotherapy. Pomeranz et al. reported the incidence of subdural hematomas or intraparenchymal hemorrhages in 13/273 (4.7%) leukemia patients. SDH was not found in the aplastic anemia and beta-thalassemia cohort. It is of note that no leukemia patients suffered morbidity and mortality from SDH; however intraparenchymal hematoma has a different story with a 100% mortality rate.²³

Initiation of platelet infusion, along with packed red cells should be performed aggressively in the setting of thrombocytopenia of aplastic anemia. However, the consensus regarding the optimal value of the thrombocyte level target was still unclear. Many have targeted as of 50,000 μ L of thrombocytes. As our case has a good outcome with thrombocyte level 120K. This would be the plausible explanation of the recovery of the patient.

Conclusion

Thrombocytopenia, is a detrimental factor for the development of a spontaneous subdural hematoma. The choice whether conservative or operative treatment in SDH patients with low-level thrombocyte should be judiciously considered.

Disclosure

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