CASE REPORT

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Acquired prothrombin complex deficiency with hydrocephalus in a 43-day-old infant: A case report

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ABSTRACT

Background: Acquired prothrombin complex deficiency (APCD) is a spontaneous bleeding disorder caused by decreased activity of vitamin K-dependent coagulation factors that can be found in newborns up to six months of age. We would like to report a case of APCD with intracranial hemorrhage and its complication that have been found in Dr. Soetomo General Hospital, Surabaya, Indonesia. This study aimed to identify complications that can be found in patients with APCD.

Case presentation: A 43-day-old child came to the hospital with a chief complaint of fever 11 days before with a history of a general seizure lasting more than 15 minutes. The patient experienced bleeding in the injection site and got the treatment injection of vitamin K and a fresh frozen plasma (FFP) transfusion as the initial treatment of APCD. The physical examination showed the baby was compos mentis with a Glasgow Coma Scale (GCS) of 15 (E4V5M6). Laboratory examination showed hemoglobin levels of 8.7g/dL, prothrombin time (PT) of 13 seconds, activated partial thromboplastin time (APTT) was extended by 112 seconds, and platelet showed a normal value of 279 x 10^3µL. Head computed tomography (CT) scan examination showed bleeding and hydrocephalus. The patient was treated with non-operative and operative management.

Conclusion: Patients with APCD should be treated not only with non-surgical treatment but also with surgical treatment also.

Keywords: acquired prothrombin complex deficiency, case report, hydrocephalus, vitamin K deficiency.
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INTRODUCTION

Hemorrhagic disease of the newborn (HDN) is a bleeding disorder that occurs in newborns. The term HDN itself was first used in 1894 by Charles Townsend. He reported 50 neonates with cases of bleeding that occurred 2 – 3 days after birth.[1] The term vitamin K deficiency bleeding (VKDB) appears to be used in place of the term. Then the term acquired prothrombin complex deficiency (APCD) was first introduced by Bhancet in 1966.

Acquired prothrombin complex deficiency is a disorder manifested in the form of spontaneous bleeding caused by decreased activity of vitamin K-dependent coagulation factors (factors II, VII, IX, and X), while fibrinogen levels and platelet counts are still within normal limits.[2] In 1985, Lane and Hathaway classified APCD into three groups based on the onset of symptoms: early onset (occurring within the first 24 hours after birth), classic (occurring within 2 – 7 days), and late-onset (occurring between 2 – 12 weeks and up to 6 months of age).

Several etiologies are thought to be the etiology of APCD such as lack of intake containing vitamin K, history of medications consumption by the mother during pregnancy, especially drugs that can inhibit the activity of vitamin K (anti-epileptics, anti-tuberculosis drugs, and also vitamin K antagonists), and some of them are idiopathic. It is also associated with breastfeeding and it is said that the levels of vitamin K in breast milk (breast milk) are lower than in formula milk. In addition, prophylactic deficiency of vitamin K in children and mothers before birth and the presence of diseases with impaired absorption of vitamin K, for example, cystic fibrosis, biliary atresia, and liver disorders with cholestasis are also thought to be the etiology of this disorder.[3]

Epidemiological data of this case are not available yet in Indonesia. APCD appears with various manifestations, both in the form of gastrointestinal bleeding, ecchymosis, and even intracranial hemorrhage (ICH). This should be prevented by giving a prophylactic injection of vitamin K in a single dose of 1 mg intramuscularly to a newborn by the first hour of life after the mother initiates early breastfeeding or before getting hepatitis B immunization with an interval of 1 – 2 hours. However, these manifestations still appear after getting the injection and the complication can also appear.

CASE PRESENTATION

A 43-day-old child came to the hospital with a chief complaint of fever 11 days before with a history of a general seizure lasting more than 15 minutes. After the seizure, the patient cried. The patient experienced bleeding in the injection

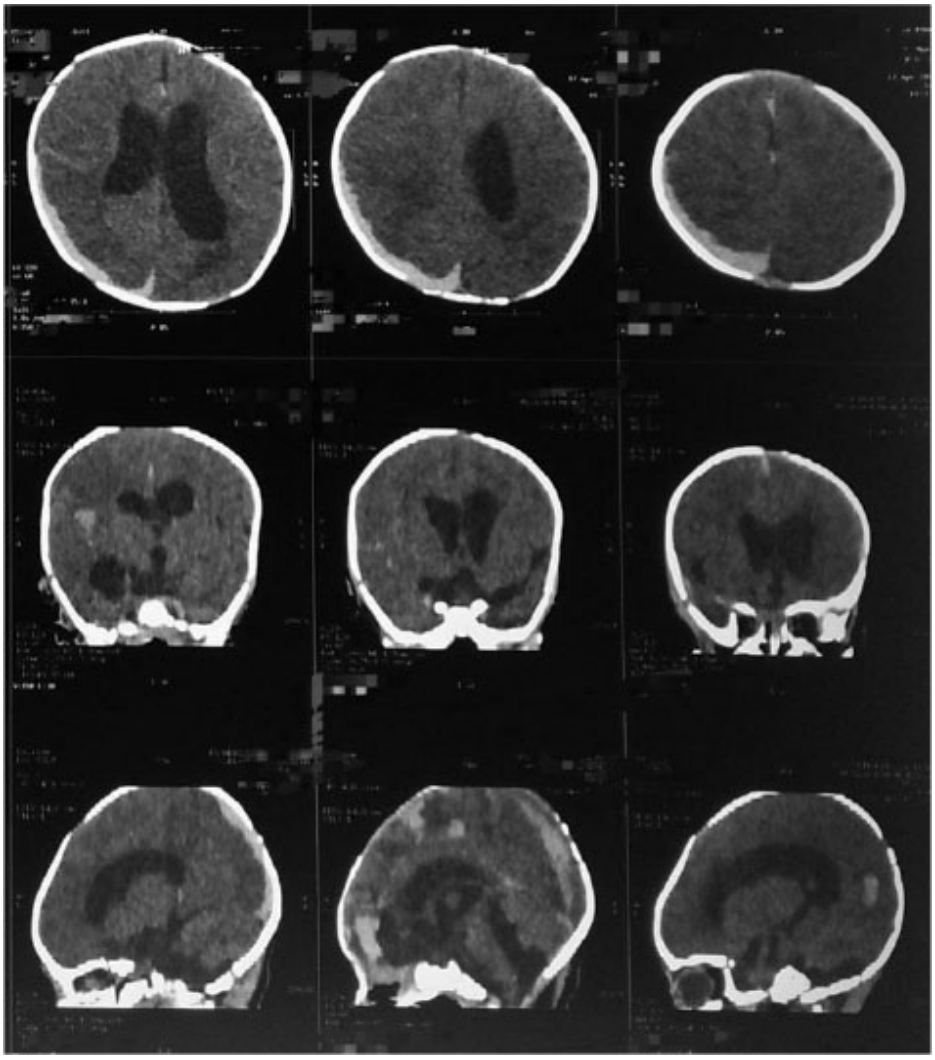

Figure 1. Head computed tomography scan examination.

site and got the treatment injection of vitamin K and a fresh frozen plasma (FFP) transfusion as the initial treatment of APCD. During pregnancy, there was no history of taking drugs that can inhibit the activity of vitamin K. The baby had a normal delivery with term pregnancy and was assisted by midwifery. After delivery, the baby was good without a history of jaundice, cyanosis, and head trauma. The patient was given breast milk and a prophylaxis vitamin K injection after delivery.

On physical examination, the baby was fully conscious with a Glasgow Coma Scale (GCS) of 15 (E4V5M6). Examination of vital signs showed the pulse rate was 134 beats per minute, an irregular respiratory rate of 32 breaths per minute, and a body temperature of 38°C. The weight was 3.5 kilograms and the birth weight was 3.0 kilograms. The head was normal with flat anterior fontanel with equal pupils of 3 mm. Physical examination of the chest was normal. The bowel sound was increased, which was related to a history of diarrhea.

Laboratory examination showed hemoglobin levels of 8.7g/dL, prothrombin time (PT) of 13 seconds, activated partial thromboplastin time (APTT) was extended by 112 seconds, and platelet showed a normal value of 279 x 10^3µL. These results indicated the patient was appropriate with the diagnosis of APCD. Head computed tomography (CT) scan examination showed bleeding and hydrocephalus (Fig. 1).

The patient was treated with non-operative and operative management. The patient was given a transfusion of a packed red cell (PRC) of 35 mL and fresh frozen plasma (FFP) of 35 mL for two days. This patient was also given an infusion of normal saline 350 cc per 24 hours, intravenous paracetamol 4 g every 6 hours, intravenous phenobarbital 6 g every 8 hours, and monitoring for vital signs.

DISCUSSION

In this case, the patient experienced bleeding at the injection site. This manifestation is an unusual finding in infants, but this occurrence was reported in India.[4] This finding served as an initial suspicion of APCD because the case that we reported was a 43-days old, and the age of the patient was still the onset age of APCD. The patient was given prophylaxis vitamin K after birth, this condition did not mean we could rule out the possibility to experience APCD. Also, this patient was only given breastfeeding, which had a higher risk of developing APCD. So, the oral intake of vitamin K must be given to fulfill the necessity of vitamin K.[5]

Based on Indonesia Pediatrician Association guidelines, APCD patients at least have minimal three clinical findings at initial assessment such as pallor, bulging fontanel, and seizure.[6] However, this patient only experienced seizure without pallor and bulging fontanel. Therefore, we did another examination to diagnose the patient with APCD by laboratory examination and head CT scan. Laboratory examination revealed prolonged PT and APTT which indicated the patient in severe vitamin K deficiency.[7] The head CT scan examination revealed intracranial hemorrhage (ICH) that localized at the subdural and subarachnoid, and also hydrocephalus as a common complication in patients with subarachnoid hemorrhage.[8] Hydrocephalus that have been found in this patient was communicating hydrocephalus. These findings related to flat anterior fontanel as the clinical manifestation in this patient.[9]

The patient had undergone four managements for hydrocephalus. The first operation performed was external ventricular drainage (EVD) to treat acute hydrocephalus. After the EVD was installed, the patient received surgery again because the EVD was removed during performing irrigation through endoscopy and re-insertion of the EVD at

the same point followed by an examination of cerebrospinal fluid (CSF) for culture analysis. CSF analysis showed sterile fluid. After three months of re-insertion of EVD, the patient comes back to the hospital with complaints of fever, seizure, and tachycardia without motoric disability. These conditions direct the patient to get another management called the ventriculoperitoneal shunt. After several months, the patient experienced vomiting and decreased consciousness which was suspected of having a shunt malfunction. Therefore, the patient received another management which was a revision of the shunt at the same point which was the last operation the patient had undergone.

CONCLUSION

Acquired prothrombin complex deficiency (APCD) is a spontaneous bleeding disorder caused by vitamin K deficiency with several clinical manifestations. Intracranial hemorrhage is one of the clinical manifestations of APCD. Not only injection prophylaxis of vitamin K to prevent bleeding, but good oral intake to fulfill the necessity of vitamin K. Management and treatment of APCD were adapted to the condition individually.

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CONFLICT OF INTEREST

There is no conflict of interest related to the materials or methods used in this study.

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AUTHOR CONTRIBUTION

APU provided the study design and contributed to collecting data, analyzing, interpreting, and participating in writing the manuscript. MAP provided the study design in the manuscript. MAP and SUYB provided input on the interpretation of the result.

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