A case report of Acquired Prothrombin Complex Deficiency in a 57 day-old infant

Aulia Putri Utomo[a], Muhammad Arifin Parenrengi[b], Siprianus Ugroseno Yudho Bintoro[c],

[a]Medical Program, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia
[b]Department of Neurosurgery, RSUD Dr. Soetomo, Surabaya, Indonesia
[c]Department of Internal Medicine, RSUD Dr. Soetomo,Surabaya, Indonesia

Abstract

Introduction: Acquired Prothrombin Complex Deficiency (APCD) is a disorder characterized by spontaneous bleeding caused by decreased activity of vitamin K-dependent coagulation factors (factors II, VII, IX, and X), despite normal fibrinogen and platelet counts**.** It can occur in newborns as young as 6 months old. This disease is linked to drug use during pregnancy, a lack of vitamin K-containing foods, and other secondary diseases. The manifestations of this disease vary, ranging from skin organs to gastrointestinal organs to the brain, with high morbidity and mortality rates. The aim of this case report is to describe an infant with diagnose of APCD and their management.

Case Presentation: A 55-day-old patient had been in a coma and had been unconscious for three days before being admitted to the hospital. The patient vomited, developed a fever, and had a seizure since four days before came to hospital. The laboratory examination showed that haemoglobin levels was 5.6g/dl, prothrombin time (PT) was 21 seconds, activated partial thromboplastin time (APTT) was extended by 54 seconds, and platelet showed abnormal 524 x 10^3µL in the result. The PT and APTT results indicated APCD, but there is an abnormality in platelets, which should be normal in APCD patient.

Conclusion: This is a case reported of Acquired Prothrombin Complex Deficiency (APCD) based on laboratory and CT-Scan examination.

Keywords: Acquired Prothrombin Complex Deficiency, Infant, Intracranial bleeding

1. Introduction

Hemorrhagic Disease of the Newborn (HDN) is a type of bleeding disorder that affects newborns. The term HDN was coined by Charles Townsend in 1894. He reported 50 neonates who experienced bleeding between 2-3 days after birth. The etiologies HDN are varies, such as due to vitamin K deficiency, clotting factor deficiency, trauma, and etc [1]. In its place, the term VKDB (vitamin K deficiency bleeding) appears to be used. Bhancet first coined the term Acquired Prothrombin Complex Deficiency (APCD) in 1966.

Acquired Prothrombin Complex Deficiency (APCD) is a disorder characterized by spontaneous bleeding caused by decreased activity of vitamin K-dependent coagulation factors (factors II, VII, IX, and X), despite normal fibrinogen and platelet counts [2]. Lane and Hathaway classified APCD into three groups based on the onset of symptoms in 1985: early onset (within the first 24 hours after birth), classic (within days 2 to 7), and late onset (occurring between 2-12 weeks and up to 6 months of age). There are numerous etiologies assumed to be the cause of APCD, including a lack of vitamin K consumption, the mother medication history during

pregnancy, particularly those that can inhibit the activity of vitamin K (anti-epileptics, anti-tuberculosis drugs, and even vitamin K antagonists), and some of them are idiopathic [3].

Unfortunately, epidemiological data on this incident isn't yet accessible in Indonesia. APCD can manifest as gastrointestinal bleeding, ecchymosis, or even intracranial hemorrhage (ICH). According to the guidelines, this can be avoided by giving the newborn a prophylactic injection of vitamin K in a single dose of 1 mg intramuscularly within the first hour of life after the mother begins early breastfeeding or before receiving Hepatitis B immunization (B0) with a 1-2 hours interval. However, these symptoms persist after the injection, and complications may occur [4].

2. Case

A 55-day-old patient had been in a coma in the hospital. Back before than the patient had been unconscious for three days before being admitted to the hospital without initial information of management. The patient vomited, developed a fever, and had a seizure since four days before came to hospital. However, there is no additional information about the seizure because this presentation didn’t record in medical record. During her pregnancy, the patient's mother visited the midwifery on a regular basis, with no history of illness or use of drugs and/or herbs. The delivery was a normal term pregnancy with midwifery assistance. The patient was in good condition after delivery and had no history of cyanosis or jaundice. There is no history of head trauma during growth and development. There is no history of taking injection of prophylaxis vitamin K after delivery. We had been asked by phone number but there is no answer from their parents.

The patient was found to be in a coma, with a Glasgow Coma Scale (GCS) of 4. (E1V1M2). The patient's head appeared abnormal, with a bulging fontanel and anisocoric pupils measuring 4 mm and 2 mm. The chest and abdomen were examined physically and found to be normal.

The laboratory examination showed that haemoglobin levels was 5.6g/dl, prothrombin time (PT) was 21 seconds, activated partial thromboplastin time (APTT) was extended by 54 seconds, and platelet showed abnormal 524 x 10^3µL in the result. The PT and APTT results indicated APCD, but there is an abnormality in platelets, which should be normal in APCD patient. Head computed topography (CT) Scan showed bleeding that localized subdural and subarachnoid, and also this patient showed hydrocephalus non-communicans.

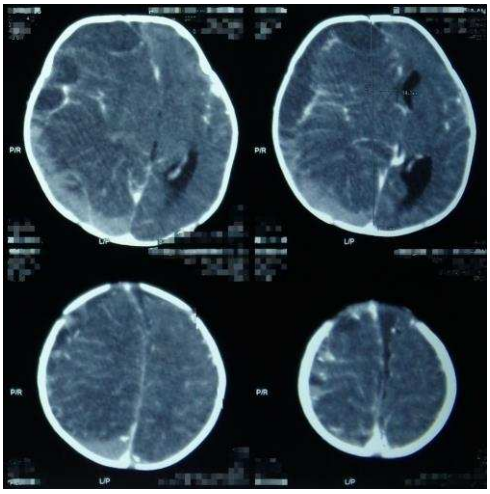

Figure 1 : CT-Scan Examination

3. Discussions

According to IDAI guidelines, APCD patients must have at least three clinical findings at the time of their initial assessment, including pallor, bulging fontanel, and seizure. All of these were discovered in this patient. So, we performed another examination to confirm the patient's diagnosis of APCD, which included a laboratory examination and a CT-scan. The PT and APTT times were both prolonged in the laboratory. The patient's CT-Scan examination revealed an intracranial hemorrhage (ICH) that was localized in the subdural and subarachnoid spaces. The CT-Scan also showed hydrocephalus. The hydrocephalus found in this patient is hydrocephalus non-communicans. In this patient, these findings were associated with a bulging fontanel as a clinical manifestation [5].

The patient in this case had an abnormal platelet count that was higher than normal, a condition known as thrombocytosis. The results for this patient also revealed leukocytosis. Physical examination also showed that the patient had a fever, tachypnea, and tachycardia. This claim was also supported by the use of ceftriaxone as an antibiotic.

The patient underwent craniotomy-assisted SDH evacuation and decompression. After the operation, the patient's Glasgow Coma Scale was 12 (E3V4M5), and he was also given ceftriaxone, metamizole, and continued to breastfeed.

4. Conclusion

Main clinical manifestation of APCD were pallor, seizure, and fontanel bulging. All those manifestation appeared in this patient. In patient’s result of CT-Scan examination is Intracranial Hemorrhage (ICH) that localized at subdural and subarachnoid, also hydrocephalus as common complication in patient with subarachnoid haemorrhage. But, this patient only had undergone one operative management called SDH evacuation and decompression used to reduce intracranial pressure and should be closely monitored after surgery. After the operation, the patient's Glasgow Coma Scale was 12 (E3V4M5), and he was also given ceftriaxone 2x100 mg via intravenous, metamizole 3x100 mg via intravenous to reduce pain for almost seven days and also continued to breastfeed.

Acknowledgments

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