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Original Article

Risk factors of mortality in children with acquired prothrombin complex deficiency at Dr. Zainoel Abidin General Hospital, Banda Aceh

Syifa Munawarah, Nora Sovira, Anidar Anidar, Heru Noviat Herdata, Eka Destianti Edward, Jufitriani Ismy

Abstract

Background Acquired prothrombin complex deficiency (APCD) is a rare but life-threatening bleeding disorder in children. Intracranial hemorrhage (ICH) is the leading cause of death, with an estimated risk affecting 50-80% of cases. Key risk factors associated with mortality in APCD include onset of disease, presence of ICH, and the initial Glasgow coma scale (GCS) score. Routine intramuscular administration of vitamin K at birth has been shown to effectively prevent early and late-onset vitamin K deficiency bleeding. However, in settings where vitamin K prophylaxis is not administered or is delayed, the risk of APCD increases significantly. Despite these concerns, other potentially relevant clinical factors contributing to APCD outcomes remain under-investigated.

Objective To identify risk factors associated with APCD mortality in children treated at Dr. Zainoel Abidin General Hospital, Banda Aceh.

Methods This cross sectional study analyzing children diagnosed with APCD at Dr. Zainoel Abidin General Hospital from October 2022 to October 2024. Data were collected from the medical records of 30 children and analyzed using Chi-square and logistic regression tests.

Results This study included 30 subjects, the majority of whom were male and aged 8 days to 6 months. Most of subject were born full term, delivered vaginally, and had birth weight ≥2.500 grams. Notably, 25/30 children did not receive vitamin K prophylaxis, 14/18 children were exclusively breastfed without vitamin K prophylaxis, and 25/30 children had good nutritional status. Late-onset APCD was observed in 14 out of 30 cases. Intracranial vs extracranial hemorrhage was occurred in 21 *vs.* 9 children. Initial GCS scores ≤ 8 at initiation of treatment were noted in 11/30 children. The mortality rate was occurred in 12/30 subjects (40%). Chi-square analysis revealed significant associations between increased mortality and late onset APCD (P=0.030), intracranial hemorrhage/ICH (P=0.049), and initial GCS score ≤ 8 (P=0.009). Logistic regression analysis revealed initial GCS score was associated with the highest risk of mortality in APCD, with a 16-fold increase in risk (OR 15.9; 95%CI 1.5 to 168.9; P=0.022).

Conclusion Intracranial hemorrhage, late-onset APCD, and initial GCS scores ≤ 8 are significantly associated with increased APCD mortality, with initial GCS emerging as the most influential risk factor. **[Paediatr Indones. 2025;65:253-9; DOI: https://doi.org/10.14238/pi65.3.2025.253-9].**

Keywords: *APCD; risk factors; mortality*

Acquired prothrombin complex deficiency (APCD) is a severe bleeding disorder that occurs in the early infantile period.[1] APCD is also known as vitamin K deficiency bleeding (VKDB) or hemorrhagic disease of newborns (HDN).[2-4] Since 1966, it has been reported as one of the most serious diseases in infants in North America, Europe, Australia, and Asia, causing high mortality and permanent neurologic sequelae.[1] The highest APCD cases were reported in Japan and Thailand, with an incidence rate of 35.5 per 1 million births in Thailand in 1966.[1,5] To date, the exact epidemiological

From the Department of Child Health, Dr. Zainoel Abidin Public Hospital Banda Aceh, Aceh, Indonesia

Corresponding author: Syifa Munawarah. Department of Child Health, Medical Faculty of Universitas Syiah Kuala. Jalan Teuku Tanoh Abee, kopelma Darussalam, Banda Aceh, 23111, Aceh, Indonesia. Email: Syifamunawarah@gmail.com.

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data of APCD cases in Indonesia is limited. However, a previous study reported 22 cases between 1997 and 2001 at Dr. Cipto Mangunkusomo Hospital, Jakarta.[6] Additionally, another study reported 32 infants with ICH at Dr. M. Djamil Hospital, Padang, West Sumatera, from January 2010 to 2013.[7] In 2017, a study reported two serial cases of APCD at Dr. Zainoel Abidin Aceh Public Hospital.[8]

The APCD often has a poor prognosis. While bleeding most commonly occurs in the brain, it can also involve the mucosa, skin, or gastrointestinal tract.6 High mortality in APCD cases is predominantly linked to ICH,[9] with the risk estimated at 50-80% of all cases.[4] Other factors contributing to high mortality rates are the Glasgow coma scale (GCS) score[10] and onset of disease.[11] Late-onset APCD is a bleeding disorder that typically occurs between the second week and six months of life. This condition is the most common cause of ICH and is strongly associated with decreased consciousness.[12]

In Indonesia, vitamin K prophylaxis has been implemented as a national standard of neonatal care, typically administered as a 1 mg intramuscular injection of vitamin K1 shortly after birth. This recommendation was established following a *Health Technology Assessment* (HTA) conducted in collaboration with the Ministry of Health in 2003, which also led to the development of a technical guideline to support its implementation. Since the introduction of this prophylaxis, the incidence of bleeding due to vitamin K deficiency has significantly decreased.[13] However, cases of late-onset APCD continue to be reported.[14]

A study demonstrated that intramuscular vitamin K prophylaxis significantly reduces the risk of late-onset APCD compared to no prophylaxis (RR 0.02; 95%CI 0.00 to 0.10).[15] Despite its inclusion in national policy, the coverage of this program has yet to reach full saturation, particularly in remote or underserved areas where access to trained health professionals and essential medicines remains limited. According to national health reports, gaps in implementation persist due to logistical, educational, and cultural challenges. These issues highlight the need for continued surveillance and evaluation of APCD cases.[14,16] Other reported risk factors for APCD include gender, prematurity, mode of delivery, nutritional status, breastfeeding practices, and maternal drug history.[17,18]

Vitamin K deficiency is a major factor in the pathogenesis of APCD, contributing to high morbidity and mortality. However, there is little data on the risk factors that may exacerbate mortality in APCD. Mangunatmadja *et al*.[6] examined the incidence, clinical manifestations, and outcomes of APCD. However, they did not investigate onset timing or the GCS and mortality in children with APCD. Currently, no studies in Indonesia have specifically addressed the relationships between mortality and onset of disease ICH or GCS value in children with APCD. Therefore, this study aimed to evaluate these critical risk factors in APCD patients.

Methods

This cross-sectional study was carried out from October 2022 to October 2024 at Dr. Zainoel Abidin General Hospital using medical records. Inclusion criteria were patients diagnosed with APCD, aged 0-6 months, available brain CT scan data, and laboratory results for PT and APTT.

Patients with incomplete medical records were excludes. Data related to vitamin K administration and patient outcomes were recorded for data collection. The sample was obtained by total sampling.

Vitamin K prophylaxis was defined as intramuscular administration of vitamin K1 within the first six hours of life, following Indonesian neonatal care standards.[14,16] Breastfeeding with vitamin K prophylaxis referred to cases in which infants were exclusively breastfed and also documented to have received intramuscular vitamin K prophylaxis.[15,19]

Early-onset APCD was defined as bleeding occur within the first 24 hours of life. Classic-onset APCD referred to bleeding occur between 2 to 7 days life. Late-onset APCD was defined as bleeding occur after the 7th day of life. Non-late-onset APCD comprised both early and classic onset categories.[20] Prolonged activated partial thromboplastin time was defined as an APTT value exceeding 37 seconds (normal range: 26-37 seconds).[21] Thrombocytopenia was defined as a platelet count $<150.000/\mu L$.[22]

This study was approved by the Health Research Ethics Committee of the Dr. Zainoel Abidin General Hospital, Banda Aceh. Bivariate analysis with Chi-

square test, and multivariate analysis with logistic regression were conducted using *IBM SPSS version 22* software. A significance level of $P < 0.05$ was applied.

Results

The subjects consisted of 30 children diagnosed with APCD from October 2022 to October 2024. Basic characteristics of subjects were presented in **Table 1** and **2**. **Table 1** shows that the majority of subjects were aged 8 days to 6 months, predominantly male and most of them had good nutrition. The majority of subjects had a birth weight of ≥ 2.500 grams, born at a gestational age ≥ 37 weeks, by vaginal delivery. Most subjects who had APCD did not receive vitamin K prophylaxis (25/30 children). Eighteen children were breastfed, of whom 4 of breastfed children received vitamin K, while 14 children of breastfed children did not.

Table 1. Baseline characteristics of children with APCD

Characteristics	(N=30)
Age at diagnosis, n	
0-24 hours	4
2-7 days	12
8 days-6 months	14
Gender, n	
Male	22
Female	8
Birth weight, n	
≥2.500 g	28
<2.500 g	2
Nutritional status, n	
Normal	25
Mild malnutrition	4
Severe malnutrition	1
Prematurity, n	
≥37 weeks	28
<37 weeks	2
Birth history, n	
Vaginal	25
Caesarean section	5
History of vitamin K prophylaxis, n	
Yes	5
No	25
Breastfeeding, n	
Breastfed	18
Breastfed with vitamin K	4
Breastfed without vitamin K	14
Not breastfed	12

Table 2 shows that 12/30 subjects were died (mortality rate 40%). Most subjects had a non-late onset APCD (16/30 children), while early onset was recorded in 4 children and classic onset in 12 children. Late-onset cases were noted in 14/30 children. Intracranial hemorrhage was observed in 21/30 children, which included intracerebral hemorrhage in 6 children, subdural hemorrhage (SDH) in 4 children, a concurrent intracerebral hemorrhage and SDH in 7 children, a concurrent SDH and subarachnoid hemorrhage (SAH) in 2 children, and a concurrent epidural hemorrhage (EDH) and intracerebral hemorrhage in 2 children. Extracranial hemorrhage was found in 9 children, including umbilical cord hemorrhage in 7 of breastfed children and gastrointestinal tract hemorrhage in 2

Table 2. Clinical and laboratory characteristics

Characteristics	(N=30)
APCD onset, n	
Non-late onset	16
Early onset	4
Classic onset	12
Late onset	14
Types of bleeding, n	
Intracranial hemorrhage	21
ICH only	6
SDH only	4
ICH + SDH	7
SDH + SAH	2
EDH+ICH	2
Extracranial hemorrhage	9
Umbilical cord hemorrhage	7
Gastrointestinal bleeding	2
Initial GCS upon treatment treatment, n	
>8	19
≤8	11
APTT score, n	
Normal	21
Elongated	9
PT score, n	
Normal	19
Prolonged	11
Platelets, n	
Normal	30
Low	0
Hemoglobin, n	
Hb ≥ 8 g/dL	16
Hb < 8 g/dL	14
APCD outcomes, n	
Survived	18
Died	12

of breastfed children. Based on the GCS score, most children had score > 8. Most of the APTT results were within the normal range (21/30 children), and prothrombin time (PT) values were also normal in 19 children. All subjects had platelet counts within the normal range, more than half of the subjects had Hb levels ≥ 8 g/dL. Finally, only 18 survived.

As shown in **Table 3**, most children with non-late-onset APCD survived, whereas most children with late-onset APCD died. Children with an initial GCS score of > 8 at the beginning of treatment predominantly survived, whereas those with an initial GCS score of ≤ 8 mostly died. Chi-square test revealed significant relationships between mortality and late onset (P=0.030), presence of ICH (P=0.049), and GCS score ≤ 8 (P=0.009). Children with late-onset APCD had a 7.8 times higher risk of death compared to those without late-onset APCD. Similarly, children with history of ICH were 8.8 times more likely to die compared to those without such a history. Furthermore, children with a GCS score of ≤ 8 had a 10 times higher risk of death compared to those with a GCS score of >8.

As shown in **Table 4**, multivariate analysis showed that initial GCS score at the time of treatment was a significant risk factor for mortality in children with APCD (P=0.022). Children with an initial GCS score of ≤ 8 had an approximately 16 times higher risk of death compared to those with a GCS score of > 8. Conversely, the onset of APCD and the presence of ICH did not retain significance as risk factors for mortality in children with APCD, as their P values exceeded 0.05 (P=0.444 and P=0.174, respectively).

Discussion

Bivariate analysis in our study revealed that most of our patients who died had late-onset APCD and an initial GCS score of ≤ 8. Intracranial hemorrhage was observed in 21 children, the ICH were manifested as intracerebral hemorraghe, SDH, SAH, EDH, either occuring in isolation or in combination. A previous study reported that ICH were located in the parenchymal (31.3%), subdural space (25%), extradural space (12.5%), and ventricles (12.5%), while 18.8% involved multiple sites. The most common ICH were multi-site and parenchymal hemorrhages, resulting from coagulation factor deficiencies affecting small blood vessels in brain tissue.[13] These findings aligned with another study which reported that among 50 APCD patients in Multan, Pakistan, 20% died, due to intracranial hemorrhage and late-onset diagnosis.[18]

A multivariate analysis identified GCS score of ≤ 8 as an independent risk factor for mortality.[23] In

Table 3. Analysis of possible risk factors for mortality in children with APCD

Variables	APCD outcomes		OR (95%CI)	P value
	Survived (n=18)	Died (n=12)		
Onset of APCD, n			7.8 (1.476 to 41.214)	0.030
Non-late onset	13	3		
Late onset	5	9		
Intracranial hemorrhage, n			8.8 (0.929 to 83.353)	0.049
Yes	8	1		
No	10	11		
Initial GCS at treatment, n			10 (1.781 to 56.150)	0.009
>8	15	4		
≤8	3	8		

Table 4. Risk factors that most influence mortality in children with APCD

Variables	Wald	SE	OR (95% CI)	P value
Initial GCS at treatment	5.276	1.205	15.923 (1.501 to 168.931)	0.022
Intracranial hemorrhage	1.852	1.693	10.014 (0.363 to 276.521)	0.174
Onset of APCD	0.585	1.166	2.440 (0.248 to 23.989)	0.444

our subjects more than one third had an initial GCS score of ≤ 8. The GCS score below 8 indicates severe brain injury, often caused by cerebral parenchymal hemorrhage, a critical complication of APCD. A lower GCS score signifies greater impairment of consciousness, typically associated with extensive brain damage due to bleeding in brain tissue. Larger ICH correspond to lower GCS scores and an increased risk of mortality.[4]

Most of our patients were male, consistent with findings in a study in India which observed a predominance of male APCD patients, though the underlying reasons for male predilection remain unclear.[24] Most of our subjects were delivered vaginally, while the remaining were delivered via cesarean section. A previous study noted that infants delivered at home often did not receive vitamin K prophylaxis.[18] Furthermore, most of our patients presented with non-late-onset APCD (early onset: 0-24 hours, and classic onset: 1-7 days). However, they reported that late-onset APCD, occurring between 2 weeks and 6 months of age, accounted for 72% of all APCD cases.[18]

Eighteen out of our thirty subjects were breastfed, of whom 14 did not receive a prophylactic vitamin K injection administered intramuscularly. These findings align with study which reported that among 30 APCD patients in India, 83.3% were breastfed.[19,25] Similarly, another study revealed that among 16 APCD patients with ICH, 18.7% received vitamin K prophylaxis at birth.[13] Newborns have immature gut flora and cannot produce sufficient amounts of vitamin K. Exclusively breastfed infants who do not receive vitamin K prophylaxis are particularly susceptible to vitamin K deficiency. Administering vitamin K prophylaxis at birth significantly reduces the risk of APCD.[19,25]

Bivariate analysis revealed a significant association between mortality, late-onset APCD and ICH (P=0.03 and 0.049, respectively). Late-onset APCD patients were found to have a 7.8 times greater risk of death compared to non-late-onset APCD patients. Similarly, patients with ICH had an 8.8 times greater risk of death that those without ICH. These findings were consistent with a study reported a high mortality rate in late-onset APCD patients, a condition often associated with severe and life-threatening ICH. [26]

We also found a significant relationship between initial GCS score at admission and mortality in APCD patients (P=0.009). Patients admitted with initial GCS score of ≤ 8 had a 10 times higher risk of death than those with GCS score > 8. This finding was in line with another study, who reported that GCS score of ≤ 8 significantly increased the risk of mortality.[23] Multivariate analysis further indicated that the initial GCS score at admission retained statistical significance as a risk factor for mortality in APCD. Patients with an initial GCS score of ≤ 8 were 15.9 times more likely to die compared to those with GCS score > 8. Severe damage to the ascending reticular activating system (ARAS) in the reticular formation can result in death. A study described four cases of vitamin K deficiency in Sandakan, Malaysia, where patients presented with bleeding and cord infections in three cases, and cord bleeding and decreased consciousness in one case. Despite treatment and intubation, the patient with decreased consciousness did not survive.[27] Another study reported that 46% of APCD patients who admitted with GCS score < 8, requiring intubation and vasoactive agent. The GCS score was a predictor of poor outcomes in this study.[28] All studies above emphasize that decreased consciousness is a critical determinant of poor prognosis in APCD.

Our finding suggest that absence of vitamin K prophylaxis remains a significant contributing factor to APCD in the population studied.[4] These results reaffirm the necessity of strengthening the implementation and monitoring of the national prophylaxis program, especially in peripheral and low-resource settings.[15,29] Moreover, the presence of coagulopathy despite prophylaxis in a few cases raises the possibility of underlying malabsorption syndromes or liver dysfunction, warranting further investigation.4 Enhancing clinician awareness and ensuring universal prophylaxis coverage may contribute to the reduction of APCD incidence and improve neonatal outcomes.[16,19]

The APCD is a rare disease. We acknowledge the limitation of our study regarding the number of variables analyzed. Due to relatively small sample size (n=30), we intentionally limited the number of variables to maintain statistical validity and reduce the risk of overfitting. We focused on the most clinically relevant variables based on previous literature and expert consensus. This approach was deemed

appropriate for an explanatory study with a limited sample. A more comprehensive investigation into additional variables, such as the length of treatment, severe anemia, and accompanying infections, would require a larger sample size. Furthermore, this study was conducted at a single hospital center, which may limit the generalizability of the findings.

Over a two-year period (October 2022-October 2024), 30 cases of APCD were recorded in children at Dr. Zainoel Abidin General Hospital, with a mortality rate of 40%. Mortality was significantly associated with GCS score ≤ 8 at the time of treatment initiation, while onset timing of APCD and ICH did not retain significance in multivariate analysis. Further study with a larger sample size is needed.

Conflict of interest

None declared.

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