# CARDIOGENIC PULMONARY EDEMA ET CAUSA HYPERTENSION ENTERING THE EMERGENCY UNIT UKI GENERAL HOSPITAL FROM JANUARY-NOVEMBER 2018

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**Abstract**

Acute pulmonary edema is one of the emergency conditions that require immediate management. Acute pulmonary edema occurs because of the underlying disease such as hypertension, coronary heart disease, and other heart diseases. From January 2018 to November 2018, there were 13,332 patients who came to the ED at the UKI Hospital, 99 patients with acute cardiogenic pulmonary edema. Of the 99 patients, there were 39% male sex data, 60% female, 36-45 years old age data 8%, 46-55 years 20%, 56-65 years 41%,≥ 66 years 30%, the cause of acute pulmonary edema due to hypertension (52%), coronary heart disease (42.6%), and other cardiovascular diseases (5.4%), mortality (8%).

Key Words: acute pulmonary edema, hypertension, coronary heart disesase

## 1. Introduction

Pulmonary edema is an accumulation of fluid in the lungs that occurs suddenly. This can be caused by high intravascular pressure (cardiac pulmonary edema) or due to increased capillary permeability (non-cardiac pulmonary edema) which results in rapid extravasation of fluid in the alveoli resulting in impaired air exchange and resulting in hypoxia. Pulmonary edema is an emergency with a high mortality rate. 1

Hypertension or an increase in blood pressure is a chronic disease and has always been a problem in public health in general, both in developed and developing countries.

Hypertension is also known as the "silent killer" which is often asymptomatic, but can damage several important organs such as the heart, brain, and kidneys. 1

Based on the body organs that are the target of damage from hypertension, uncontrolled hypertension can increase the risk of developing it. Dead. Statistics show that hypertensive complications cause an estimated 9.4 million deaths worldwide each year. 2

Based on data from Riskesdas in 2013, the prevalence of hypertension at age ≥18 years in Indonesia was 25.8%. In DKI Jakarta alone, the prevalence of hypertension based on a diagnosis of health personnel obtained by measuring blood pressure reached 20% . 3 According to a study in 1994, there were 74.4 million people with pulmonary edema in the world. In the UK about 2.1 million sufferers pulmonary edema which needs treatment and comprehensive surveillance. In the United States an estimated 5.5 million people suffer from pulmonary edema. In Germany about 6 million inhabitants. This is a large number that needs attention from nurses in caring for pulmonary edema clients in a comprehensive bio, psycho, social and spiritual way. 1

Pulmonary edema disease was first discovered in Indonesia in 1971. Since then the disease has spread to various regions, so that until 1980 all provinces in Indonesia. In Indonesia, the largest incidence occurred in 1998 with the incidence rate (IR) = 35.19 per 100,000 population and CFR = 2%. In 1999 IR decreased sharply by 10.17%, but in subsequent years IR tended to increase, namely 15.99 (2000), 19.24 (year 2002) and 23.87 (year 2003). Acute cardiogenic pulmonary edema is pulmonary edema that often occurs and has a detrimental and lethal impact with a mortality rate of 10-20% . 4

### 1.1 Formulation Problem

Based on the background of the problem above, the problem formulation of this study is what is the prevalence of sufferers of acute pulmonary edema in the Emergency Unit of the General Hospital of the Christian University of Indonesia in 2018.

### 1.2 Research Objectives

#### 1. General Purpose

To find out the prevalence of acute pulmonary edema in the Emergency Unit of the Indonesian Christian General Hospital in 2018.

#### 2. Special Purpose

a. This is to determine the prevalence of acute pulmonary edema

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b. This is to determine the risk factors in patients with acute

pulmonary edema such as cardiac (hypertension, coronary heart disease) and non-cardiac (chronic renal failure).

### 1.3 Benefits

#### 1 For the author

The results of this study can be used as additional information and knowledge about pulmonary edema and the authors note the importance of proper prevention and management in pulmonary edema patients.

#### 2 For Health Services

The results of this study can be used by all health care workers to realize the importance of prevention, early diagnosis and proper management of patients with pulmonary edema in order to prevent complications.

#### 3 For Education

The results of this study can be used as reference data for further research related to the prevalence and causes of acute pulmonary edema.

## 2. Literature Review

### 2.1 Definitions

Pulmonary edema is defined as a condition where there is movement of fluid from the pulmonary vascular to the interstitial and pulmonary alveoli. In pulmonary edema there is an accumulation of serous fluid or serosanguinosa in the interstitial space and alveoli of the lung. [1]

### 2.2 Classification and Etiology

Pulmonary edema can be classified as cardiogenic pulmonary edema and non-cardiogenic pulmonary edema. Cardiogenic pulmonary edema is caused by an increase in pulmonary capillary hydrostatic pressure which can occur due to excessive perfusion from both infusion, blood, and other blood products and fluids, while non-cardiogenic pulmonary edema is caused by increased pulmonary capillary permeability, among others, post lung transplantation and expansion of pulmonary edema. including ischemia-reperfusion-mediated injury.

Pulmonary edema is usually the result of increased pulmonary capillary vascular pressure and alveolar capillary permeability. Pulmonary edema due to increased lung cancer permeability is often called acute respiratory distress syndrome (ARDS).

Although the causes of cardiogenic and non-cardiogenic pulmonary edema are different, they both have a similar clinical appearance that makes diagnosis difficult. Appropriate therapy is needed to save patients from further damage due to fluid balance disorders in the lungs.

Approximately 90% of hypertension sufferers are classified as primary or essential hypertension when the cause of hypertension cannot be identified (idiopathic). Meanwhile the other 5-10% are classified

as secondary hypertension, which results from an underlying disease such as kidney disorders, use of drugs or hormonal disorders. [1,5]

| Examination<br>Anamnesis | Cardiogenik<br>Vascular event (+) | Non-Cardiogenik<br>Penyaki yang mendasari (+) |
|---|---|---|
| Physical Examination | | |
| ● Akral | Cold | Warm, strong pulse |
| ● S3 gallop/cardiomegali | (+) | (-) |
| ● Jugular venous pressure | Increase | Does not increase |
| ● ron Ronki | We[t] | Dry |
| Support | | |
| ● ekg ECG | Ischemia / infarction | Normal |
| ● chest x ray | Perihilar distribution | Usualy normal |
| ● Enzim kardiak | High | Normal |
| ● PCWP | Can increase | normal |
| ● Rasio PaO2/FiO2 | >18 mmHg | <18 mmHg |
| ● Hipoksemia | normal | normal |
| ● Edema and plasma protein ratio | <0,5 | >0,7 |

**Table 1. Difference between cardiogenic and non-cardiogenic pulmonary edema [1]**

The classification of hypertension based on JNC VII 2003 can be seen in the following table:

**Table 2. Classification of hypertension**[5]

| Classification | Systolic blood pressure (mmHg) | Diastolic blood pressure (mmHg) |
|---|---|---|
| Normal | < 120 | dan < 80 |
| Pre-hipertensi | 120 – 139 | atau 80 -89 |
| Hipertensi tingkat 1 | 140 –159 | atau 90 – 99 |
| Hipertensi tingkat 2 | ≥ 160 | atau ≥ 100 |
| Hipertensi tingkat 3 | ≥ 180 | atau ≥ 110 |
| Isolated systolic hypertension | ≥ 140 | dan <90 |

## 2.3 Pathophysiology

Under normal conditions in the lungs there is a continuous flow of intravascular fluid and protein to the interstitial tissue and back to the bloodstream system through the lymph channels which comply with Starling's law [6,7,8,9]

**Q = K (Pc-Pt) - d (c-t)**

Q : Flow of liquid

K : Constant

Pc : Hydrostatic pressure in capillaries

Pt : Hydrostatic pressure at interstitium

D : Reflection coefficient

C : Oncotic pressure in the capillaries

Q : Oncotic pressure in the interstitium

**Jv : LpS {(Pc – Pi) – αd ( πc – πi)}**

Jv : Fluid filtration rate (volume flow) across the microvascular barrier

Lp : Hydraulic conductivity (permeability)

S : Surface area of the barrier

Pc : Microvascular hydrostatic pressure

Pi : Peri microvascular hydrostatic pressure

πc : Microvascular plasma colloidosmotic/oncotic pressure

πi : Peri microvascular plasma colloidosmotic/oncotic pressure

αd : Average osmotic reflection coefficient of the barrier

Pulmonary edema occurs when more fluid is filtered by the microvascular walls than can be removed, which results in the alveoli being filled with fluid so that gas exchange is not possible. The determining factors that play a role here are differences in hydrostatic and oncotic pressures in the capillary and intersitic lumen, permeability of endolter cells to water, solutions, and the size of molecules such as plasma proteins. An imbalance of one or more of the above factors will cause pulmonary edema[7].

In cardiogenic pulmonary edema (volume overload edema) an increase in hydrostatic pressure in the pulmonary capillaries leads to an increase in transvascular filtration. When the interstitial pressure of the lung is greater than the intrapleural pressure, the fluid moves towards the visceral pleura which causes pleural effusions. If the endothelial capillary permeability remains normal, the edema fluid leaving the circulation has a low protein content. The increase in pulmonary capillary hydrostatic pressure is usually caused by increased pressure in the pulmonary veins that occurs as a result of increased left ventricular end-diastolic pressure and left atrial pressure (> 25mmHg). Under normal circumstances the pulmonary capillary pressure is around 8-12

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1. Pulmonary congestion causes desaturation and decreased myocardial oxygen supply, thus worsening heart function
2. Hypoxemia and increased fluid in the lungs cause pulmonary vasoconstriction, thereby increasing the right ventricular pressure, which through the ventricular interdependence mechanism will further reduce left ventricular function.

   12 mmHg insufficiency and a plasma colloid osmotic pressure of 28 mmHg. The incident will create a vicious cycle which continues to be worsened by the following processes: : [7,8]

3. Increased circulation causes acidosis, which worsens heart function.

   The discharge of edematous fluid from the pulmonary alveoli depends on active transport of Na + and Cl ions across the epithelial barrier contained in the apical membrane of alveolar type I and II epithelial cells and the distal airway epithelium. Na + ions are actively transported out into the interstitial space by the action of Na / K-ATPase which is located on the basolateral membrane of type II cells. Water passively follows, possibly through aquaporins, which are drains to type I cells. [7,8,9]

   There are three physiological levels of accumulation in cardiogenic pulmonary edema:

   1. Grade I: Fluid and colloids move from the pulmonary capillaries to the pulmonary interstitials but there is an increase in fluid out of the lymphatic flow
   2. Level II: The pumping capacity of the lymphatic system has been exceeded so that fluids and colloids begin to accumulate in

the interstitial spaces around the bronchioles, arterioles and venules.

3. Level III: Increased fluid accumulation causes alveolar edema. At this stage, the gas exchange interruption begins.

### 2.4 Clinical Features

The clinical picture of pulmonary edema is that from the history, it is found that there is a sudden shortness of breath associated with a history of chest pain and a history of heart disease. The development of ray edema can be gradual or sudden as in the case of acute pulmonary edema. In addition, large amounts of sputum are frothy and pink in color. Other common symptoms that may be found are: fatigue, quicker feeling of shortness of breath during normal activity (dyspnea on exertion), rapid breathing (tachypnea), dizziness, or weakness. Low levels of blood oxygenation (hypoxia) may be detected in patients with pulmonary edema. On auscultation can hear abnormal lung sounds, such as crackles or crakles. [1,10]

Supporting examinations are carried out to establish a diagnosis, namely:

1. Chest X-ray shows cardiomegaly (in patients with CHF) and alveolar edema with pleural effusions and bilateral infiltration in a butterfly pattern, pulmonary vascular and hilar cloudy features and the presence of interlobular Kerley B lines. Another feature associated with heart disease in the form of left ventricular dilation is common. Unilateral pleural effusions are also common and are associated with left heart failure.
2. ECG shows heart problems such as left atrial enlargement, left ventricular enlargement, arrhythmias, ischemic myocardial infarction or infarction.
3. Echocardiography is performed to determine whether there is a decrease in the function of the left ventricle and valve abnormalities- heart valve.
4. Laboratory tests of cardiac enzymes are necessary to help establish a diagnosis of myocardial infarction. Increased levels of brain natriuretic peptide (BNP) in the blood in response to increased pressure in the ventricles, BNP levels > 500 pg / ml can help diagnose cardiogenic pulmonary edema.
5. Blood gas analysis (AGDA) can showdecreased PO2 and PCO2 in the initial state but later in disease progression the PO2 decreased while PCO2 increased. In severe cases hypercapnia and respiratory acidosis are usually seen.
6. Right heart catheterization: Measurement of P pw (pulmonary capillary wedge pressure) through right heart catheterization is the gold standard for patients with cardiogenic pulmonary edema, which is around 25- 35 mmHg while in patients with ARDS P pw 0-18 mmHg.
7. Edema fluid protein content: Measurement ratio

   Edema fluid protein concentration versus plasma protein can be used to differentiate cardiogenic and non-cardiogenic pulmonary edema. The examination material is taken by suctioning pulmonary edema fluid through an endotracheal tube or bronchoscope and taking plasma. In cardiogenic pulmonary edema, the

protein concentration of edema fluid is relatively low compared to plasma (ratio <0.6). In non-cardiogenic pulmonary edema the edema fluid protein concentration is relatively higher (ratio> 0.7) because the nicrovascular barrier is reduced.

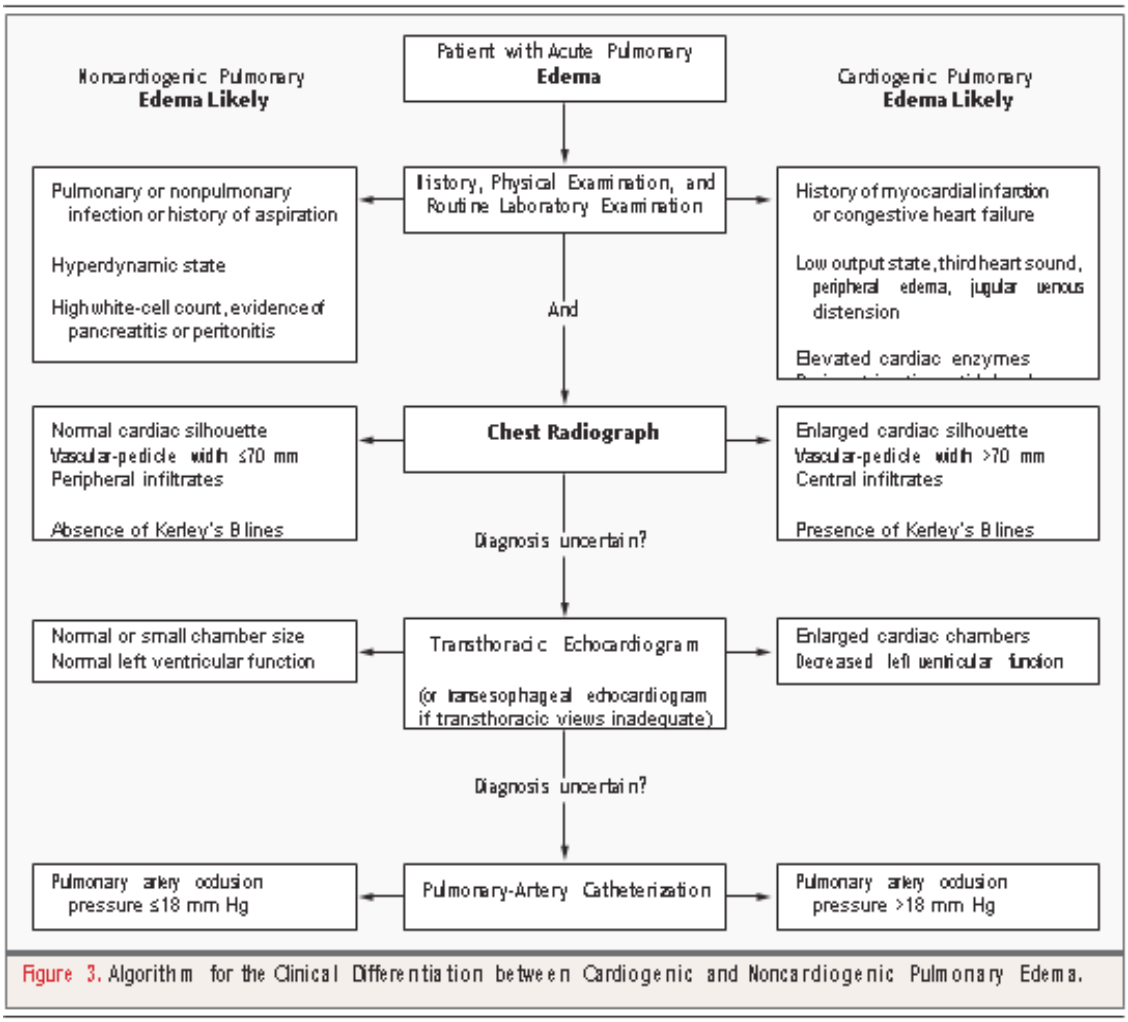


Figure 1. Algorithm of steps to differentiate between cardiogenic and non-cardiogenic pulmonary edema.
Source: Ware LB and Matthay MA, 2005

## 2.5 Management

Pulmonary edema is a medical emergency that needs to be treated as soon as possible after a diagnosis is made. Primary management includes supportive treatment aimed primarily at maintaining lung function (eg gas exchange, organ perfusion), while the underlying causes should also be investigated and treated as soon as possible. [10]

The principles of management include adequate oxygen administration, fluid restriction and maintaining cardiovascular function. Initial considerations are clinical evaluation, ECG, chest X-ray, and AGDA (blood gas analysis). [1]

### 2.5.1 Oxygen supplementation

Generalized hypoxemia in pulmonary edema is a major threat to the central nervous system, either in the form of decreased consciousness to coma or shock. Therefore oxygen supplementation is an important interventional therapy to increase gas exchange and decrease the work of breathing, optimize the lung functional unit as much as possible and reduce alveolar overdistention. [11]

In mild cases, oxygen can be given with a nasal cannula or a face mask. Continuous positive airway pressure (CPAP) is especially helpful in patients with cardiogenic pulmonary edema. Masip et al found that use of CPAP decreases the need for intubation and increases mortality.

In patients with acute cardiogenic pulmonary edema, induction of non-invasive ventilation in respiratory distress and metabolic disorders increases more rapidly than standard oxygen therapy but has no effect on short-term mortality. Noninvasive ventilation with CPAP has been shown to reduce endotracheal intubation and mortality in patients with cardiogenic acute pulmonary dedema. According to the study of Agarwal et al, noninvasive pressure support ventilation (NIPSV) appears to be safe and efficacious as CPAP, rather than working with titration at fixed pressure. [12]

The study of Winck et al. Supports the use of CPAP and non-invasive positive pressure ventilation (NPPV) in cardiogenic acute pulmonary edema. risk of acute myocardial infarction CPAP is considered to be the first intervention for NPPV that does not show better efficacy even in patients with more initial conditions, but is cheaper and easier to implement in clinical practice. Intubation and use of mechanical ventilation with positive end-expiratory pressure (PEEP) are required in severe cases. [11]

### 2.5.2 Medicines

a. Medicines that decrease preload

Nitroglycerin (NTG) can reduce preload effectively, quickly and the effect can be predicted. Intravenous administration of NTG is initiated with a low dose (20 µg / min) and then increased gradually (maximum dose 200 µg / min) . [1,11]

Loop diuretics (furosemide) can reduce preload via two mechanisms, namely diuresis and venodilation. The dose of furosemide can be given orally 20 - 40 mg / day in mild conditions to 5 - 40 mg / hour by infusion in severe conditions.

Morphine sulfate is used to reduce preload at a dose of 3 mg intravenously and can be given repeatedly. [11]

b. Medicines that are lower afterload

Angiotension - converting enzyme inhibitors (ACE inhibitors) reduce after load, as well as improve stroke volume and cardiac output. Administration of intravenous (Enalapril 1.25mg) or sublingual (Captopril 25mg) will improve patient complaints. In a meta-analysis it was found that administration of ACE inhibitors will reduce mortality. [1,11]

c. Inotropic drugs

Inotropic drugs are given to cardiogenic pulmonary edema with hypotension, namely dobutamine 2 - 20mcg / kg / minute or dopamine 3 - 20 mcg / kg / minute.

## 3. RESEARCH METHODOLOGY

### 3.1. Types of research

The research design used was descriptive method with cross sectional approach.

### 3.2. Location and Time of Research

The research was conducted in the medical records of the UKI Hospital from January 2018 to December 2018

### 3.3. Population and Sample

The population and sample in this study were all patients with acute pulmonary edema who came to the UGD at RSU UKI in 2018.

### 3.4. Method of collecting data

The data were obtained from the medical records of UKI Hospital with consecutive sampling technique

### 3.5. Operational definition

a. Acute pulmonary edema used according to ESC in 2012

b. Hypertension or increased blood pressure was used according to the JNC VIII criteria in 2013

c. Age is the length of time a patient lives until he comes to the UGD at RSU UKI which is expressed in years. If there is an excess of more than 6 months, it will be rounded up, if there are 6 months or less it will be rounded down

d. Gender is the gender of the patient, that is, male or female.

## 4. RESULTS AND DISCUSSION

### 4.1. General Characteristics

After conducting research through medical record data at UKI Hospital, 99 patients with acute cardiogenic pulmonary edema et causa hypertension came to the ER at UKI Hospital during the period

January - November 2018. Of the total patients, 39 were male (39%). ) while 61 women (60%). In this study, the prevalence of cardiogenic acute pulmonary edema et causa hypertension was found more in women. This is also in accordance with research from NHANES, that women at premenopause are at risk

The incidence is lower than that of men of the same age, but as they age women will have a higher percentage of hypertension than men.16 Several factors have been studied that the role of the hormones estrogen and progesterone can help prevent damage to blood vessels due to oxidative stress and inflammation, and has an effect on the smooth muscle contraction of blood vessels. Estrogen itself can also increase the sensitivity of the pressure relationship and natriuresis and increase sodium excretion [6,7]

Table 3.Distribution of patients with acute cardiogenic pulmonary edema et causa hypertension in the ER at UKI Hospital for the period January - November 2018 based on gender (N = 99)

| GENDER | Total (n) | Percentage(%) |
|---|---|---|
| Man | 38 | 39 |
| Woman | 61 | 60 |
| Total | 99 | 99 |

The results of the study showed that the highest prevalence of cardiogenic acute pulmonary edema et causa hypertension was at the age of 56-65 years as many as 41 people. (41%), while at the age 36 - 45 years as many as 8 people (8%), aged 46 - 55 years as many as 20 patients (20%), and age more than 66 years as many as 30 patients (30%).

Table 4. Age distribution of patients with acute cardiogenic pulmonary edema et causa hypertension ER UKI Hospital for the period January - November 2018 based on age classification (N = 99).

| AGE(YEAR) | TOTAL (n) | PERCENTAGE (%) |
|---|---|---|
| 36 – 45 | 8 | 8 |
| 46 – 55 | 20 | 20 |
| 56 – 65 | 41 | 41 |
| ≥ 66 | 30 | 30 |
| Total | 99 | 99 |

The results in this study also indicate that the occurrence of acute pulmonary edema is due to hypertension (52%), coronary heart disease (42.6%), and other cardiovascular diseases (5.4%).

Table 5. Distribution of causes of cardiogenic acute pulmonary edema in the ER at UKI Hospital for the period January - November 2018

| Cause | Total(n) | Persentage (%) |
|---|---|---|
| Hypertension | 51 | 52 |
| Coronary artery Disease | 42 | 42,6 |
| Other cardiovascular Disease | 6 | 5,4 |
| Total | 99 | 99 |

The results of other studies indicate that there is an increase in mortality in patients with cardiogenic acute pulmonary edema et causa hypertension in 8% or about 8 patients.

## Conclusions and Recommendations

Acute cardiogenic pulmonary edema is one of the most common, detrimental and fatal diseases with a mortality rate of 8%. This occurs due to increased hydrostatic pressure in the pulmonary capillaries which results in increased transvascular fluid filtration. Generally, from studies of patients with acute pulmonary edema found in men and women, but tends to be more in women. Patient age is more common in the age of 56 - 65 years. Cardigenic acute pulmonary edema in this study was mostly caused by hypertension.

This research is expected to be a material for consideration and input to improve the quality and quality of health services, especially in reducing the incidence of hypertension through preventive action. The need for early detection and public awareness of hypertension is also important in preventing complications from hypertension.

In this study there are still many shortcomings and limitations. Therefore, it is necessary to carry out further research, especially on the risk factors for hypertension related to proper prevention in hypertension control programs in the community.

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