Persistent ST Segment Elevation After Repeated Percutaneous Coronary Intervention: A Dressler Syndrome?

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ABSTRACT

In the era of percutaneous coronary intervention (PCI), Dressler syndrome has become an extremely rare phenomenon. Originally known as post-myocardial infarction syndrome, it is characterized by fever, pleuritic chest pain, and pericardial or pleural effusion after myocardial infarction. It is one of the sub-entities of post-myocardial infarction pericarditis (PMIP).

A 62-year-old man presented with persistent chest pain and diffuse ST segment elevation even after repeated PCIs. This condition was accompanied by fever and bilateral pleural effusion upon chest X-ray. The patient showed improvement in ST segment elevation and clinical condition after 2 weeks of steroid administration. The findings in this case suggest the possibility of PMIP. Although uncommon, physicians should be aware of the potentials of this condition in the differential diagnosis of chest pain after myocardial infarction and PCI so that immediate effective treatment can be given.

Keywords: *Case report, percutaneous coronary intervention, myocardial infarction, Dressler syndrome, post myocardial infarction pericarditis*

INTRODUCTION

Patients with acute myocardial infarction are recommended to undergo myocardial reperfusion, with primary percutaneous coronary intervention (PCI) as the preferred reperfusion strategy.[1] However, in some cases, patients who have undergone PCI can experience persistent chest pain and ST segment elevation in electrocardiograms (ECG). The possible mechanisms are early stent thrombosis, which in turn leads to reinfarction,[2] and injuries to the pericardium, such as acute pericarditis and Dressler syndrome.

Dressler syndrome was originally known as post-myocardial infarction syndrome, which can be followed with or without pericardial effusion.[3] It was first described in 1956 by William Dressler after he observed the late period development of an acute myocardial infarction.[4] Dressler syndrome usually develops 2–10 weeks after myocardial infarction and is believed to be caused by immune complex formation, which results in a systemic immune-inflammatory response.[5] Nowadays, it has become quite rare in developed countries due to advanced developments in myocardial infarction management, with less than 5% incidence.[6]

CASE ILLUSTRATION

A 62-year-old man presented to the emergency department with 11 hours of worsening back pain. The pain was described as a burning sensation that radiated to the chest and was not influenced by level of activity. The patient had a history of smoking and hypertension and denied any family history of cardiovascular or metabolic diseases.

Upon physical examination, the patient's blood pressure was 170/92 mmHg, heart rate 72 beats/min, respiration rate 20 breaths/min, temperature 36.5°C, and pulse oximetry 99% on room air. There was no sign of pulmonary edema or other problems. The results of the laboratory tests were leukocytosis (leucocyte 12.86 x 10^9/L), hemoglobin 15.8 g/dL, platelet count 280000/µL, potassium 3.7 mEq/L, urea 35.7 mmol/L, creatinine 1.1 mg/dL, and estimated glomerular filtration rate (eGFR) 71 mL/min/1.73m^2. Cardiac enzymes were within normal limits. A chest radiograph showed no abnormalities. The initial ECG from admission showed an inverted T wave on lead V2–V6. The patient was then diagnosed with unstable angina pectoris and admitted to the Intensive Coronary Care Unit (ICCU). Echocardiography showed segmental hypokinetic with ejection fraction (EF) at 46% and no pericardial effusion. A serial ECG was done 10 hours after the first ECG, and the results showed worsening conditions (**Figure 1**), so the patient was sent to have urgent percutaneous coronary intervention (PCI).

Coronary angiography revealed 70–80% diffuse stenosis in the proximal to distal left anterior descending artery (LAD); thus, two drug eluting stents were deployed in the proximal and distal LAD. Post-stenting angiography with LAD injection showed adequate flow in the proximal to distal LAD (**Figure 2**). However, we failed to notice that the first LAD diagonal branch (D1) showed no continuity of dye flow.

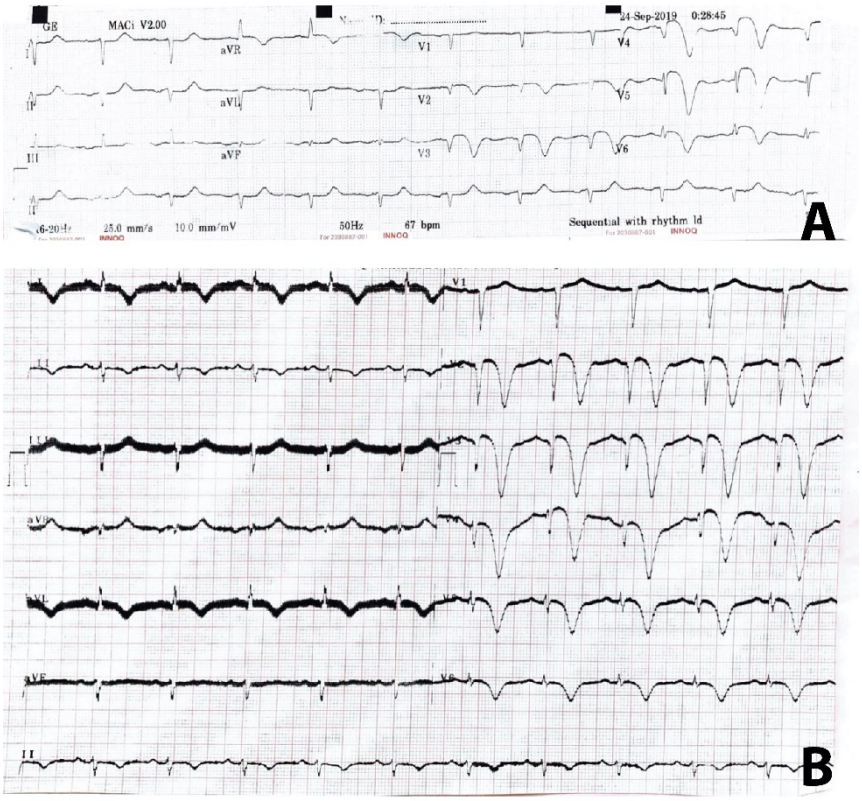

Figure 1. Twelve lead electrocardiogram reading of the patient before PCI. (A) Admission ECG demonstrating widespread inverted T wave on lead V2-V6. (B) serial ECG in ICCU 10 hours after admission showed deeper T inversion than before.

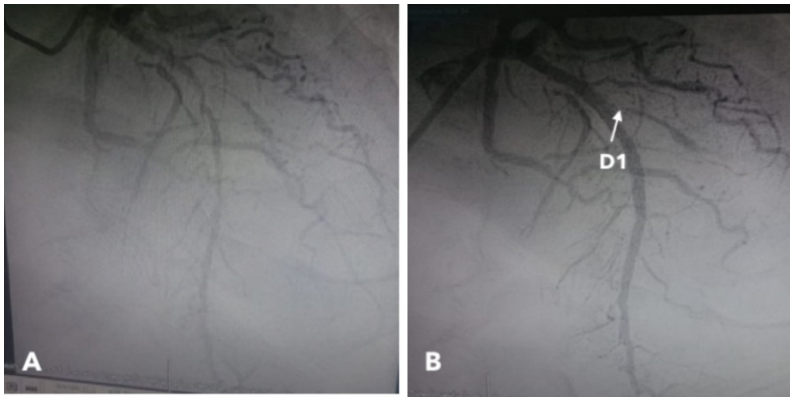

Figure 2. Coronary angiogram showing percutaneous coronary intervention of the LAD (A) pre-stent LAD showing 70-80% diffuse LAD disease (B) post-stent LAD showing adequate flow in most vessels with near occluded first diagonal branch of LAD. Notice that the first LAD diagonal branch (D1) still showed no continuity of dye flow.

Over the next 24 hours after the first PCI, the patient suddenly experienced chest pain that persisted even after nitroglycerin drips. The patient also began to develop a fever (38°C). Leukocyte count was increased to 25.4 x 10^9/L. A repeat echocardiography revealed a segmental akinetic and hypokinetic low systolic left ventricle, EF further reduced to 29%, and moderate pericardial effusion. Another ECG showed ST segment elevation in lead I, aVL, and V2–V6 (**Figure 3**), and a chest X-ray showed bilateral pleural effusion. As a result, the patient had an urgent PCI for the second time due to suspected acute stent thrombosis.

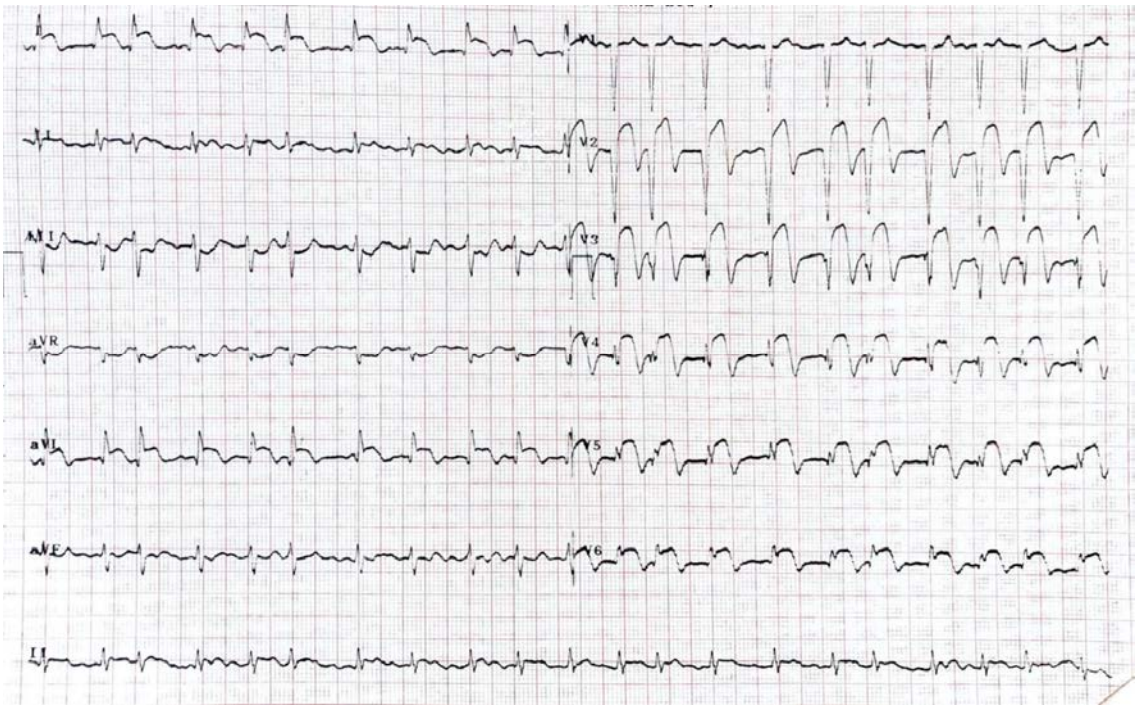

Figure 3. ECG taken one hour after first PCI showed diffuse ST segment elevation with T inversion in all leads.

The angiography results showed patency of both stents in the LAD, and we decided to open the occlusion of the D1 of the LAD with a drug-eluting stent in the osteo-proximal D1. The final angiogram post-stenting showed optimal results without any complications (**Figure 4**). But even after the second PCI was done, there was no significant improvement in the patient's clinical symptoms or ECG changes. The patient still complained of chest discomfort, epigastric pain, and breathing difficulty. The fever also persisted, and an ECG showed a worsened ST segment elevation in I, aVL, and V2–V6 **(Figure 5**).

After evaluating the patient's overall symptoms, Dressler syndrome was considered, and the patient was given ibuprofen 3 x 600 mg and a morphine drip 0.5 mg/hour. The next day, the patient's creatinine increased from 1.1 mg/dl to 4 mg/dl and urine production decreased to 0.4 ml/kg/hour, so ibuprofen was stopped and replaced with methylprednisolone 1 x 62.5 mg, and hemodialysis was performed on the patient. Furthermore, the patient felt subjective improvement, leukocytes returned to normal at 10.030 x 10^9/L, and the patient was finally discharged from the hospital 12 days later. An ECG showed improvement but a remaining, slight persistent ST segment elevation (**Figure 6**).

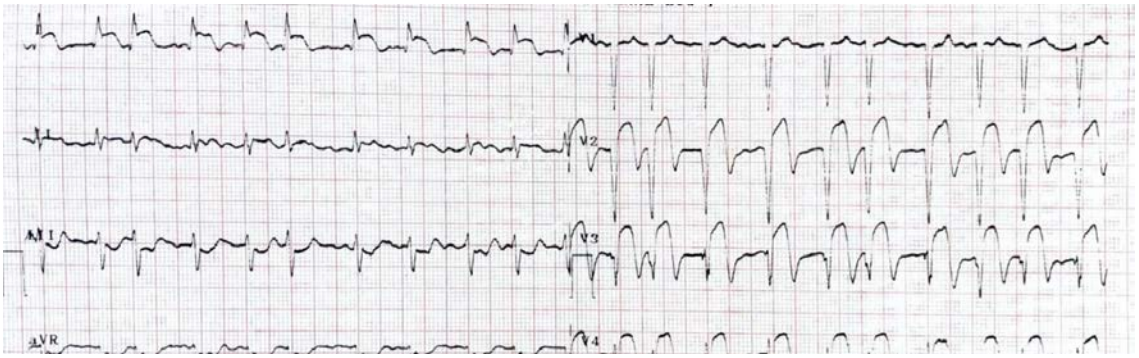

Figure 4. Coronary angiogram in second PCI. (A) Corangiography before stenting showed nearly occluded first diagonal branch (D1) of LAD. (B) View of the LAD after deployment of drug eluting stents in osteo-proximal first diagonal branch.

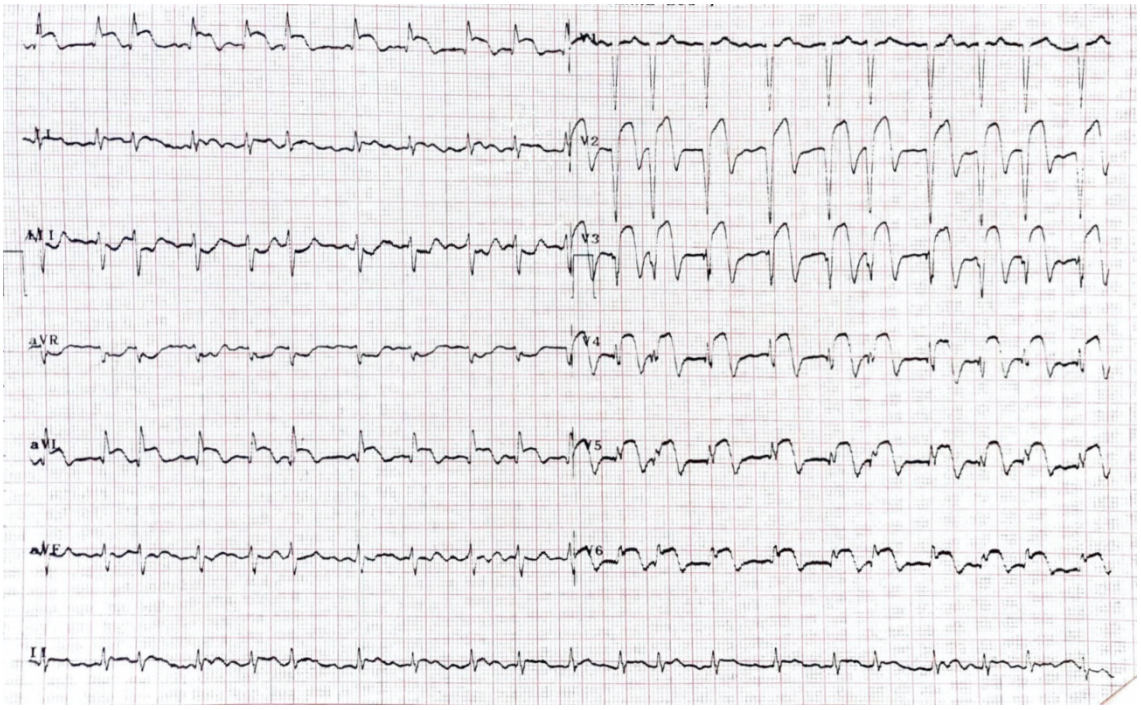

Figure 5. ECG taken after second PCI showed no improvement and persistent diffuse ST segment elevation in all leads.

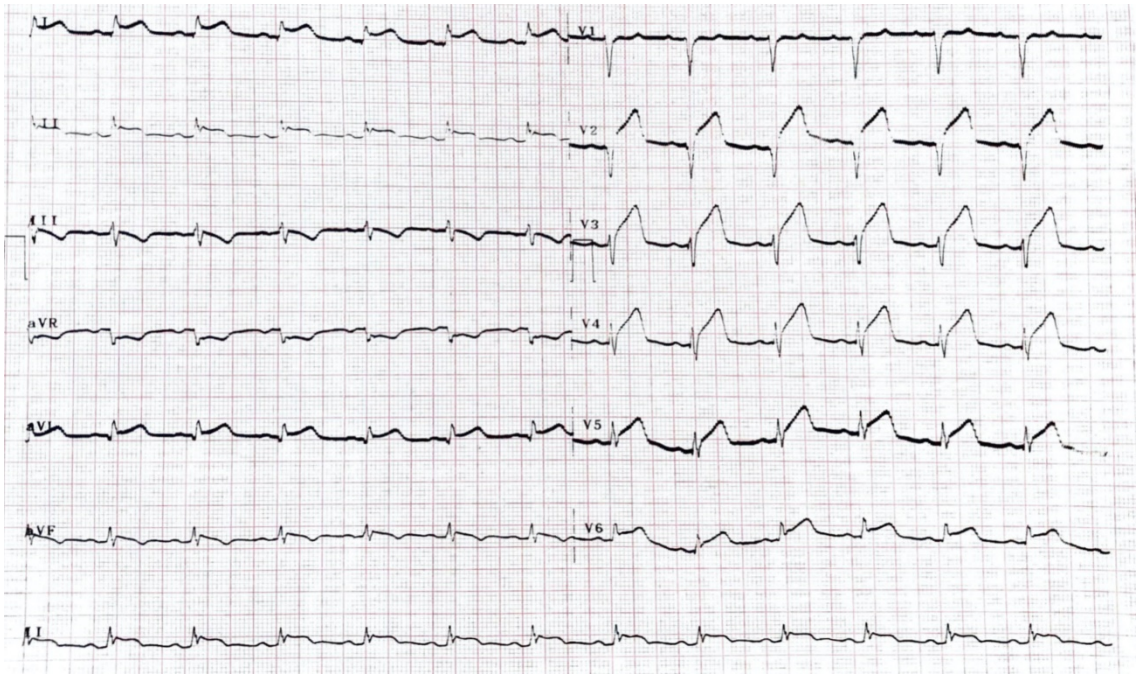

Figure 6. ECG taken 2 weeks after of steroid therapy showed marked improvement of ST segment elevation and diminished T inversion

DISCUSSION

Dressler syndrome is a form of secondary pericarditis that occurs as a result of heart or pericardium damage. It was first reported in 1956 as a benign triad of fever, pericarditis, and pericardial effusion post-myocardial infarct. Dressler syndrome should be suspected if the patient complains of prolonged weakness, especially after cardiac surgery or myocardial infarction.[7] The exact cause of Dressler syndrome is still unknown, but it is thought to be an immune-related event triggered by the initial damage to pericardial and/or pleural tissues as caused by myocardial necrosis, which in turn causes a systemic inflammatory response in the patient's body.[8] This condition is a sub-classification of post-cardiac injury syndrome (PCIS), which refers to a heterogeneous group of autoimmune-mediated conditions resulting from various injuries to the pericardial, epicardial, and myocardial.[9]

A PCIS diagnosis may be established if a patient presents with at least two of these clinical criteria after cardiac injury: (i) fever without alternative causes, (ii) pericarditis or pleuritic chest pain, (iii) pericardial or pleural rubs, (iv) evidence of pericardial effusion, and/or (v) pleural effusion with elevated C-Reactive Protein (CRP).[10] Therefore, patients with Dressler syndrome may experience complaints of fever, pleuritic chest pain, malaise, shortness of breath, palpitations, arthralgia, or irritability. Upon physical examination, tachycardia can be obtained with a friction rub upon auscultation, which can occur due to accumulated pericardial fluid. Pulsus paradoxus can also be found in the patient. In the thorax, pleural effusion can be found, though not always.[11]

On further investigations, the most sensitive diagnostic procedure for evaluating Dressler syndrome is echocardiography, which can reveal pericardial effusion, evaluated cardiac output, and contractility. If it is difficult to see posterior pericardial effusion or loculated-pericardial effusion, cardiac magnetic resonance imaging (MRI) can be used. Chest radiography can be done if echocardiography is not available. Flattened costophrenic angles can be found along with enlargement of the heart, which can indicate the presence of pleural and pericardial effusion. ECGs can have global ST segment elevations and T wave inversions, as with pericarditis. Low voltage QRS can also be used if pericardial effusion is high. Blood cultures in

Dressler syndrome show negative results. Other blood tests can reveal increased white blood cells with shift to the left and increased acute phase reactants (CRP).[4,5]

The management of Dressler syndrome is by administering anti-inflammatory drugs (NSAIDs) (e.g., ibuprofen, aspirin), tapered down in 4–6 weeks, while evaluating pericardial effusion. If the patient does not respond to NSAIDs or is contraindicated, corticosteroids (e.g., prednisone) can be given, which are reduced in 4 weeks.[12] Recurrence is possible, and colchicine can be given as prophylaxis.

In this current case, our patient was initially diagnosed with late onset angina pectoris (11 hours) that worsened over time, as proven by the deepening of the T inversion in the serial ECG, thus indicating an ongoing ischemic process of the myocardium. Coronary angiography showed diffuse stenosis of the LAD. After undergoing the first and second PCI successfully, the patient still experienced chest pain, accompanied by fever and evidence of pericardial and pleural effusion. His ECG showed persistent global ST segment elevations and T wave inversions. Laboratory examination also revealed leukocytosis. These findings are consistent with the clinical criteria of Dressler syndrome. After being given steroids, the patient showed clinical improvement. Leukocytes returned to normal and chest radiography showed remission of pleural effusion. The dramatic clinical improvement after the administration of corticosteroids strongly suggests an immune-mediated etiology in this case.

The clinical criteria and response to standard corticosteroid treatment strongly suggest a diagnosis of Dressler syndrome. However, according to 2015 European Society of Cardiology (ESC) guidelines, Dressler syndrome is defined as late post-myocardial infarction pericarditis (PMIP) with intervals of 2–8 weeks after infarction, while our patient's condition arose within the same week of the myocardial infarction event. Per the newest guidelines, PMIP can be grouped based on timing. Late infarct–associated pericarditis, known as Dressler syndrome, and early infarct–associated pericarditis typically occur less than 7 days post-myocardial infarct. The diagnostic criteria does not differ from acute pericarditis, which is the presence of two of four clinical criteria: (i) pericardial chest pain, (ii) pericardial rubs, (iii) ECG changes, and (iv) pericardial effusion. In early PMIP, also termed peri-infarction pericarditis, ECG changes may be overshadowed by changes due to myocardial infarction, but elevated ST segments may still occur.[10] Patients are usually asymptomatic, and the treatment is generally supportive, as most cases are self-limited. Still, for patients who have persistent symptoms that require more than supportive care, the choice of therapy remains the same as with other PCIS.[10,13]

While the timing of the pericarditis better matched early PMIP or acute pericarditis criteria, the clinical findings in this case are more suggestive of Dressler syndrome since PMIP or acute pericarditis do not usually present with severe symptoms, such as those in our patient. Both diagnoses have the same choice of therapy.

PMIP has been reported to be declining in incidence in recent years, which is why physicians nowadays sometimes overlook this disease when faced with patients with similar conditions and thus focus more on the possibility of myocardial infarction evolution. Thorough examination and understanding of the course of disease is required to diagnose this condition accurately. Differential diagnosis may include acute myocarditis, acute stent thrombosis, or reinfarction.

CONCLUSION

Our case supports the fact that any persistent ST segment elevation after a percutaneous coronary intervention should be thoroughly evaluated, as well as that the possibility of PMIP should be considered even in the early phase. Physicians must be aware of this entity and suspect it whenever they encounter patients with pericarditis-like symptoms after myocardial infarction, whether PCI was done or not. An immediate diagnosis is necessary to initiate proper treatment.

CONFLICT OF INTEREST

The author(s) declare that there is no conflict of interest.

AUTHOR CONTRIBUTION

TH and EG participated in manuscript ideation. TH wrote the draft. EG reviewed the draft, and finally approved it.

ETHICAL APPROVAL AND INFORMED CONSENT

Ethical approval is not required for case reports at our institution. Written informed consent was obtained from the patient for clinical and education purposes as per standard practice at our institution.

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