



# Severe Ischemic Stroke in a Patients with Idiopathic Thrombocytopenic Purpura: A Case Report

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## Abstract

In idiopathic thrombocytopenic purpura (ITP), a quite prevalent autoimmune hematological disease, thrombocytopenia is observed because of platelet destruction caused by immunological mechanisms. ITP usually leads to bleeding. Nevertheless, ITP, although rarely observed, may lead to thrombotic complications, including ischemic stroke. The present case is a female patient with ITP who developed cerebral infarction due to thrombus in the left middle cerebral artery. Our report reveals that severe ischemic stroke as well as bleeding may be encountered while monitoring an ITP case.

**Keywords:** Idiopathic thrombocytopenic purpura, stroke, thrombocytopenia, thrombus

## Introduction

Idiopathic thrombocytopenic purpura (ITP) is a quite prevalent autoimmune hematological disease. In ITP, antibodies bind to platelet-specific antigens, and antibody-bound platelets are removed from circulation by splenic macrophages. Consequently, they lead to thrombocytopenia and generally to bleeding diathesis (1,2). Nevertheless, ITP, even rare, may lead to thrombotic complications, including ischemic stroke (2,3). Herein, we reported a female case with ITP who developed cerebral infarction due to the thrombus in the middle cerebral artery (MCA).

## Case Report

A 55-year-old female patient was admitted to the emergency room with the complaints of weakness in the right upper and lower extremities, inability to speak, and somnolence. On

neurological examination, her conscious state was determined to be drowsy, articulation was totally aphasic, and she displayed right central facial asymmetry. The muscle strength revealed that right lower and upper extremities were totally plegic. She had 27 points on the National Institutes of Health Stroke Scale. With the suspicion of cerebrovascular syndrome, computed cranial tomography (CCT) was performed, and the result was unremarkable (Figure 1). Therefore, diffusion-weighted cranial magnetic resonance imaging was performed, and acute infarction was detected within the area supplied by the left MCA (Figure 2). On 3-dimensional time-of-flight image, left MCA was detected to be totally occluded (Figure 3). Results of posteroanterior lung graph and transcatheter and transesophageal echocardiography were unremarkable. Patient was admitted to the neurology intensive care unit (ICU). Because platelet count was found as 40,000 mm<sup>3</sup>

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on laboratory analyses, the patient was consulted with the hematology clinic. Her history revealed that the case had irregularly been followed up depending on ITP for three years. Protein C, protein S, antithrombin-3, and homocysteine levels were within the normal limits. Neither factor 5 leiden mutation nor methylene tetrahydrofolate reductase gene mutation were detected. Cholesterol level, thyroid function test, folic acid, B<sub>12</sub> level, fasting blood glucose, oral glucose tolerance test, and HbA1c levels

were normal. Vasculitic markers were found to be negative. The patient was administered with 1 mg/kg of methyl prednisolone. When thrombocyte level reached to 70,000 mm<sup>3</sup>, 150 mg of acetyl salicylic acid was commenced. For anti-edema, 20% mannitol was administered. In addition, the patient was administered with amlodipine at a dose of 5 mg/day because of mild arterial blood pressure elevation (arterial blood pressure, 140/90 mmHg). When her blood pressure was under control, CCT was performed on day 15 of her hospital stay, and a hypodense area was observed in the left frontotemporoparietal region (Figure 4). The patient was transferred from ICU to the in-patient clinic on day 15 of follow-up. The patient was consulted with the physical medicine and rehabilitation department, and exercise program was recommended. No remarkable improvement was observed on week 4 of treatment. The patient was informed regarding the purpose of the study, and she gave written consent to participate.

### Discussion

The present case is a female patient with ITP who developed cerebral infarction due to thrombus in the left MCA. Likewise, Mahawish *et al.* (4) reported a 79-year-old male case with weakness in the right side of the face and arm due to ITP. Rhee *et al.* (5) reported a 33-year-old male case with recurrent cerebral

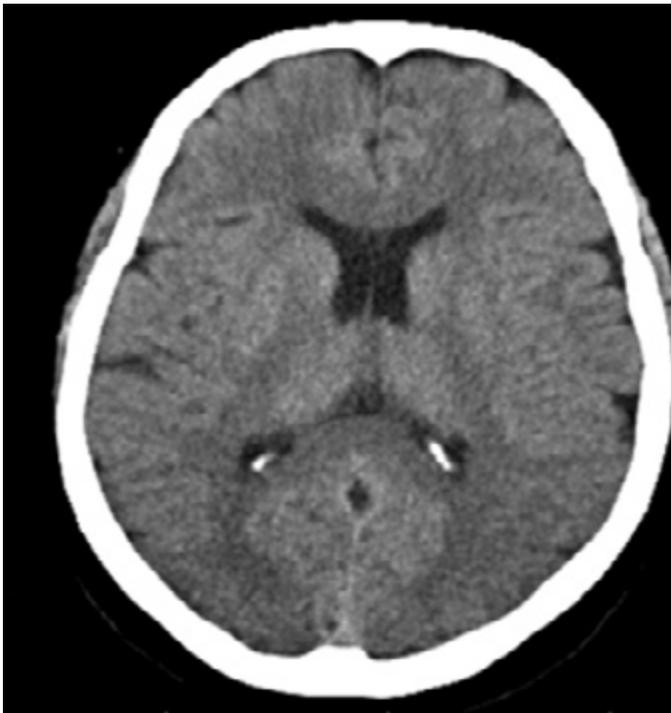


Figure 1. Cranial computer tomography: normal

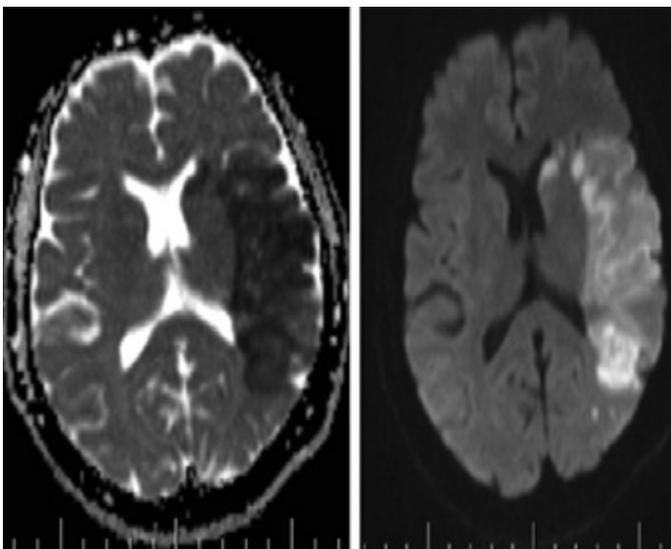


Figure 2. Diffusion magnetic resonance imaging and apparent diffusion coefficient imaging: restricted diffusion in the blood supply area of the left middle cerebral artery

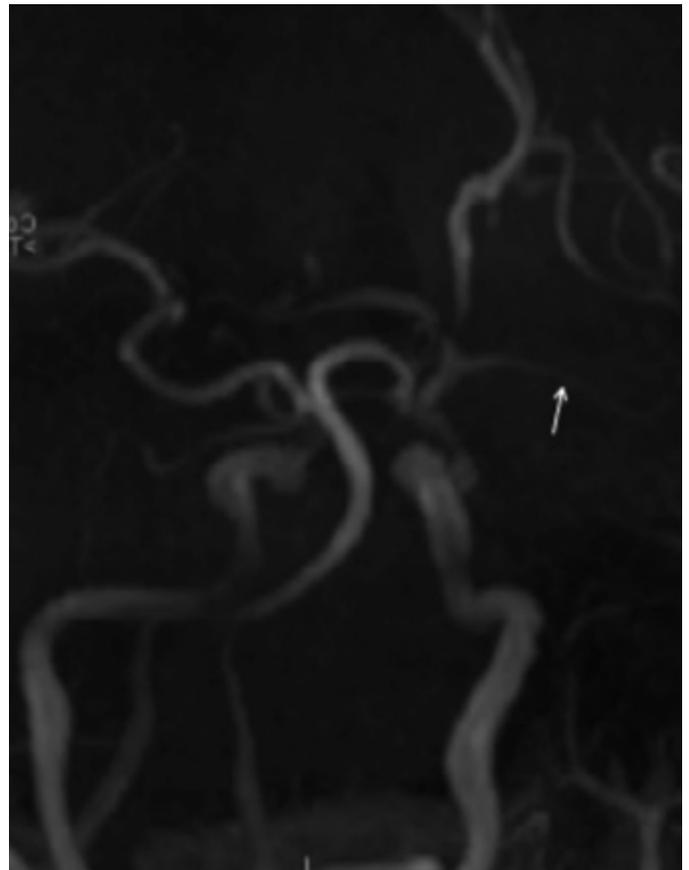


Figure 3. 3-D time-of-flight imaging: occluded image in the area supplied by the left MCA

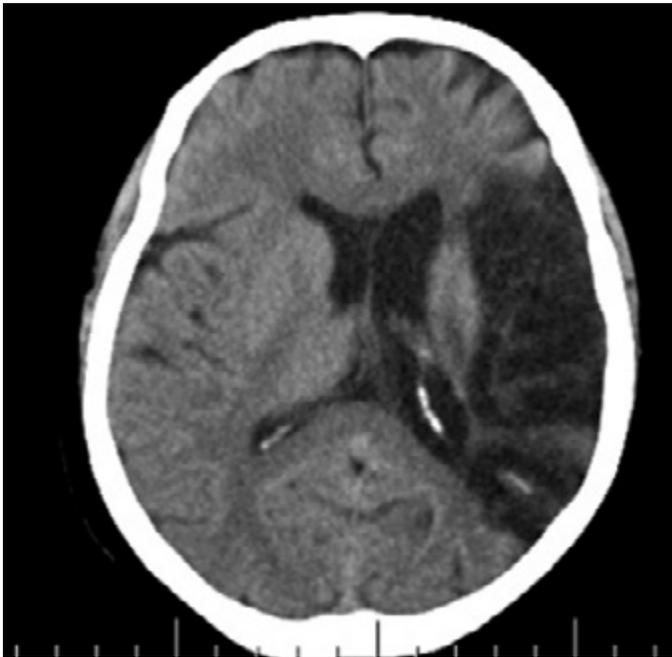


Figure 4. Cranial computer Tomography: hypodense area in the left frontotemporoparietal region

ischemic stroke stemming from ITP. Otsuki et al. (6) reported a female case with ITP and lupus anticoagulant with multiple ischemic infarctions. Theeler et al. (7) reported a 63-year-old male case with acute right occipital lobe infarction and acute left visual loss due to ITP.

In ITP, a quite prevalent autoimmune hematological disease, thrombocytopenia is observed because of platelet destruction caused by immunological mechanisms. Therefore, ITP usually leads to bleeding; however, although rare, it may cause thrombotic complications (5). Thrombotic complications in ITP may occur because of the disease itself after treatment modalities of ITP, such as splenectomy, IV immunoglobulin, danazol, and the association between ITP and other diseases such as antiphospholipid syndrome or thrombotic thrombocytopenic purpura (TTP) (5). Because the present case received no treatment for ITP or had neither antiphospholipid syndrome nor TTP, we considered that cerebral ischemic stroke resulting from MCA thrombus was caused by the disease itself. In this mechanism, humoral factors and platelet microparticles (PMPs) are released because of platelet destruction. PMPs induce thrombin activation and other coagulation factors (8). In ITP, circulating PMPs may enhance thrombotic events despite thrombocytopenia (7,8). Rhee et al. (5) reported that antibodies directly influencing the antigens on both platelets and endothelial cells induce endothelial damage, thrombus formation, and embolic occlusion of the artery.

Treatment of acute ischemic stroke developing on the basis of ITP still remains controversial (7). The present patient had previously received no regular treatment for ITP. We rapidly increased the platelet levels using steroid and subsequently provided required efficacy by antiplatelet treatment. Nanri et al.

(8) reported that prednisolone stopped migraine and returned the platelet count to the normal levels in a 31-year-old ITP case, who had concomitant ischemic cerebral infarction and migraine with visual aura. Consistent with the study by Nanri et al. (8), platelet count returned to normal levels in the present case with prednisolone. Moreover, we initiated 5 mg/day of amlodipine, a calcium channel blocker. In the literature, it has been reported that platelet fragmentation is a calcium-dependent process, and calcium channel blockers may hinder PMP release by inhibiting extracellular calcium influx (9,10). On admission, the patient's right lower and upper extremities were flaccid, and she was aphasic. No alteration was observed in the post-treatment clinical status of our patient. We attributed this to the poor prognosis of MCA thrombus.

## Conclusion

We consider that the treatment prevented the development of a new ischemic stroke attack and that the treatment of ischemic stroke in a patient with ITP should be individualized by taking the pathophysiological mechanism and hemorrhagic complications of stroke into consideration. Our report reveals that severe ischemic stroke and bleeding may be encountered while monitoring an ITP case.

**Informed Consent:** Written informed consent was obtained patient who participated in this case.

**Peer-review:** Externally peer-reviewed.

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**Conflict of Interest:** No conflict of interest was declared by the authors.

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