Oral Contraceptives as Predictor of Ischemic Stroke in a Young Primigravida: a Case Report From Khz Musthafa General Hospital, Tasikmalaya

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Abstract. Oral contraceptives (OC) are a recognized risk factor for stroke in young women. Combined oral contraceptives (COCs) containing estrogen and progesterone increase the risk of thrombus formation up to threefold compared to non-users, especially during the first year of use. Pregnancy also constitutes a risk factor due to physiological changes in hemodynamics, blood viscosity, connective tissue, and the immune system. The coexistence of both factors may further elevate the risk of cerebrovascular events. OC use may impair arterial elasticity and endothelial function, thereby predisposing to thrombosis. Pregnancy amplifies this risk through a procoagulant–anticoagulant imbalance, hemodynamic overload, and immune-mediated vascular changes that facilitate thrombus formation. The synergistic effect of these mechanisms likely contributed to ischemic stroke in this patient. Further research is needed, as this represents only a single case report. Stroke in young pregnant women may occur as a consequence of oral contraceptive use as a primary risk factor, although other vascular risk factors such as dyslipidemia and obesity cannot be excluded as potential contributors.

Keywords: *Ischemic stroke, Oral contraceptives, Pregnancy, Young women*

A. Introduction

Stroke in young women is an uncommon event, with an estimated incidence of 6–20 cases per 100,000 women under the age of 35.[1] Several unique risk factors contribute to stroke in this population, including oral contraceptive (OC) use[2], migraine with aura, preeclampsia, and pregnancy.[3] A previous study reported that women who use oral contraceptives have a threefold higher risk of venous thrombosis compared to non-users.[4] OC remains the most frequently used form of birth control in the United States, with approximately 25% of women aged 15–44 years relying on this method. According to national data in Indonesia (PK-23), the most common contraceptive method used nationwide is injection (35.4%), followed by pills (7.3%) and implants (6.2%). In West Java, injectable methods are used by 40.2% of women, followed by pills (8.9%) and intrauterine devices (6.9%).[5]

Most OCs contain a combination of estrogen and progesterone, which prevent ovulation by suppressing the release of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) from the pituitary gland.[6] In addition to their contraceptive effects, OCs provide protective benefits against certain conditions, including endometrial and ovarian cancers, dysmenorrhea, menorrhagia, iron deficiency anemia, ectopic pregnancy, pelvic inflammatory disease, and ovarian cysts.[6] However, OC use is also associated with adverse effects such as an increased risk of venous thromboembolism, stroke, breast and cervical cancers, and cardiovascular events—particularly among smokers.[7] There are two main OC types: combined estrogen–progestin pills and progesterone-only pills.[8] A cohort study by Lidegaard et al. demonstrated that combined oral contraceptive use significantly increases the risk of ischemic stroke.[9]

Pregnancy is one of the most important sex-specific risk factors for both ischemic and hemorrhagic stroke in young women, with an estimated incidence of 30 per 100,000 pregnancies.[10,11] The risk of stroke during pregnancy and the postpartum period is threefold higher compared to non-

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pregnant women of the same age.[12] Maternal stroke accounts for approximately 18% of all strokes in women aged 12–35 years, whereas only 1.4% of strokes in women aged 35–55 years are pregnancy-related.[12,13] Physiological changes during pregnancy—including alterations in blood viscosity, circulation, connective tissue structure, and immune responses—serve as adaptive mechanisms to support the gestation process.[13] Nonetheless, these changes may predispose to vascular complications. In young women, pregnancy-related stroke can also result from rarer mechanisms, such as cervical artery dissection,[14] cerebral venous thrombosis, and reversible cerebral vasoconstriction syndrome.[10,12]

B. Method

Case Report

A 27-year-old woman, G1P0A0, at 17 weeks and 5 days of gestation, presented with sudden onset of dysarthria, dysphagia, as well as weakness and numbness in all four extremities during activity, four days prior to admission. She had a history of nausea, vomiting, and vertigo for one month. The patient had been using COCs for two years, and rarely exercised. Physical examination, she was compos mentis, obese (BMI = 32), with vital signs within normal limits. Motor strength was upper : 2/5//2/5, lower : 1/5//1/5, and positive bilateral Babinski signs. Laboratory evaluation showed GDP 60 mg/dL, thrombocytosis (557,000/mm³), hypercholesterolemia (265 mg/dL), hypertriglyceridemia (254 mg/dL), elevated LDL (196 mg/dL), and normal potassium levels. ECG demonstrated sinus rhythm. Head CT Scan without contrast revealed hypodensity in the brainstem. Therapy: aspilet 1x80 mg, rosuvastatin 1x30mg, mecobalamin 1x500 mcg.

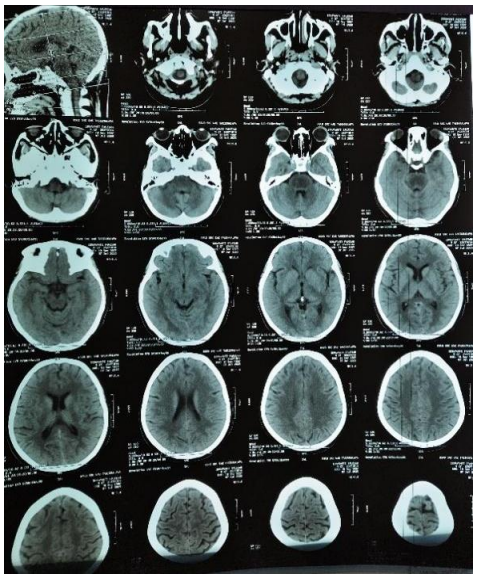

Figure 1. Head CT Scan without contrast showing a hypodense lesion in the brainstem (blue arrow).

C. Result and Discussion

Stroke is characterized as a cerebrovascular disease associated with high morbidity and mortality rates.[15] In general, stroke risk factors are classified into modifiable factors, such as hypertension, diabetes mellitus, dyslipidemia, cardiovascular disease, atrial fibrillation, sedentary lifestyle, obesity, smoking, and alcohol consumption, and non-modifiable factors, including age and sex.[15] In the United States, the incidence of stroke among individuals aged 20–44 years has increased from 17 per 100,000 in 1993 to 28 per 100,000 in 2015.[2] Additional factors specific to the younger population that may further increase stroke risk include migraine, use of oral contraceptives, pregnancy and the postpartum state, patent foramen ovale, and illicit drug use.[2]

The patient, a 27-year-old woman, based on the World Health Organization (WHO) 2015 classification, falls within the “young adult” age group (25–44 years).[16] She had a history of combined oral contraceptive (COC) use containing ethinyl estradiol and levonorgestrel. COCs are the most

commonly used type of oral contraceptive, composed of ethinyl estradiol and various forms of progestins.[6] According to Therese et al., during the first year of OC use, the incidence of all types of stroke increases; thereafter, the risk becomes comparable to that of non-users,[17] with a greater risk observed for ischemic stroke than for hemorrhagic stroke.[18,19] A large Danish cohort study involving 1,626,158 women reported that the absolute risk of thrombosis among OC users increased by 0.9–1.7 times for those taking preparations containing 20 μg ethinyl estradiol and by 1.3–2.3 times for those using 30–40 μg preparations.[2]

In this case, the patient had been taking a COC containing 30 μg ethinyl estradiol for two years. This finding supports previous evidence suggesting that using COCs for more than one year remains a predictor of stroke risk. Feng et al. demonstrated that each 10 μg increase in estrogen dosage in OCs was associated with a 20% increased risk of ischemic stroke, while each additional five years of use increased the risk by 24%. Thus, higher estrogen doses and longer duration of OC use are significantly associated with an elevated risk of ischemic stroke.[20]

Estrogen-containing OCs elevate plasma levels of coagulation factors II, VII, X, VIII, fibrinogen, and thrombin-activatable fibrinolysis inhibitor (TAFI). These changes promote thrombus formation and inhibit fibrin clot breakdown.[6] OCs also induce early vascular alterations through reduced flow-mediated dilation (FMD), indicating endothelial dysfunction, and increased pulse wave velocity (PWV), reflecting arterial stiffness and smooth muscle dysfunction, both of which contribute to cardiovascular disease (CVD) risk. A 1% decrease in FMD corresponds to a 13% increased risk of CVD, while a 1 m/s increase in PWV increases CVD mortality by 15%.[21]

Venous thromboembolism development in OC users is associated with increased blood coagulability, including alterations in coagulation factors, inhibitors, fibrinolytic activity, and platelet function, accompanied by structural changes in veins and arteries such as endothelial proliferation and altered venous flow. Conversely, arterial complications such as myocardial infarction and stroke are more complex, involving enhanced platelet activity, fibrin deposition, elevated arterial pressure, and changes in glucose tolerance and lipid metabolism.[6,22]

In this case, the patient was a primigravida in her second trimester who developed stroke. Stroke during pregnancy most commonly occurs from the third trimester to up to six weeks postpartum, coinciding with elevated rates of pregnancy-induced hypertension and gestational hypercoagulability.[13,23,24] The incidence of pregnancy-related stroke ranges from 3.8 to 34.2 per 100,000 deliveries, with the highest risk occurring during the early postpartum period.[13,25] Two major mechanisms contribute to this increased risk: (i) hypertensive disorders (isolated hypertension or preeclampsia/eclampsia),[26,27] and (ii) hormonal changes leading to generalized endotheliopathy that predisposes to stroke.[27] Ischemic stroke during pregnancy is less frequent than in the general population, where approximately 87% of cases are ischemic; among pregnant women, around 60% are hemorrhagic strokes, including intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH).[12,24,28] Thus, ischemic stroke occurring during the second trimester is considered rare.

Pregnancy is characterized by several physiological changes. A hypercoagulable state develops due to an imbalance between procoagulant and anticoagulant activity, accompanied by decreased fibrinolytic activity caused by elevated levels of plasminogen activator inhibitor type 2 (PAI-2) produced by the placenta.[4] Consequently, hypercoagulability during pregnancy can lead to cerebral venous thrombosis and venous infarction.[4] In this patient, platelet count was elevated (557,000/mm³), consistent with a hypercoagulable state during pregnancy. This condition typically begins around 11 weeks of gestation, with increased levels of coagulation factors VII, X, XII, fibrinogen, and PAI, along with decreased levels of anticoagulant proteins C and antithrombin, particularly during the third trimester and early puerperium.[4]

Other physiological changes include hemodynamic alterations such as increased cardiac output and blood volume starting from early pregnancy, as well as cardiac remodeling characterized by physiological ventricular hypertrophy.[13,29] Increased blood volume may impose pressure on vessel walls, and elevated cardiac output can exacerbate structural vascular changes, resulting in turbulent blood flow in major arteries and increasing the risk of thrombus formation and subsequent ischemic stroke.[30]

Pregnancy also causes significant immunological changes, involving both innate and adaptive (T-cell–mediated) immunity as part of gestational adaptation. However, in certain conditions, such as infection or systemic inflammation, excessive activation of proinflammatory pathways occurs,

particularly via T-helper 17 (Th17) cells, a subtype of $CD4^+$ T cells producing cytokines such as IL-17, IL-21, and IL-22. Elevated Th17 activity plays a key role in ischemic stroke pathogenesis in both the general and pregnant populations, contributing to endothelial dysfunction, increased blood–brain barrier (BBB) permeability, and activation of coagulation and local inflammatory cascades, thereby predisposing to cerebral thrombosis.[12,31] Primigravida women, due to incomplete hemodynamic and vascular adaptation, are at higher risk of complications compared with multigravida women who exhibit more optimized physiological adjustments.[32]

The patient also had dyslipidemia and obesity. Obesity is defined as a body mass index (BMI) >30 kg/m².[33] In Asian populations, lower BMI thresholds are used to better identify cardiometabolic risk: ≥23 kg/m² for overweight and ≥25 or ≥27.5 kg/m² for obesity.[34] A European study on ischemic stroke patients aged 18–55 years reported several modifiable traditional risk factors, including tobacco use (55%), physical inactivity (48.2%), hypertension (46.6%), dyslipidemia (34.9%), high-risk alcohol consumption (33.0%), and obesity (22.3%).[35] Thus, the patient's obesity served as an additional risk factor reinforcing the likelihood of ischemic stroke. According to Okoth et al., women with dyslipidemia who use COCs have an increased risk of myocardial infarction and cerebrovascular disease.[19] Each unit increase in BMI is independently associated with a 6% higher relative risk of stroke.[33] Mikkola et al. reported that women who were obese at age 14 had an 87% higher risk of thromboembolic stroke or transient ischemic attack (TIA) than those with normal weight.[36]

Additionally, the patient experienced dizziness, nausea, and vomiting for one month prior to stroke onset, suggesting possible vertebrobasilar insufficiency (VBI), defined as inadequate blood flow through the posterior cerebral circulation supplied by the two vertebral arteries forming the basilar artery. The vertebrobasilar system supplies blood to the brainstem, thalamus, hippocampus, cerebellum, and the occipital and medial temporal lobes.[37] The main risk factor for VBI is atherosclerosis,[38] with others including smoking, hypertension, age, sex, hyperlipidemia, obesity, family history, and genetic predisposition.[37]

Transient symptoms of VBI are highly variable, with vertigo being the most common clinical manifestation of posterior circulation ischemia. Other symptoms include hearing and visual disturbances, ataxia, dysphagia, hemiparesis, and cardiorespiratory abnormalities.[37] Grad and Baloh described posterior circulation ischemia as presenting in two forms: transient VBI episodes and established infarctions, with VBI potentially progressing to brainstem infarction.[38]

The patient experienced an ischemic stroke located in the brainstem. Ischemic strokes occur more frequently in the brainstem than hemorrhagic ones.[39,40] The brainstem, comprising the midbrain, pons, and medulla oblongata, lies in the posterior part of the brain and connects the cerebrum, cerebellum, and spinal cord.[39] The most common causes are atherosclerosis of the perforating arteries and basilar artery occlusion.[39] Clinical features of brainstem stroke include motor and sensory deficits such as hemiparesis or quadriparesis, sensory loss in the face or limbs, cranial nerve involvement leading to dysphagia, dysarthria, facial weakness or asymmetry, diplopia, and ocular movement abnormalities. Brainstem strokes can also affect autonomic functions, leading to cardiac rhythm disturbances, blood pressure instability, and even respiratory compromise or apnea. In many cases, patients present with coordination disorders such as vertigo and ataxia, and in severe cases, reduced consciousness or coma may occur.[41]

The pons is the most common site of brainstem infarction, involved in approximately 60% of ischemic brainstem strokes.[41] Pontine ischemia occurs within the extensive posterior circulation and can also affect younger individuals with underlying vascular abnormalities such as CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy), hypercoagulable states, or vertebral artery dissection.[42] The patient's sudden onset of dysarthria and dysphagia corresponds to multiple pontine infarctions affecting the perforating arteries, leading to pseudobulbar palsy characterized by severe dysphagia and dysarthria. Some patients may also exhibit pathological crying or laughter.[42] In addition, the patient experienced sudden weakness and numbness in all four extremities, consistent with quadriparesis.

D. Conclusion

Stroke in young pregnant women may occur as a consequence of oral contraceptive use as a primary risk factor, although other vascular risk factors such as dyslipidemia and obesity cannot be excluded as potential contributors.

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