Intracerebral Hemorrhage Associated with Varicella Zoster-Virus Infection: Case Report

Varisella Zoster Virüs Enfeksiyonuna Eşlik Eden İntraserebral Kanama

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ABSTRACT Varicella-zoster virus (VZV) infection causing ischemic stroke, especially in children, is a known entitiy, but its association with an intracerebral hemorrhage is extremely rare. Herewith we describe an immuncompetent 30-year-old female patient with right sided-hemiplegia and aphasia. She had had vesicular exanthema due to varicella zoster infection on her left shoulder extending to C4 dermatoma four weeks before her admission. However we could found no lesion in our examination. The initial brain computerized tomography showed left frontotemporal hematoma. Plasma white blood cell count; serum HSV type I IgG, VZV IgG and IgM levels were elevated. Other stroke risc factors were absent. Anti-edema treatment was administered. One month later her neurological examination improved except motor dysphasia. VZV infection causes focal fibrinoid necrosis and disruption of the medial elastica of the cerebral arteries. These changes disrupt the integrity of the vessel wall and give rise to the intracerebral hemorrhage. Therefore, patients recovered from varicella-zoster infection should be followed for a few months.

Key Words: Cerebral hemorrhage; stroke; vasculitis

ÖZET Varisella-zoster virüs (VZV) enfeksiyonunun özellikle çocuklarda iskemik inmeye yol açtığı bilinmektedir, ancak intraserebral hematom ile birlikteliği oldukça nadirdir. Biz burada sağ hemipleji ve afazisi olan immün sistemi sağlam 30 yaşında kadın olguyu bildiriyoruz. Olguda bize başvurmadan 4 hafta önce sol omuzundan C4 dermatomuna uzanan alanda VZV enfeksiyonun yol açtığı veziküler egzantem mevcutmuş. Ancak biz muayenemizde herhangi bir lezyon bulamadık. İlk beyin bilgisayarlı tomografisi sol frontotemporal hematom gösterdi. Serum beyaz küre, serum HSV tip I IgG, VZV IgG ve IgM değerlerinde yükseklik saptandı. Diğer inme risk faktörleri mevcut değildi. Antiödem tedavisi uygulandı. Bir ay sonra, hastanın nörolojik muayenesi motor afazi dışında düzelmişti. VZV enfeksiyonu serebral arterlerin medial elastika tabakasında bozulmaya ve fibrinoid nekroza neden olur. Bu değişiklikler damar duvar bütünlüğünü bozar ve intraserebral hemorajiye yol açar. Bu nedenle VZV enfeksiyonundan iyileşen hastalar bir kaç ay izlenmelidir.

Anahtar Kelimeler: Serebral hemoraji; inme, felç; vaskülit

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Central nervous system complications due to varicelle-zoster virus (VZV) infection, especially in children, is a well known etiology with an incidance of 0.1-0.75%.[1] VZV affects the major cerebral arteries and damages the vascular endothelium. This damage leads mostly to ischemic syndromes.[2,3] However, according to our knowledge, intracerebral hemorrhage after VZV infection in an adult has been reported in only a few patient.[4-7] Herewith, we report an young adult intracerebral hematoma following VZV infection.

TABLE 1: Laboratory findings of the patient.

Laboratory tests	VZV IgM	VZV IgG	HSV IgM	HSV IgG	aPTT	PT	Blooding time	White blood cell
Patient values	18	6062	negative	positive	21.9	11	1 minute	18900
Normal values	0-10 U/ML	0-50 U/ML	negative	negative	25-36 second	11-15.5 second	1-3 minute	4500-11000/mm^3

CASE REPORT

A 30-year-old female suffering from acute onset of headache, drowsiness, speech disturbance, and weakness of right arm and leg presented to our emergency department. She had had vesicular exanthema due to varicella zoster infection on her left shoulder extending to C4 dermatoma four weeks before her admission. Her vital signs were as follows: respiratory rate, 22/minute; pulse rate, 74/minute; blood pressure, 110/70 mm Hg; and temperature, 37°C. She experienced no pregnancy and she has also no systemic illnesses such as hypertension and diabetes mellitus. Her neurologic examination revealed confusion, global aphasia, and right hemiplegia. Laboratory findings were consist of, elevated white blood cell count; serum HSV type I IgG, VZV IgG and IgM (Table 1). VZV IgM values decreased three weeks after her admission. Other stroke risc factors such as coagulapathy, vasculitis, abnormal immunological and infectious parameters were absent. The initial brain computerized tomography (CT) and magnetic resonance imaging (MRI) showed left frontotemporal hematoma of 65 x 35 mm and 0.5 cm midline shift (Figure 1a,b). Magnetic resonance angiography (MRA) and conventional angiography revealed no abnormality. Anti-edema treatment (mannitol and dexametasone) was administered in order to decrease the intracranial pressure and reduce the midline shift. Our patient responded well to the therapy during her follow-up in the intensive care unit. Her neurological examination improved except motor dysphasia. Her control CT of the brain on the sixth month revealed encephalomalasia of the frontotemporal area due.

Informed consent had been obtained.

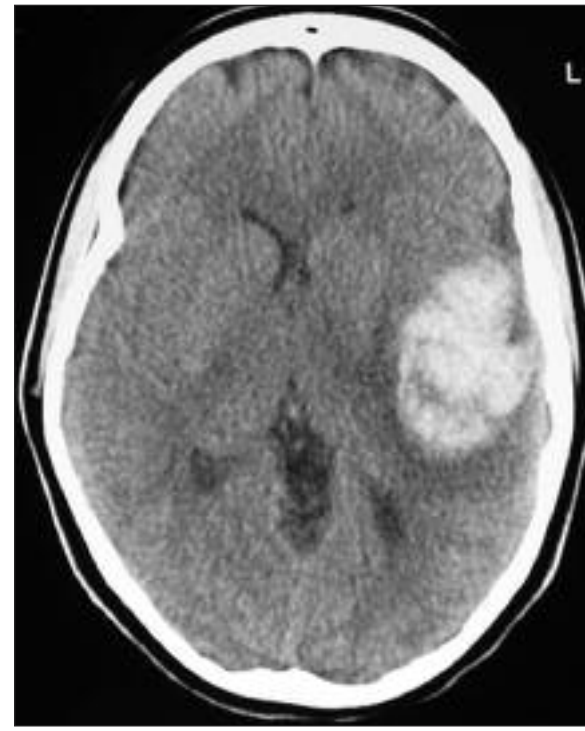

FIGURE 1a: Axial brain CT show left temporoparietal intracerebral hemorrhage.

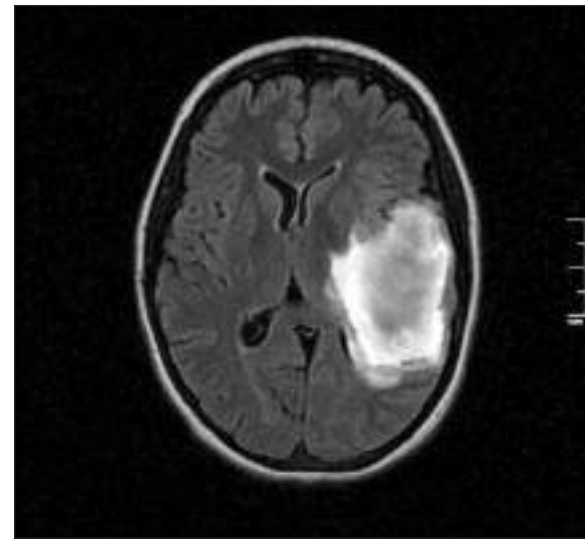

FIGURE 1b: Axial FLAIR MR scan obtained one week after onset of symptoms show left temporoparietal hemorrhage.

DISCUSSION

VZV infections are associated with stroke syndromes in both immunocompetent and immunocompromised patients, but their association with primary intracerebral hemorrhage is extremely rare.[5,6,8] Although immunocompetent patients with VZV infection is associated with stroke syndromes have been reported, VZV infections of the central nervous system causing intracerebral hemorrhage have been reported increasingly, especially in children.[1,9] The median interval time between the episode of VZV infection and the onset of neurologic complaints is approximately two months.[5,6,9]

The etiology of central nervous system complication remains still unclear. Trigeminovascular connections are the possible pathways of virus transmission from the trigeminal nerve to the circle of Willis. Hematogenous seeding or spreading by the sympathetic nervous system is another mechanism suggested for virus spread to the intracranial vessels.[5,6,8,10] The pathological studies of the disease revealed that VZV infection associated with cerebral arteritis is an inflammatory process and gives rise to a severely weakened necrotic vessel wall.[5,8] The infection causes arteritis involving cerebral arteries with focal fibrinoid necrosis and disruption of the medial elastica that had elicited a giant-cell response.[3] Granulomatous angiitis, with multinucleated giant cells, was found at autopsy in the basilar artery wall of aneurysm due to VZV infection.[11]The pathological changes occurred in the vessel wall of our patient probably gave rise to an intracerebral hemorrhage.

In accordance with the literature, one month ago, our patient also had a history of VZV infection and elevated VZV IgG and IgM antibodies due to infection determined by enzyme immunassay. The diagnosis of VZV arteritis is supported by the clinical history, serum IgG and IgM levels of VZV, and absence of other aetiology. We could not perform lomber punction in order not cause cerebral herniation due to the mass effect of ICH.

Our patient had no trigeminal neuralgia, so the virus could have spread heametoenously or by the sympathetic nervous system. Her MRA was normal and cerebral angiography also revealed no pathology probably due to the low specificity and sensitivity.[12,13]

CONCLUSION

Because vasculitis occurred during the VZV infection may lead to an ICH, patients even recovered from VZV infection should be followed at least for a few months.

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