INTRODUCTION

Sudden onset of bilateral abducens nerve palsy related to anterior communicating artery (ACoA) aneurysmal rupture is very rare. A review of literature disclosed only four patients in three case reports regarding isolated bilateral abducens nerve palsy following ruptured ACoA aneurysm. Patients underwent surgical clipping of the aneurysms. The mechanisms of the paresis were speculated to be vasospasm of pontine branch of basilar artery or primary compression of the sixth-nerve caused by entrapment of cerebrospinal fluid (CSF) or blood clot. The recovery period varied from post-operative day 3 to 3 months after onset.

To the best of our knowledge, we describe the first case of slow recovering bilateral abducens nerve palsy following endovascular coil embolization of ruptured ACoA aneurysm and discuss possible mechanisms.

CASE REPORT

A 32-year-old man visited our institution for the evaluation of horizontal diplopia, dizziness and headache after the onset of a 5-minute generalized tonic-clonic seizure. On admission, the patient was alert and did not have any possible vascular risk factors such as hypertension, diabetes mellitus, hyperlipidemia, advanced age and cigarette smoking. The attending neurosurgeon and opthalmologist did not find any abnormality except slight nuchal rigidity and bilateral abducens nerve palsy. Initial cranial computed tomography revealed no definite high density lesion in the brain including pachyma, ventricle and cisternal space, but CT angiography showed focal intraventricular hemorrhage without brain stem abnormalities including infarction or space-occupying lesion. Endovascular coil embolization was conducted to obliterate an aneurysmal sac followed by lumbar cerebrospinal fluid (CSF) drainage. Bilateral paresis of abducens nerve completely recovered 9 weeks after ictus. In conclusion, isolated bilateral abducens nerve palsy associated with ruptured ACoA aneurysm may be resolved successfully by coil embolization and lumbar CSF drainage without directly relieving cerebrospinal fluid pressure by opening Lillequist’s membrane and prepontine cistern.

Key Words: Anterior communicating artery · Subarachnoid hemorrhage · Abducens nerve palsy.
Abducens Nerve Palsy after Ruptured Anterior Communicating Artery Aneurysm

PRES also can cause abducens nerve palsy and seizure. But, the patient had no risk factors of PRES such as HTN, chronic liver failure, or organ transplantation. In addition, MRI revealed no signal change in the cortico-subcortical area of occipital and parietal lobes. Thus, PRES can be ruled out in this case.

Because a ruptured ACoA aneurysm can induce a profound clot in the basal cistern, primary brain stem compression by cisternal hematoma and secondary effect by acute hydrocephalus should be monitored closely. In particular, the abducens nerve courses through the prepontine cistern, so a direct mass effect by CSF entrapment or clot can cause isolated bilateral abducens nerve palsy after ruptured ACoA aneurysm. Although, no prominent prepontine cistern and substantial cisternal change in the subsequent MRI was observed, we may speculate local CSF entrapment as the most probable mechanism for isolated bilateral abducens nerve palsy in this patient.

Lumbar CSF drainage was maintained for 6 days to prevent CSF accumulation and controlling headache refractory to med-
ichotomy. After lumbar drain insertion, headache was relieved. No supporting data about the effect of lumbar drainage for the treatment of abducens nerve palsy were found in previous articles. However, the possibility of reducing the CSF accumulation may exist. Therefore, we suspect that external drainage of CSF may lessen the compression effect of abducens nerve by CSF entrapment.

The recovery period of isolated abducens nerve palsy related to ruptured ACoA aneurysm has reportedly varied. Ziyal et al. and Göksu et al. described relatively early full recovery of abducens nerve palsy (3 days and 1 month after clipping, respectively). On the other hand, Nathal et al. reported a 3-month recovery time and we experienced a 9-week recovery interval. The difference may be explained by a different pathophysiology of the nerve paresis and treatment modality. Even though coil embolization cannot allow us to open prepontine cistern and Liliequist’s membrane, isolated bilateral abducens nerve paralysis was fully recovered by 9 weeks from onset.

CONCLUSION

Isolated bilateral abducens nerve palsy associated with ruptured ACoA aneurysm may be resolved successfully by endovascular intervention followed by lumbar CSF drainage without direct decompression of CSF entrapment in prepontine cistern and Liliequist’s membrane.

References