

CLINEU 00281

## Case report

# A case of pontine lacunar infarction with ulcerative colitis

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### Summary

We present the case of a young man who experienced a sudden onset of pure motor hemiplegia in association with ulcerative colitis. Based on a review of earlier reports, ulcerative colitis was suspected to be the background disease leading to cerebral infarction. A cerebral infarction of the lacunar type in the right ventromedial aspect of the upper pons was seen on magnetic resonance images. These images detected small lesions in the brain stem, and were used to follow-up their transitions.

### Introduction

The causes of cerebral infarction in the young are various [1], and ulcerative colitis has been suspected as one of the background diseases leading to cerebral infarction [2]. Thirteen patients with ulcerative colitis have been previously reported to suffer from cerebral infarction (Table 1). We report the case of another patient with ulcerative colitis who experienced the sudden onset of pure motor hemiplegia.

### Case report

An 18-year-old boy experienced sudden motor weakness of the left arm and leg but no impairment of consciousness. He came to our hospital the second day after

onset. The patient had a 5-year history of ulcerative colitis and reported that his diarrhea had recently become more severe.

The patient's neurologic examination revealed left hemiparesis including left lower facial weakness, but his sensory systems were not disturbed and no other neurologic deficits were apparent. His blood pressure and pulse rate were within normal range, and both an electrocardiogram and echocardiogram showed good function of his heart. Laboratory tests revealed slight microcytic anemia (red blood cell count 5 120 000; hematocrit value 35.6; hemoglobin 9.9), but other data including a platelet count (420 000) were all within normal limits. Partial thromboplastin time, prothrombin time, fibrinogen content, fibrinogen degradation products, and anti-thrombin III were all normal. All coagulation factors and protein C were measured and found normal. No inflammatory signs were detected.

To uncover any background diseases, his ASLO, antibodies to viruses, rheumatic serology, and the concentration of anti-nuclear antibodies were examined. Along with a lupus erythematosus test, these elements were

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found to be normal. His cerebrospinal fluid showed no abnormal findings.

A computed tomogram of the brain revealed an unclear, abnormal low-density area in the right pons. A T<sub>2</sub>-weighted magnetic resonance (MR) image showed a high-intensity area on the right ventromedial aspect of the upper pons that did not cross the midline (Fig. 1B). No abnormalities were detected on a digital subtraction angiogram. Based on these findings, the patient was diagnosed with a pontine infarction and treated conservatively. Follow-up MR images did not contradict this diagnosis. An abdominal x-ray taken after a barium enema showed the characteristic figures of ulcerative colitis. The patient recovered dramatically from his left hemiparesis and was discharged after 2 months.

## Discussion

In the young, the etiologic factors of cerebral infarction differ greatly from those in older individuals [3], and

many cases are described only as 'idiopathic stroke'. Some of these patients have abnormal conditions, but the etiologic relationship with the infarction is unclear. Many authors have reported the existence of a variety of background diseases before the onset of stroke, including convulsive episodes [3], infection, fever of unknown origin and minor head trauma [4]. Mayeux and Fahn [5] were the first to suspect ulcerative colitis as one of the background disturbances having an etiologic connection to cerebral infarction in three of their patients. Other reports soon followed; ours is the fourteenth such case (Table 1).

Because embolic complications such as pulmonary embolism or thrombophlebitis are well known with ulcerative colitis, the cerebrovascular occlusion associated with ulcerative colitis was usually believed to be a thromboembolus resulting from thrombocytosis or a disturbance in coagulation [6]. Other than anemia, which may result from bloody diarrhea, or the usual inflammatory signs of colitis, the hematologic findings in these patients concentrated on the platelet or coagulation test (Table 1).

TABLE 1  
REPORTS OF CEREBRAL INFARCTION IN PATIENTS WITH ULCERATIVE COLITIS

Author	Age (yrs) sex	Location of infarction	Hematologic findings	Clinical stage of UC
Mayeux and Fahn [5]	12, M	—	PLT 1,2 million	active
	17, F	right fronto-parietal lobe	normal	active
	26, F	(not detected on CT)	ESR 20 mm/h, Hct 35%	50 hrs after colectomy
Schneiderman et al. [18]	34, M	left ICA occlusion	PLT 545,000, ESR 16-20 mm/h	active
	52, F	brain stem	PLT 950,000, fibrinogen 440 (200-400), ESR 34-76 mm/h	remission
	12, F	brain stem	Factor VIII 290% (50-200), PTT 26s (29-40), ESR 9-38 mm/h	active
Hilton-Jones and Warlow [19]	20, M	basal ganglia	normal	active
Paradis et al. [20]	12, F	left hemisphere	PTT 23.2s (36.8), WBC 14,000	active
Talbot et al. [9]	45, M	—	—	active
	33, F	—	PLT 662,000	3 months after colectomy
Nelson et al. [7]	18, M	bilateral hemispheres	WBC 10,900, ESR 27 mm/h	active
Jorens et al. [21]	31, M	left internal capsule	PLT 520,000, prothrombin 49% (70-130), protein C 46% (70-130), protein S 55% (70-130), ESR 60 mm/h Hb 10.5	active
Karacostas et al. [8]	32, F	right frontal lobe	WBC 12,200, Hb 9.75	active
Present case	18, M	right upper pons	Hct 35.6, Hb 9.9	active

—: not stated in reference, UC: ulcerative colitis, ICA: internal carotid artery, PLT: platelet, PTT: partial thromboplastin time, WBC: white blood cell, ESR: erythrocyte sedimentation rate, Hct: hematocrit, Hb: hemoglobin. The number in parentheses indicates normal values in each examination.

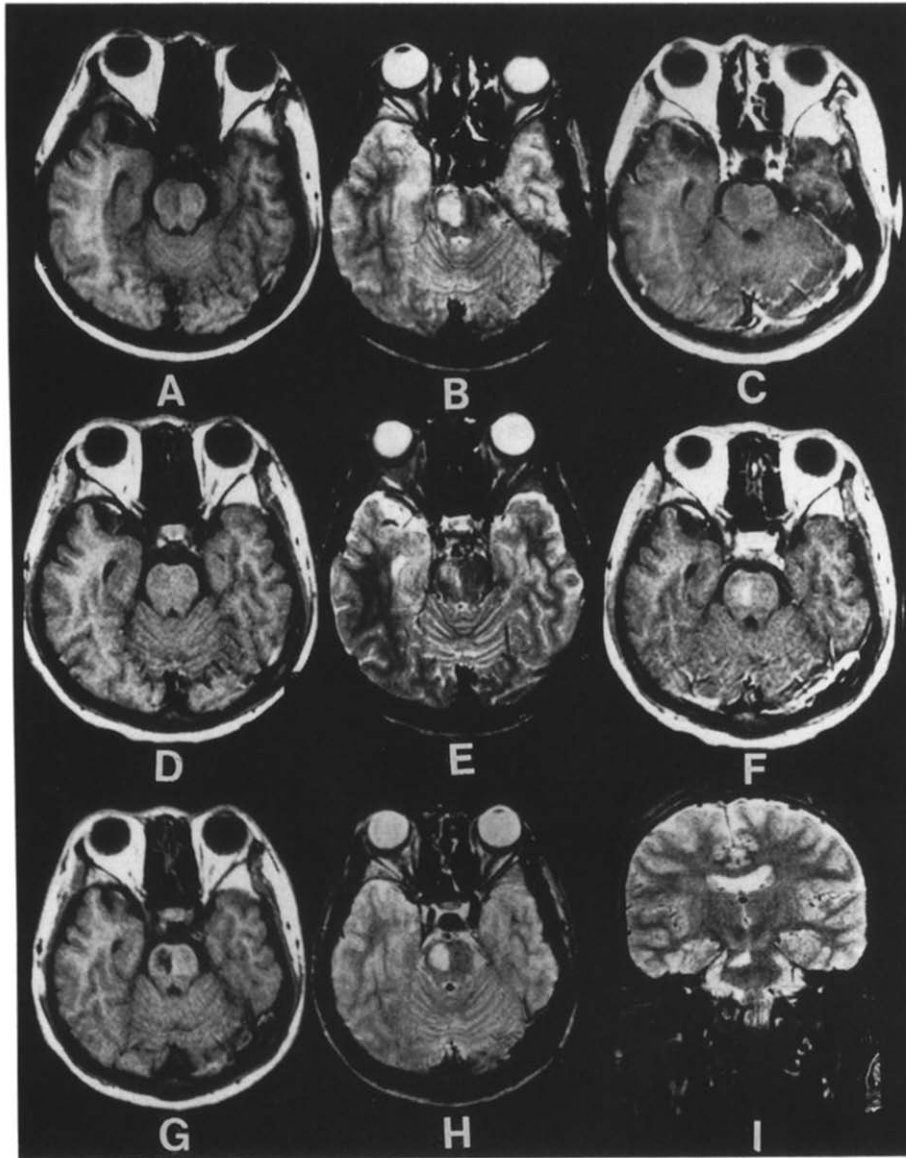


Fig. 1. Magnetic resonance images taken at admission (A–C), 2 weeks (D–F) and 3 months (G–I) after onset. The left side of the body appears at the right side of the figure. At the time of admission (2 days after onset) a slight low-intensity area was seen on the T<sub>1</sub>-weighted image (A) and a high-intensity area was seen on the T<sub>2</sub>-weighted image (B). Enhancement with contrast was not observed (C). Two weeks after onset, the lesion seemed to be small on both the T<sub>1</sub>- (D) and T<sub>2</sub>-weighted (E) images. Enhancement was observed (F), however, and the lesion was believed to be in the subacute stage of infarction. Three months after onset, the lesion was clearly of low intensity on the T<sub>1</sub>-weighted image (G) and of high intensity on the T<sub>2</sub> (H). The lesion did not cross the midline on any axial views and the coronal section of the T<sub>2</sub>-weighted image (I) showed the lesion to be localized in the upper pons.

Two groups of investigators, however, believed cerebral vasculitis to be an important mechanism [7,8]; this has also been found to be a vascular complication of ulcerative colitis [9,10]. Thus, steroids may be more effective than anticoagulants in treating patients with vasculitis [11].

The lesion in our patient appeared to be caused by the occlusion of the right paramedian branch of the basilar artery [12]. Pure motor hemiplegia [13] usually occurs

after a small infarction located in the internal capsule or the pons; consequently, diagnosis using a CT scan is sometimes difficult [14,15]. In patients with multiple lesions, CT scans were not always able to confirm the lesion responsible for the symptoms [16].

On the other hand, MR images are useful not only for detecting a small lesion, but also for delineating the acute cerebral infarction responsible for symptoms. Using MR

images, we could detect the lesion in the right upper pons clearly, and depict the transition of the lesion (Fig. 1). At the time of the patient's admission, the low-intensity region on the T<sub>1</sub>-weighted image was not clear (Fig. 1A). After 2 weeks, however, the area of infarction was seen after enhancement (Fig. 1F). After 3 months, the low-intensity area was well-demarcated (Fig. 1G), showing that the infarction was in the chronic stage.

The mean age of patients in the case reports is 25.9 years. In all but one patient, the clinical stage of ulcerative colitis was active or in the postlaparotomy phase at the time of stroke. These factors lead us to believe that adequate treatment of ulcerative colitis may be necessary to prevent the onset of stroke. Furthermore, when confronted with a juvenile patient suffering ischemic stroke, neurologists should consider ulcerative colitis as a background disease along with more common diseases such as mitral valve prolapse and syphilis [17].

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