

TO THE EDITOR

RE: Neuroscience in Nazi Europe Part I: Eugenics, Human Experimentation, and Mass Murder**Can J Neurol Sci. 2011; 38:696-703**

Dear Dr. Young,

As the author of this article,¹ I wanted to make a small annotation to "Table 1. Neuroscientists who collaborated in the Nazi euthanasia programs or performed other experiments on humans." It is mentioned in the table regarding Hugo Spatz, Hans Joachim Scherer, and Willibald Oscar Scholz that they "actively acquired and researched brains from euthanasia victims," similar to Julius Hallervorden. There certainly is evidence that Hallervorden may have initiated and was "active" in the acquisition of brains from mentally ill patients murdered in the Nazi "euthanasia" programs during World War II.² He is quoted in an interview after the war as stating, "I heard that they were going to do that, and so I went up to them and told them, 'Look here now, boys. If you are going to kill all those people, at least take the brains out so that the material can be utilized.' They asked me, 'How many can you examine?' And so I told them, 'An unlimited number - the more the better.' There was wonderful material among those brains, beautiful mental defectives, malformations and early infantile diseases. I accepted the brains, of course. Where they came from and how they came to me was really none of my business."² Hugo Spatz was director of the institute where Hallervorden worked, and tried to cover up Hallervorden's and the institution's involvement.² There is also evidence that Spatz's own laboratory studied 105 brains from various hospitals and clinics that were either certainly or very probably from "euthanasia" victims as well.³ Hans Joachim Scherer conducted pathological studies on brains of 350 "euthanized" Polish and German children from the Loben/Lubliniec mental hospital.⁴ Willibald Scholz's institute in Munich participated in neuropathological research on at least 194 brains from at least four specialized pediatric killing wards at state psychiatric hospitals in Bavaria during the war.³ At least 11 papers were published based on this research, and Scholz was

main author and editor of volume 13, focusing on neuropathology, of the 1956 *Handbuch der speziellen Pathologischen Anatomie und Histologie*, which frequently features "euthanasia" cases.^{3,5} It is unclear from the literature and data I have seen that Spatz, Scherer, and Scholz were as "actively" involved in the "euthanasia" programs as Hallervorden was. It is conceivable that they were, but the documentation was intentionally destroyed, did not survive the war, or has not been discovered yet. But based on the available evidence and literature, all I believe can be stated is that Spatz, Scherer, and Scholz "took part in neuropathological studies on murdered victims in the Nazi 'euthanasia' programs," and that is what should be stated in Table 1 regarding these individuals. Hopefully with further research, the extent of the unethical roles these three individuals played in murderous Nazi programs will become more apparent.

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TO THE EDITOR

Lumbar Radiculopathy-Mimicking Cortical Infarction of the Precentral Region

Restricted sensory abnormalities to a small area, mimicking a radicular or peripheral nerve distribution, is uncommon in cerebral infarction. Restricted sensory symptoms in most reported cases were median or ulnar nerve distributions, i.e. peripheral nerve distribution of upper limb.¹⁻³ Sensory deficits showing peripheral nerve distribution of lower limb are quite rare. There have been no reports of cortical infarction. We present a patient with focal cortical infarction mimicking lumbar radiculopathy.

CASE REPORT

A 49-year-old woman was admitted for the evaluation of left leg weakness and radiating pain with acute onset. She had

hypertension, diabetes mellitus, and no history of previous stroke. She had a history of intermittent low-back pain. On neurological examination, proximal leg weakness was more severe than distal leg weakness. Medical Research Council (MRC) grade of hip flexion and knee flexion was 3/5. The MRC grade of hip extension/abduction/adduction, knee extension, and ankle dorsiflexion/plantar flexion was 4/5. There was hypesthesia for all modalities and paresthesia in the L4 and L5 dermatome distribution, but it did not match either a classic radicular or peripheral nerve pattern. Sensory deficit was more severe around the knee region (Figure 1). The deep tendon reflexes were normal. There was no Babinski sign. Four days later, her sensory deficit was improved and the pain sensation became normal. Lumbar spine magnetic resonance imaging (MRI) was performed with normal results. Brain MRI including diffusion-weighted image revealed acute infarct in the high level of precentral gyrus of left hemisphere, medial occipital area, and

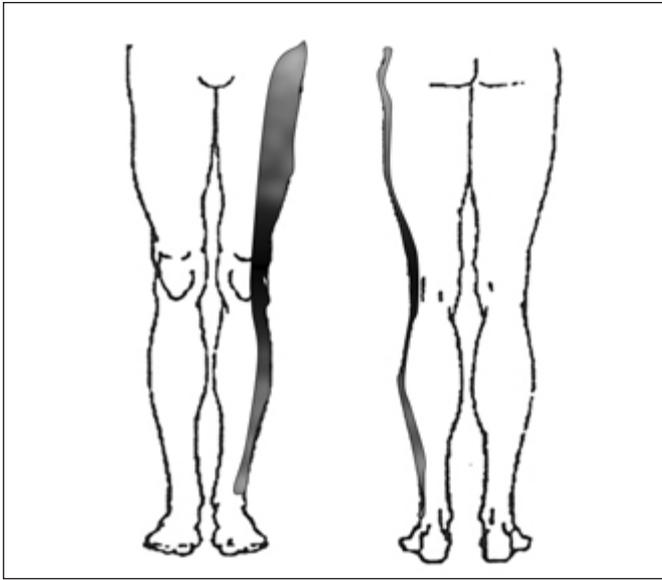


Figure 1: Schematic drawing of sensory dysfunction (shaded area). The degree of darkness indicates the severity of the sensory dysfunction.

centrum semiovale (Figure 2A-C). The lesion in the precentral gyrus was thought to be associated with her leg weakness and sensory deficits. Right internal carotid angiogram showed an unruptured, paraclinoid saccular aneurysm (Figure 2D). Motor and sensory nerve conduction studies were normal. Two weeks later, because of the size of the aneurysm and its presumed etiology for the multiple embolic infarcts, endovascular coil embolization was performed. She was treated with antiplatelet medication. Two months later the leg weakness and the sensory deficits were completely resolved.

DISCUSSION

Our case demonstrated that cortical infarction might mimic lumbar radiculopathy. Her clinical features were quite similar to lumbar radiculopathy because of leg weakness, dermatomal distribution of sensory deficits and radiating pain. Her intermittent back pain was confusing for clinicians. However, close temporal relationship between brain MRI findings and her symptom onset and her MRI of normal lumbar spine were clear for the diagnosis.

We found two similar cases in the literatures.^{4,5} One described an infarction in the centrum semiovale deep to the primary motor and sensory cortices which imitated sacral radiculopathy, but the association was unclear because brain MRI did not provide the lesion was acute and the elderly patient

had suffered from herpes zoster for several months. The other case showed sensory level at the thoracic level after small ruptured arteriovenous malformation in the right parietal lobe, but there was no radiating pain.

In conclusion, cerebral infarction can show a clinical spectrum of wide diversity. It is important to be suspicious of cerebral ischemia in the setting of abrupt onset of symptoms, known risk factors, and rather atypical weakness pattern for radiculopathy.

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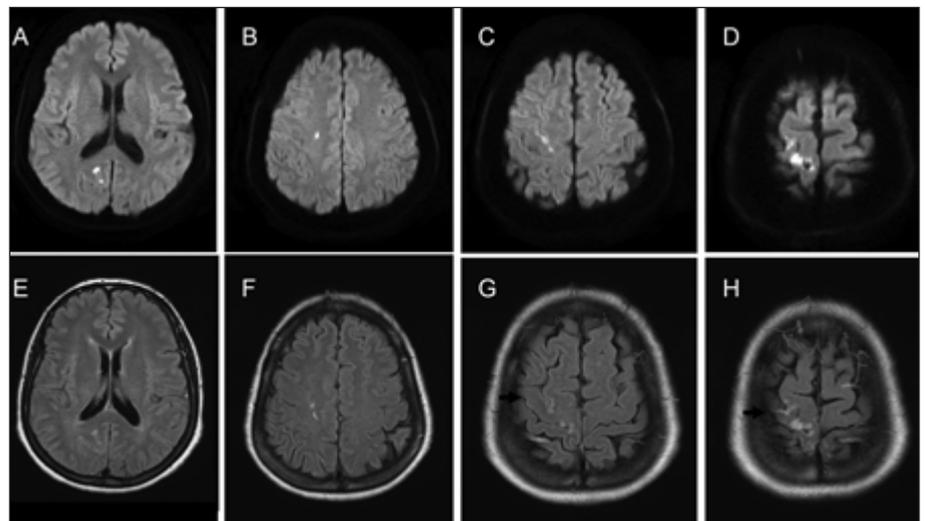


Figure 2: Diffusion-weighted image and fluid attenuated inversion recovery reveals acute infarction involving precentral region, medial occipital lobe, and centrum semiovale in the right hemisphere. The arrow indicates the central sulcus.

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