Case report

Laughing at funerals

Y. MADANI1, S. AZAD2, P. NACHEV3 and D. COLLAS4

From the 1 Queen’s Hospital, Department of Respiratory Medicine, Romford, Essex, 2 The Heart Hospital, University College London Hospitals NHS Foundation Trust, Cardiology, London, 3 Institute of Neurology, Queen Square, Neurology London and 4 Watford General Hospital, Stroke Unit Watford, Hertfordshire, UK

Address correspondence to Dr Y. Madani, Department of Respiratory Medicine, Queen’s Hospital, Rom Valley Way, Romford, Essex, RM7 0AG, UK. email: yasser.madani@doctors.org.uk

Learning Point for Clinicians

Ischaemic strokes are a cause of pathological laughter (PL). PL can be an acute or delayed presentation of strokes. Several neuroanatomical sites have been implicated in PL. PL can be mood incongruent or it can be exaggeration of a normal response. As with action and cognition, acute disturbance of emotional expression should also be recognized as having a neural basis. Serotonin reuptake inhibitors have been used in the management of delayed-onset PL after strokes.

Case history

A 58-year-old man was referred to clinic with a complaint of episodic laughter. He had been playing charades with his family at Christmas when he found himself unable to continue owing to uncontrollable laughter. Though the game had been lively, nothing especially amusing had occurred. He thought little of it, until the episode recurred at a funeral, where he laughed throughout the service without any cause, and was profoundly embarrassed. The most recent episode was while visiting his mother after she had been discharged from hospital, and was in anything but an amused mood. These events caused him to retire from social contact, for he found the incongruity of his displays very distressing. Of note, there was no history of seizure phenomena. A year earlier, he had suffered an ischaemic stroke presenting with right arm and facial weakness and dysarthria. Computed tomography scan of the head at the time was unremarkable. His symptoms had begun 2 months after the stroke. His other past medical history included hypertension, hypercholesterolaemia and diabetes mellitus. Magnetic resonance imaging (MRI) of the head a year after the stroke demonstrated several scattered focal areas of high signal consistent with focal infarcts in the parietal white matter, left temporal lobe and the left cerebral peduncle, with associated focal degeneration of the white matter tracts (Figure 1). The patient was prescribed citalopram to take this prophylactically before an event which was likely to evoke laughter. The patient used citalopram on two occasions to which he responded well. He did not require any further doses after this.

Discussion

PL has been reported as an acute or delayed presentation of strokes. Acute strokes presenting as a display of laughter, ‘le fou rire prodromique’1, have been reported in infarcts involving the internal capsule, thalamus, basal ganglia and pons. PL has been associated with pseudobulbar palsy, gelastic seizures, multiple sclerosis, cerebellar pontine tumours, Alzheimer’s disease, Pick’s disease and Wilson’s disease.
Several mechanisms of PL have been hypothesized. Mendez et al.\textsuperscript{2} devised a neuroanatomical circuit involving five main sites: anterior cingulated gyrus, amygdalae, caudal hypothalamus, ventral pontomedullary and corticobulbar tracts. Other anatomical sites involved in PL described by Parvizi et al.\textsuperscript{3} include the internal capsule, cerebral peduncles and the cerebellum.\textsuperscript{3} Most authors agree that there is a final common pathway in the brainstem integrating autonomic reactions and facial expressions. The occurrence of delayed PL after a stroke indicates that there is a different mechanism involved than just simple disruption of the pathways. Delayed PL reflects the time required for the formation of autonomous motor centres and it has been suggested that there is formation of new neuronal activity or new laughter control centres.\textsuperscript{4}

The neuroimaging in this case demonstrated multiple lesions in the parietal white matter, cerebral peduncle and temporal lobe, all of which have been implicated in PL. The corticobulbar pathways play an important role in modulating emotional expression by inhibiting involuntary laughter. Thus, a lesion affecting the corticobulbar tract can cause disinhibition of laughter. The corticobulbar tract runs through the cerebral peduncle, one of the areas implicated in this case.

The type of PL seen in this case was a paroxysmal event which was mood incongruent. This is different from the PL seen in pseudobulbar palsy where it is an exaggeration of a normal response. Pseudobulbar palsy causes non-specific emotionally lability, such that patients will laugh at things only mildly funny and cry at things only moderately sad.

While acute disturbance of action and cognition is readily recognized as having a neural basis, acute disturbance of emotional expression is not. But since emotion has the brain as its substrate in exactly the same way as action and cognition, changes in it ought to be interpreted and investigated in the same way.

Serotonin reuptake inhibitors have been used in the management of delayed-onset PL after strokes.\textsuperscript{5} In this case, the laughter subsided with the occasional use of citalopram. Previous case reports have described improvement in PL with the resolution of neurological symptoms and complete recovery within few months following this.\textsuperscript{6}

Conflict of interest: None declared.

References

Figure 1. Axial brain MRI views (T2 FLAIR) showing a focal area of high signal in the left cerebral peduncle.