

## Case Report

# Acute-Onset Stuttering as a Stroke Mimic

Jennifer L. Dearborn<sup>1\*</sup>, Cassie Davis<sup>2</sup> and John W. Krakauer<sup>3</sup><sup>1</sup>Department of Neurology, Yale School of Medicine, USA<sup>2</sup>Department of Psychiatry, Seattle University of Washington School of Medicine, USA<sup>3</sup>Departments of Neurology and Neuroscience, Johns Hopkins School of Medicine, USA

## Corresponding author

Jennifer Dearborn, Yale University School of Medicine, Department of Neurology, LCI 1003, 15 York Street, New Haven, CT 06510, USA, Tel: 203-737-1057; Fax: 203-737-4382; Email: jennifer.dearborn@yale.edu

Submitted: 20 May 2015

Accepted: 21 July 2015

Published: 23 July 2015

ISSN: 2333-7087

## Copyright

© 2014 Dearborn et al.

## OPEN ACCESS

## Keywords

- Stuttering
- Adult-onset stuttering
- Speech dysfluency and stroke
- Conversion disorder
- Psychogenic stuttering

## Abstract

**Background and purpose:** The acute onset of stuttering in an adult is not uncommon and rarely is caused by ischemic stroke. A neurologist is likely to encounter this presentation and it is important to identify distinguishing features between ischemic stroke and other causes. Adult-onset acquired stuttering is classified as either neurogenic if secondary to brain injury or psychogenic in the absence of structural brain lesions. There are particular features of dysfluency in acquired stuttering that are distinct from developmental stuttering.

**Clinical presentation:** We present a case of a 35-year old woman without a childhood history, who had witnessed acute onset of stuttering which resulted in arrival in the emergency department and activation of the acute stroke team. She progressed to muteness and then improved to a phenotype of neurogenic stuttering.

She had almost fully recovered by hospital discharge, however her stuttering recurred consistent with psychogenic stuttering. The diagnosis was confirmed at 6 month follow-up.

**Conclusion:** This case is notable because of the evolution the phenotype and the absence of an obvious culprit lesion or obvious psychological precipitants.

## INTRODUCTION

Stuttering, or speech dysfluency, is a phenomenon characterized by blocking and prolongations in sounds of words and often represents a developmental disorder, which can be present in up to 11% of children [1]. In an emergency department setting, the acute onsets of speech dysfluencies can present as a stroke mimic, and create a diagnostic challenge in the early evaluation [2]. Neurogenic stuttering refers to a person who may not have stuttered in childhood, and is often caused by neurologic conditions such as Parkinson's disease, dementia, stroke or brain tumors [3,4]. If an underlying cause is found, stuttering is referred to as "neurogenic" and when no organic cause is found, patients often receive diagnosis of a psychogenic disorder [5]. This is in contrast to persistent developmental stuttering (PDS), which first manifests in children, occasionally persisting into adulthood [6]. The optimal approach to an adult patient with the acute onset of stuttering in the absence of an obvious culprit brain lesion should be multi-disciplinary to best characterize the complex interplay between childhood predispositions, psychosocial stressors and the phenotype.

## CASE PRESENTATION

A 35-year-old woman presented with the acute onset of difficulty speaking. She initially presented to an outside emergency

department with nausea and vomiting without an obvious precipitant, and when in triage, she became unable to speak fluently. She had a history of frontal headaches that did not meet criteria for migraines, and of anxiety. There was no history of stuttering, developmental delay, or language problems during childhood.

The treating physician found no other neurologic deficits and described her speech impairment as dysarthria with stuttering. During her first examination, she was noted to have extreme difficulty saying words, and appeared frustrated, prolonging word sounds and initial syllables; however the word would be correct when spoken. She was unable to say more than one word at a time, and had difficulty stringing together syllables (dysfluency). She had other behaviors such as facial grimacing and eye blinking that often did not coincide with speech. She was able to follow commands, although she seemed challenged with tests of apraxia such as copying hand gestures. There were no other neurological deficits including aphasia.

Although there was some concern for an acute stroke, thrombolytics were not administered because of the uncertainty about the diagnosis. Brain MRI, was negative for acute stroke or any other structural abnormality. The following day, her pattern of speech evolved to be slightly more fluent. An electroencephalogram was normal without evidence of epileptic form discharges. Psychiatry was consulted on the second day