A Neurological Analog of the Dynamic EKG? 
Fluctuating Aphasia in a Cocaine User 

Hood-Medland E., Kravitz R., Aronowitz P. 
University of California, Davis Medical Center; Sacramento, CA

Introduction

Cocaine use has been shown to have a strong association with ischemic stroke in young adults. Proposed mechanisms include increased sympathetic drive, increased atherosclerosis, vasculitis, and vasospasm. Patients generally present with focal neurologic deficits, with infarcts most commonly found in the MCA territory.

Case Presentation

History of Present Illness:
A 28-year-old man brought in by a friend to the emergency department for altered mental status and an episode of vision loss in the right eye.
Timeline:
• 3 days ago – in usual health
• Morning of presentation – communicating by phone with friend, unable to give directions. Complained of vision loss in right eye.
• Prior to presentation - his friend picked him up from his house, and he was ‘insensible.’
• Friend brought patient to emergency department

Past Medical History/Past Surgical History:
Non-ischemic cardiomyopathy
Congestive Heart Failure with Reduced Ejection Fraction

Social History:
Cocaine and Methamphetamine use

Medications:
Carvedilol, Lisinopril, Furosemide

Physical exam:
Vital signs were significant for tachycardia and tachypnea. Benign heart, lung, and abdominal examination. On neurologic examination, he cycled from normal language function to expressive aphasia to global aphasia back to normal language function over the course of a few minutes, without other neurologic deficits

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Labs and Imaging

<table>
<thead>
<tr>
<th>Pertinent Laboratory Studies</th>
<th>Other Investigative Studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>CBC</td>
<td>Transcranial echocardiography</td>
</tr>
<tr>
<td>Chem7</td>
<td>EF 30%</td>
</tr>
<tr>
<td>LFTs</td>
<td>No thrombus or vegetation</td>
</tr>
<tr>
<td>Urine toxicology screen</td>
<td>Electroencephalogram</td>
</tr>
<tr>
<td>(+) Cocaine</td>
<td>No epileptiform activity</td>
</tr>
<tr>
<td>(-) Amphetamines</td>
<td></td>
</tr>
</tbody>
</table>

Hospital Course

- Admitting differential diagnosis: stroke, seizure, drug intoxication
- The patient was noted to have infarct on MRI/MRA. Neurology was consulted, and advised CTA, which showed no evidence of vessel stenosis or vasospasm. EEG showed no epileptiform activity.
- Despite the negative CTA, cerebral vasospasm was posited as the etiology of ischemic stroke; and dual anti-platelet therapy was withheld.
- After working with speech therapy, his symptoms resolved by hospital day 13, and he was discharged home in good condition.

Discussion

This patient presented in an unusual fashion, as his symptoms cycled rapidly from normal to aphasic over a span of minutes, with no sensory or motor deficits. Fluctuating symptoms suggested a reversible etiology such as seizure or vasospasm rather than a fixed defect such as embolism or vessel thrombosis. Ischemic stroke in young adults is rising in prevalence, with cocaine as a putative etiology in at-risk patients. One mechanism of cocaine-associated stroke is cerebral vasospasm. Although vasospasm was not apparent on CT angiography, transitory alterations in vessel physiology may not be visible on later imaging. We believe this patient’s presentation of rapidly fluctuating aphasia was due to cerebral vasospasm from cocaine use not unlike that sometimes seen in coronary vasospasm with dynamic EKG changes or fluctuating chest pain. In this case, fluctuating aphasia as detected by a careful neurologic exam may have been the neurologic analog of dynamic EKG changes in cardiology.

Conclusion

Fluctuating neurological changes may be a neurologic analogue of dynamic EKG changes in cardiology in patients experiencing cocaine-induced vasospasm of cerebral vessels.

References