Difficulties with the neurological assessment of humans following a chimpanzee attack

Case report

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Chimpanzee attacks can be vicious, mutilating, and disabling if not fatal. Stereotypically, the hands and face are targeted, and in male victims, genitalia are mutilated. The authors present a case highlighting the difficulties with early neurological assessment following such an attack. This 55-year-old woman was attacked by a 14-year-old chimpanzee. She suffered mutilation of both hands, severe midface bony, soft-tissue, and eye injuries, and scalp degloving. An emergency tracheotomy was performed at the scene, with an unclear duration of hypoxia. The patient was unresponsive without spontaneous movements, papillary or corneal reflexes, cough, or gag. Attempts to lighten sedation were not tolerated. Brain CTs were normal. Intracranial pressure monitoring was deemed infeasible. Brain MR imaging suggested diffuse axonal injury consistent with severe shaking trauma. Diffusion tensor imaging indicated intact corticospinal tracts, confirmed by somatosensory evoked potentials. Magnetic resonance imaging suggested left optic nerve transaction, and right retinal detachment was diagnosed. Electroencephalography showed severe diffuse encephalopathy. Auditory evoked potentials showed absent auditory pathway responses except for a right delayed wave V. Visual evoked potentials indicated absent visual function. At 1 month after the attack, sedation and analgesia weaning revealed lower-extremity movement to command, but no upper-limb response. Cervical spine and brachial plexus MR imaging showed brachial plexus edema. Two months after the attack, the patient regained strength in all her extremities and verbally communicated using a Passy-Muir tracheotomy valve. Chimpanzee attacks on humans can cause extensive, life-threatening injuries. The neurological assessment of such patients is challenging, complicated by limb and craniofacial disfigurement and the need for heavy sedation. Initial assessment of nervous system integrity may rely on costly imaging and electrophysiological studies. (DOI: 10.3171/2011.2.JNS101911)

Key Words • chimpanzee attack • neurological assessment • traumatic brain injury

This article contains some figures that are displayed in color online but in black and white in the print edition.
to move her foot. An emergency tracheotomy was performed at the scene, with the duration of hypoxia unclear. She was taken to a local hospital where 4 teams of surgeons operated for more than 7 hours to stabilize her. Debridement and closure of the complex head injuries, amputation of the left upper extremity below the elbow, and thumb-sparing amputation of the right hand were performed before the patient was transferred to the Cleveland Clinic for further medical management and possible consideration for face transplantation, given our center’s special expertise in that field.

Upon arrival at the Surgical Intensive Care Unit at the Cleveland Clinic, the patient was heavily sedated; she was unresponsive without spontaneous movements and had absent papillary, corneal, cough, or gag reflexes. Deep tendon reflexes were 2 at the biceps muscle bilaterally and 1 at the triceps muscle and patella bilaterally. The patient had a positive Babinski response bilaterally. There was no spontaneous movement or limb response to painful stimuli. Multiple attempts to lighten sedation were made, yet she failed to show any movement; long periods of withholding sedation were not tolerated hemodynamically, related to a sympathetic nervous system response (that is, severe hypertension and tachycardia) that was extremely difficult to control even with continuous antihypertensive infusions. This response to withholding sedation also interfered with and precluded adequate mechanical ventilation.

Her neurological status was unclear given the absence of functioning sensory facial organs as a result of the severe mutilation. In fact, on the “death by neurological criteria examination,” only a response to hypercarbia via the tracheostomy could be assessed. We were asked to evaluate the patient for neurological prognosis. This determination was crucial with respect to planning for potential complex, staged reconstructive surgeries. As the clinical examination was so greatly limited, we resorted to imaging and electrical recording studies.

**Intervention.** A noncontrast brain CT study was normal. Intracranial pressure monitoring was deemed infeasible given the extent of potential soft-tissue infection and colonization of the face, scalp, and exposed bone with rare and resistant organisms from the chimp mouth flora, including *Fusarium* species, *Zygomycetes* species, *Arthrogramphis kalrae* mold species, *Aeromonas* species, *Pseudomonas putida*, and *Candida albicans*, and given the degloving injury and the risk of introducing infection intracranially. The calvaria remained intact following the attack, so any procedure that entailed breaching the intact barrier to infection was avoided given the danger of introducing infection intracranially. The calvaria remained intact following the attack, so any procedure that entailed breaching the intact barrier to infection was avoided given the danger of introducing infection intracranially. The table summarizes the diagnostic tests that were performed in the assessment of the patient. A brain MR imaging study did not show evidence of diffuse cortical necrosis, but was suggestive of diffuse axonal injury involving the cortical white matter and corpus callosum and small scattered foci of subarachnoid hemorrhage at the convexities consistent with severe trauma, but with no gross CNS abnormality (Fig. 1). Diffusion tensor imaging was consistent with intact corticospinal tracts, which were confirmed with bedside somatosensory evoked potentials (Fig. 2). Magnetic resonance imaging demonstrated a left optic nerve transection and right-sided retinal detachment (Fig. 3). An electroencephalogram showed periodic slowing consistent with severe diffuse encephalopathy without epileptiform activity. Multiple auditory evoked potentials showed mixed results, clouded by fluid in the mastoids and middle ear, with the best results showing absent auditory pathway responses except for a right delayed wave V (Fig. 4). Visual evoked potentials indicated absent visual function (Fig. 5). Lumbar puncture, performed with the patient under anesthesia, showed no evidence of meningitis. The opening pressure was recorded as “normal,” although a specific opening pressure was not documented.

Over the course of 1 month, the patient’s blood pressure control improved, and the sedation was weaned. She started to have some minor tongue movements, but no upper- or lower-extremity movements. Repeat MR imaging of the craniospinal axis as well as the brachial plexus bilaterally showed no structural abnormality in the brain, brainstem, or spinal cord. The roots of the brachial plexus were intact without evidence of avulsion, but there was significant edema. It was extremely challenging to assess higher cortical function given her hearing impairment, visual impairment, inability to blink (due to the loss of her eyelids), and inability to write or type.

The patient had aggressive supportive therapy and multiple facial debridement surgeries. At 1 month after the attack, the sedation and analgesia were entirely weaned, and the patient demonstrated lower-extremity movement to command but no upper-limb response. She was tolerating continuous positive airway pressure trials and was weaned to a tracheostomy collar.

<table>
<thead>
<tr>
<th>Study Findings</th>
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<tr>
<td>MRI/MRA/MRV brain diffuse axonal injury in bilateral corona radiata, scattered foci of SAH overlying the high convexity region; normal MRA &amp; MRV</td>
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<tr>
<td>MRI C-spine mild degenerative changes</td>
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<tr>
<td>EEG continuous slow correlate w/ severe diffuse encephalopathy</td>
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<tr>
<td>AEP absent auditory pathway &amp; absent rt auditory pathway except for delayed wave V</td>
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<tr>
<td>posterior tibial SEP normal bilaterally</td>
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<td>VEP absent responses bilaterally</td>
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<td>MRI brachial plexus bilat edema like changes of brachial plexus roots w/o structural abnormality</td>
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<td>MRI orbit it optic nerve transaction &amp; rt retinal detachment</td>
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<td>lumbar puncture normal opening pressure; no evidence of meningitis</td>
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* AEP = auditory evoked potential; C-spine = cervical spine; EEG = electroencephalography; MRA = MR angiography; MRV = MR venography; SAH = subarachnoid hemorrhage; SEP = somatosensory evoked potential; VEP = visual evoked potential.

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TABLE 1: Summary of the diagnostic studies used to assess a patient with severe mutilating injuries precluding a neurological examination following a chimpanzee attack

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1. Diffusion tensor imaging was consistent with intact corticospinal tracts, which were confirmed with bedside somatosensory evoked potentials (Fig. 2). Magnetic resonance imaging demonstrated a left optic nerve transection and right-sided retinal detachment (Fig. 3). An electroencephalogram showed periodic slowing consistent with severe diffuse encephalopathy without epileptiform activity. Multiple auditory evoked potentials showed mixed results, clouded by fluid in the mastoids and middle ear, with the best results showing absent auditory pathway responses except for a right delayed wave V (Fig. 4). Visual evoked potentials indicated absent visual function (Fig. 5). Lumbar puncture, performed with the patient under anesthesia, showed no evidence of meningitis. The opening pressure was recorded as “normal,” although a specific opening pressure was not documented.

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Postintervention Course. Two months after the attack, the patient had regained strength in all extremities and began to verbally communicate using a Passy-Muir tracheostomy valve with intact cognition although she was amnestic to the attack. She had a prolonged hospitalization course with further facial and limb debridement surgeries as well as inpatient physical rehabilitation. At the 1-year follow-up, she was communicative, oriented, and able to walk unassisted.

Discussion

If violence is defined by fatal intraspecies attacks that are not motivated by predation or the drive for food, then chimpanzees can be very violent. Aggression is a common part of chimpanzee behavior, with both intra-\cite{3-5,7,13,16} and intergroup\cite{10,11,17,18} violence over territorial disputes being observed. Male chimpanzees are more aggressive than females. This behavior has been described as warfare among groups, with males patrolling their territory in groups and seeking other male competitors to kill.\cite{3,12} They have also been known to commit infanticide.\cite{4,17} Their attacks result in tremendous mutilation, using their large teeth to target the face, throat, hands, feet, and also the genitalia. A human’s physical resemblance to the chimpanzee as well as the similarities in the way we attempt to defend ourselves in the event of an attack probably account for the shared pattern of mutilation between human and chimpanzee victims.

Chimpanzee attacks on humans are devastating and follow the stereotypical pattern of intraspecies mutilation exhibited among chimpanzees. Chimpanzees, whose genetic profile is 95% like ours,\cite{2} can seem like cute, hairy iterations of people. Periodic violent attacks on humans following this pattern of injury are reminders that these animals possess brute strength and are wild, and thus remain unpredictable and incredibly dangerous despite our perception of their “domestication.”

In 2005 there was a similar attack on a 62-year-old
Neuroassessment after chimpanzee attack

Southern California man who was attacked by 2 male chimpanzees at an animal sanctuary. He was severely maimed; confined to a wheelchair, he wears a silicone nose prosthesis to hide his exposed septum and a glass eye prosthesis. He underwent numerous facial reconstructive surgeries to repair his broken bones in the maxillary and orbital regions, and his mouth and supporting muscles had to be entirely reconstructed as they had been ripped off.

A similar pattern of injury was observed from attacks occurring in 2006 in Sierra Leone, when male chimpanzees escaped a sanctuary and targeted tourists and workers. Several people working together were able to escape alive with only severe hand injuries. One person was isolated from the group and killed; in addition to being subjected to face, throat, hand, foot, and genital mutilation, the man was eviscerated. These attacks have served to highlight the fact that the general human population has a very unrealistic understanding of how truly dangerous an adult male chimpanzee can become.

Our patient described in this report suffered crushing and tearing injuries to both hands, head injuries involving the entire face and larynx, near enucleation of the eyes, and her hair and scalp had been ripped off. Neurological assessment of patients in whom facial sensory organs are extensively damaged is challenging and complicated by concomitant craniofacial and limb disfigurement. In fact, because of the injuries inflicted, none of the elements of the Glasgow Coma Scale could be assessed in our patient. Likewise, the only aspect of the brain death examination that could be tested was a response to hypercarbia given the destruction of many of the sensory and motor end targets of the cranial nerves. As an additional hurdle, the need for heavy sedation to maintain hemodynamic and ventilator stability, probably related to severe pain and encephalopathy, masked our ability to assess changes in her neurological status. Further complicating her assessment, the patient had suffered airway compromise during the attack and was known to have been hypoxic upon the arrival of emergency medical personnel, but the duration and severity of the hypoxia was unclear. These factors combined with the presence of diffuse axonal injury on MR imaging made it impossible to predict if or when she

![Fig. 3. Axial T2-weighted MR image demonstrating transection of the left optic nerve.](image)

![Fig. 4. Tracings of auditory evoked potentials (AEP) showing absent auditory pathway responses except for a right delayed wave V.](image)
would recover consciousness and cognition. Ultimately, costly imaging and electrophysiological studies proved to be the most effective in assessing the integrity of portions of her nervous system and should be considered early for victims of such attacks, particularly when such prognostic information is required prior to the initiation of aggressive and complex reconstructive surgeries.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Barnett. Acquisition of data: Khalil. Analysis and interpretation of data: all authors. Drafting the article: Khalil. Critically revising the article: Barnett, Spiotta. Reviewed final version of the manuscript and approved it for submission: all authors. Study supervision: Barnett.

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Fig. 5. Tracings of visual evoked potentials (VEP) showing absent visual evoked response in both eyes.