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Human Coronavirus NL63 and Acute Encephalitis

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Short Communication

An active, working 70-years-old woman was admitted in December with cough, low-grade fever and altered behavior over one week during which she stopped eating and speaking and became poorly responsive. She had mild, well-controlled diabetes and hypertension but no prior admissions.

Fever (38.6°C) and tachycardia were noted, with apathy, slow speech and disorientation. Neither meningeal irritation signs nor other abnormality were found on examination. On the next day, somnolence with fluctuations, four-limb symmetric spasticity and later, catatonia with incontinence developed. Psychosis was ruled out.

Intravenous ceftriaxone and acyclovir were started, and nasogastric tube inserted for feeding and oseltamivir treatment. Valproate was added when EEG demonstrated temporal epileptiform activity (Lt.>Rt.) and generalized slowing. Laboratory tests showed leukocytosis (15.6X10⁹/L), thrombocytosis (594X10⁹/L), and CRP 250 mg/dL without metabolic abnormality or autoantibodies. The cerebrospinal fluid was twice normal, including PCR for multiple infectious agents. Chest X-ray showed interstitial changes. Neuroimaging revealed only pansinusitis. Extensive cultures and serology tests remained negative. Repeated nasopharyngeal swabs PCR revealed coronavirus NL63 whereas other coronaviruses, rhinovirus, influenza, para-influenza, RSV, adenovirus and herpesviruses were not detected (BioFire Diagnostics).

Fever and cough abated. On the second week the patient made dramatic improvement gradually regaining her previous functional status. She was discharged home on the 15th hospital day.

Our patient had an acute, self-limited CNS infection manifesting primarily by altered consciousness, confusion, refusal to feed, catatonia and epileptifom activity which were fortunately, completely reversible.

The occurrence in the wintertime, associated respiratory infection and sinusitis, and spontaneous

Coronaviruses predominantly infect the upper (usually mild illness) and lower (potentially

severe disease) respiratory tract. No previous cases of human coronavirus NL63-associated

encephalitis have been reported. However, similar coronaviruses have strong CNS tropism, as

both clinical and experimental studies have demonstrated [3-5]. The etiology of neurologic disease remains poorly understood and could be due to misdirected host immune responses [5]. Thus, in

vulnerable populations such as the elderly, spread from the respiratory tract to the CNS must be

considered, and as our patient suggests, coronavirus NL63 should be considered among viral agents

OPEN ACCESS recovery are all consistent with human coronavirus NL63 infection [1,2] - the single pathogen identified.

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capable of causing acute encephalitis in humans.