valides, sensibilité y especificidad$^2$. Habiendo cumplido
con las expectativas del primer año de monitoreo, además de
la implementación de programas de mejora, lo que sigue
es ampliar el número de indicadores y ajustar los estándares
con la nueva evidencia disponible. La expansión del monito-
reo de calidad solo puede redundar en beneficios sin requerir
grandes inversiones ni tecnología compleja.

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H1N1 influenza virus-associated encephalitis: A case report
Encefalitis asociada al virus de la gripe H1N1: un caso clínico

Febrile respiratory symptoms represent the most common
clinical manifestations of infection with 2009 H1N1 virus and
are in general mild and self-limited. Since the 2009
H1N1 pandemic several neurologic complications have been
described. Children and young adults are preferentially
affected. We report a case of H1N1-associated encephalitis
in an adult patient.

A 56-year-old male nurse, with a past medical history sig-
nificant for hypertension and right-sided nephrectomy for
congenital hydronephrosis, was admitted, initially to the
emergency department of another hospital, with a 5-day
history of influenza-like illness including lethargy, high fever
and nonproductive cough. A nasopharyngeal swab was per-
formed to test for the H1N1 virus.

On admission the patient was febrile, but otherwise his
other vital signs were stable. He was conscious and orien-
ted. The rest of the physical and neurologic examination was
unremarkable.

Laboratory studies revealed normal white blood cell
count, thrombocytopenia of $77 \times 10^9$/L and elevated C-
reactive protein of 15.8 mg/dL. Serum electrolytes and renal
and liver function tests were within normal limits. Chest
radiograph demonstrated consolidation of the left lower
lobe. He was diagnosed with community-acquired pneu-
monia and broad-spectrum antibiotic therapy consisted of
intravenous ceftriaxone and azithromycin was initiated.
On day 2 of hospitalization antiviral therapy with Oseltami-
vir 150 mg/day was associated after nasopharyngeal swab
confirmed H1N1 virus infection.

Despite antibiotic and antiviral therapies, his respiratory
status worsened. On day 3 he developed acute respiratory
distress syndrome requiring intubation and he was trans-
ferred to the intensive care unit.

Therapy with Oseltamivir was discontinued after 9 days.
Throughout his ICU-stay he remained febrile. After success-
ful weaning from mechanical ventilation and sedation the
patient was extubated on ICU-day 10. During the following
day’s he was noted to have fever, fluctuating mental status
and disorientation.

A computed tomography scan of the brain showed bila-
teral cortical and subcortical vasogenic cerebral edema
with areas of hemorrhage, involving the right fronto-
parietal lobe, the left occipital lobe and the left cerebellar
hemisphere, with mass effect on the left ventricle with
midline shift and subfalcial and right-sided uncal
 herniation.

The patient was put on antiedemic therapy and trans-
ferred to our institution for observation by neurosurgery.
Just before being transported he required reintubation for
Figure 1  The right-hemispheric lesion area. Brain magnetic resonance images showed extensive vasogenic edema with hemorrhagic foci in the right cerebral hemisphere predominantly in the right perirolandic and fronto-temporal regions with hyperintense signal. Following intravenous gadolinium administration leptomeningeal contrast enhancement in the right temporal lobe was observed.

<table>
<thead>
<tr>
<th>Author</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Interval ILI-neurologic symptoms (days)</th>
<th>Neurologic symptoms</th>
<th>CSF</th>
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<tbody>
<tr>
<td>Fugate et al.</td>
<td>Male</td>
<td>40</td>
<td>30</td>
<td>Acute drop on the bispectral index monitor</td>
<td>No pleocytosis Elevated protein level RT-PCR H1N1-ND</td>
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<tr>
<td>Akins et al.</td>
<td>Male</td>
<td>20</td>
<td>6</td>
<td>Confusion, seizures</td>
<td>Pleocytosis Elevated protein level RT-PCR H1N1 negative</td>
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<tr>
<td>Chen et al.</td>
<td>Male</td>
<td>40</td>
<td>2</td>
<td>Tremors, clumsiness, right hemiplegia</td>
<td>Pleocytosis Elevated protein level RT-PCR H1N1-ND</td>
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<td>Ito et al.</td>
<td>Male</td>
<td>26</td>
<td>Unknown</td>
<td>Memory disturbance, disorientation, drowsiness</td>
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<td>Gonzalez et al.</td>
<td>Female</td>
<td>46</td>
<td>3</td>
<td>Confusion</td>
<td>No pleocytosis Normal protein level RT-PCR H1N1 negative</td>
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<tr>
<td>Tsai et al.</td>
<td>Male</td>
<td>46</td>
<td>4</td>
<td>Acute delirium</td>
<td>No pleocytosis Normal protein level RT-PCR H1N1 negative</td>
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Author MRI EEG Antiviral therapy Prognosis

<table>
<thead>
<tr>
<th>Author</th>
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<th>EEG</th>
<th>Antiviral therapy</th>
<th>Prognosis</th>
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<tr>
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<td>Subcortical lesions with hemorrhages and edema</td>
<td>Normal</td>
<td>Oseltamivir</td>
<td>Severe sequelae</td>
</tr>
<tr>
<td>Akins et al.</td>
<td>White matter lesions, diffuse edema</td>
<td>Bilateral diffuse continuous slow δ waves</td>
<td>Oseltamivir 150 mg/dia</td>
<td>Mild sequelae</td>
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<tr>
<td>Chen et al.</td>
<td>Cortical and subcortical areas of the frontal-parietal lobe</td>
<td>Diffuse slowing of cortical activity</td>
<td>Oseltamivir</td>
<td>Severe sequelae</td>
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<tr>
<td>Ito et al.</td>
<td>Corpus callosum</td>
<td>Normal</td>
<td>Oseltamivir 150 mg/day</td>
<td>Complete recovery</td>
</tr>
<tr>
<td>Gonzalez et al.</td>
<td>Normal</td>
<td>ND</td>
<td>Oseltamivir 150 mg/day</td>
<td>Complete recovery</td>
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<tr>
<td>Tsai et al.</td>
<td>White matter lesions</td>
<td>Bilateral diffuse continuous slow δ waves</td>
<td>Oseltamivir 150 mg/day</td>
<td>Deceased</td>
</tr>
</tbody>
</table>

ND: not done; ILI: influenza-like illness; CSF: cerebrospinal fluid; EEG: electroencephalography.
Our patient almost died from influenza with neurological complications. The patient was admitted to our ICU on suspicion of H1N1-associated encephalitis. A lumbar puncture was performed. The CSF contained white blood cell count of <1.0/µL, no red blood cells, a normal glucose level and an increased protein level. Further work-up to exclude other possible causes of encephalitis included: (1) CSF polymerase chain reaction (PCR) for neurotropic virus was negative, including RT-PCR for 2009 H1N1 virus; (2) Cultures of blood, urine, tracheobronchial aspirate and CSF were negative; (3) Serology for mycoplasma pneumonia, Chlamydia, Rickettsia, hepatitis B and C, syphilis and HIV antibody was negative; (4) Testing for autoimmune disorders was within normal limits.

Brain magnetic resonance images (MRI) revealed extensive vasogenic edema with hemorrhagic foci in the right cerebral hemisphere with hyperintense signal lesions (T2 FLAIR) in left occipital lobe, left cerebellar hemisphere and bulbus. Following intravenous gadolinium administration leptomeningeal contrast enhancement in the right temporal lobe was observed (fig. 1). After discontinuation of sedation his level of consciousness gradually improved over the following days. Further intensive care course was uneventful and the patient was extubated on day 9. He was discharged on ICU-day 12, conscious, but still with periods of disorientation, and left sided hemiplegia. Almost complete recovery of his hemiplegia was noted one month after ICU discharge.

To the best of our knowledge, this is the first reported case of H1N1-associated encephalitis in an adult patient in Portugal. Although we were not able to identify the H1N1 virus by RT-PCR in the CSF as the causative agent, the combination of clinical and radiological findings and the exclusion of other competing diagnosis are, in our opinion, most consistent with this diagnosis. H1N1-associated encephalitis was defined by the Center of Disease Control and Prevention as altered mental status ≥24h, in patients with laboratory-confirmed H1N1 virus infection, within 5 days of influenza-like illness symptoms onset plus two or more of the following: fever, focal neurological signs, CSF pleocytosis, EEG and/or abnormal neuroimaging indicative of encephalitis. Our patient almost fulfilled all of these criteria. However it is noteworthy that neurological signs and symptoms were noted almost 20 days after the initial onset of respiratory illness when sedation was discontinued.

From a review of the English literature, we found one case of acute hemorrhagic leukoencephalitis and five adult cases of H1N1-associated encephalitis/encephalopathy (table 1).

All patients were previously healthy, aged between 20 and 46 years, five were male and one was female. The most frequent initial clinical manifestations were influenza-like symptoms. Neurological symptoms included drowsiness, memory disturbance, disorientation, confusion, tremors and focal signs starting between 1 and 6 days after onset of illness. All patients had a laboratory-confirmed (nasopharyngeal swab) H1N1 virus infection. However, like in our patient, H1N1 RNA was not detected in CSF by RT-PCR. Other findings of CSF included elevated leukocyte counts and/ or elevated protein levels. Neuroimaging findings were variable ranging from normal to cortical and subcortical lesions, like in our patient, to involvement of deep brain structures with or without brain edema. All patients were treated with Oseltamivir. Two patients received simultaneously treatment with corticosteroids. There was a complete recovery of neurologic manifestations in two patients; in three other patients mild to severe neurologic sequelae were noted.

In conclusion, encephalitis is a rare neurological complication of influenza H1N1 virus in adults. By publishing this case report we hope to contribute by the further characterization of this group of patients. H1N1-associated encephalitis must be considered in the differential diagnosis in patients with influenza-like illness and altered mental status. Diagnosis is based on neurological and neuroimaging findings, and CSF analysis in combination with laboratory-confirmed H1N1 respiratory tract infection.

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