Scientific letters

**Spontaneous subdural empyema by Escherichia coli: Case report and literature review**

Empiema subdural espontáneo por Escherichia coli: descripción de un caso y revisión de la literatura

Subdural empyema (SE) is defined as a purulent collection between the dura mater and arachnoid. The most common pathogenic mechanism is contiguous infection or cranial surgery, being extremely rare an spontaneous presentation, \(^{1,2}\) and the most common etiology is streptococci and staphylococci.\(^ {3}\) We present a case of spontaneous SE by *Escherichia coli*, including a critical literature review.

A 69-year-old man debuted with 48 h of 4/5 right hemiparesis and unsteady gait. He had a history of type 2 diabetes mellitus, dyslipidemia and dysuria, ten days before that was labeled as urinary tract infection (UTI) and treated with one dose of fosfomycin. Hemogram showed leukocytosis-neutrophils, elevated C-reactive protein (206 mg/L). Unenhanced-CT showed a hypodense left frontoparietal subdural collection (30 mm) and midline shift (10 mm). On suspicion of chronic subdural hematoma (CSDH), two burr holes were performed, after durotomy, abundant purulent material was obtained, so we made a minicraniotomy to complete the evacuation. Empiric therapy was started with vancomycin (1 g/8 h), metronidazole (500 mg/8 h) and cefotaxime (2 g/8 h); Gram stain was identified Gram-negative bacilli, so vancomycin was suspended, continuing rest of therapy for 5 days until the arrival of culture. *E. coli* was isolated sensitive to cefotaxime, increasing dose to 2 g/6 h. The patient presented fever the sixth day, and a 14 mm ring enhanced collection was found on the control CT, so a new surgical revision with larger cranietomy was done for evacuation of purulent, and increase of cefotaxime to 4.5 g/6 h (200 mg/kg [90 kg]) until 6 weeks. The patient improved until complete recovery, brain MRI after 27 days showed minimal collection. The urine and blood cultures were negative.

SE has a high mortality rate and is associated with severe neurological disabilities. The most common cause is meningitis in children and adjacent infections in adults.\(^ {3-5}\) Neurological focal symptoms, leukocytosis and elevated inflammatory reactants are typical. CT and MRI show a subdural collection with contrast ring enhancement, and a restricted diffusion in the diffusion-weighted imaging.

On extensive search in the literature, only 9 cases were found (Table 1): age ranged from 5 months to 91 years, no gender predilection. Six cases presented previous infections, four UTI\(^ {1,6-8}\) and two meningitis.\(^ {2,9}\) They had history of CSDH (20%) and diabetes.

**Table 1**

Summary of previous cases of spontaneous SE by *Escherichia coli*.

<table>
<thead>
<tr>
<th>Case/ref.</th>
<th>Age/sex</th>
<th>Symptoms</th>
<th>Site</th>
<th>Surgery</th>
<th>Recurrence</th>
<th>Outcome</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Kaminogo(^ {2})</td>
<td>76 y/F</td>
<td>Headache</td>
<td>B</td>
<td>NA</td>
<td>NA</td>
<td>Satisfactory</td>
<td>UTI by <em>E. coli</em></td>
</tr>
<tr>
<td>2. Bakker(^ {1})</td>
<td>88 y/F</td>
<td>Alt.MS, paresis</td>
<td>L</td>
<td>Burr holes</td>
<td>No</td>
<td>Death</td>
<td>UTI by <em>E. coli</em>.</td>
</tr>
<tr>
<td>3. Miedema(^ {2})</td>
<td>7 m/F</td>
<td>Fever</td>
<td>R</td>
<td>NA</td>
<td>NA</td>
<td>Satisfactory</td>
<td>Blood and CSF culture positive for <em>E. coli</em>.</td>
</tr>
<tr>
<td>4. Fender(^ {10})</td>
<td>5 m/M</td>
<td>Irritability, increased HC</td>
<td>B</td>
<td>Burr holes/B Burr holes</td>
<td>Yes, Cranietomy/L</td>
<td>Satisfactory</td>
<td>PKD, chronic UTI by <em>E. coli</em>.</td>
</tr>
<tr>
<td>6. Bachmeyer(^ {9})</td>
<td>55 y/M</td>
<td>Fever, Alt.MS</td>
<td>B</td>
<td>NA</td>
<td>NA</td>
<td>Death</td>
<td>DM, evacuation CSDH/L 4 months ago. CT: pneumocephalus. UTI by <em>E. coli</em>.</td>
</tr>
<tr>
<td>7. Adamides(^ {2})</td>
<td>91 y/M</td>
<td>Alt.MS, paresis</td>
<td>L</td>
<td>Cranietomy</td>
<td>No</td>
<td>Death</td>
<td>DM, evacuation CSDH/B 15 days ago. CT: Tension pneumocephalus.</td>
</tr>
<tr>
<td>8. Yoon(^ {4})</td>
<td>79 y/F</td>
<td>Fever, paresis</td>
<td>L</td>
<td>Burr holes/Cranietomy</td>
<td>No</td>
<td>Satisfactory</td>
<td>DM.</td>
</tr>
<tr>
<td>9. Redhu(^ {5})</td>
<td>48 y/M</td>
<td>Fever, Alt.MS, Headache</td>
<td>L</td>
<td>Cranietomy</td>
<td>No</td>
<td>Satisfactory</td>
<td>DM.</td>
</tr>
<tr>
<td>10. Our case</td>
<td>69 y/M</td>
<td>DYSarthria, paresis</td>
<td>L</td>
<td>Cranietomy</td>
<td>Yes, Cranietomy</td>
<td>Satisfactory</td>
<td>DM.</td>
</tr>
</tbody>
</table>

mellitus (20%). The most common symptoms were altered mental status and fever (50% each), followed by paresis and headache. Pneumocephalus was observed in 30% at the moment of diagnosis and it has been associated with a worse prognosis.5,7,8 The surgical treatments were burr holes (40%) and craniotomy (30%). Two cases presented recurrences (minicraniotomy 1, burr hole 1), forcing an expanded craniotomy. The mortality was 40%.

Hematogenous spread seems to be the pathophysiological mechanism of spontaneous SE. In 40% of cases presented a distant infection with same germ.1-4 Other risk factors for overt infections could be previous CSDH,5-7 diabetes mellitus and immunosuppression states.5,8 In our case, we relate the genesis of spontaneous SE to hematogenous spread of an unconfirmed UTI, in association with diabetes mellitus.

The treatment of choice is surgical evacuation associated to culture adjusted intravenous antibiotics during 4–6 weeks.4,9 The most appropriate surgical approach is still under discussion, as some authors argue that the burr holes are sufficient for evacuation; others suggest that wider exposure as a craniotomy is more effective.7 Yilmaz et al.3 presented a lower recurrence rate on craniotomies (10%) vs. burr holes (38%). In cases with difficult pus extraction, as in multiloculated collections,5 parafalcine location1 and recurrences, is better to carry out a wide craniotomy. Both surgical techniques must perform thorough washing until clear liquid outlet, take care to remove the adherent material in the cortex for its lesion risk; placement of subdural drainage can be left up to 72 h. In our case, the realization of minicraniotomy does not allow an extensive cavity wash, and the initial treatment with cefotaxime (2 g/8 h) was with low dose; these may favor the recurrence.

In conclusion, the spontaneous SE should be suspected in patients with fever and hypodense subdural collections in the head CT, and the administration of intravenous contrast may to increase its sensitivity. To achieve a favorable outcome must make an early surgery and begin antibiotic therapy. It seems that the craniotomy is associated with a lower relapse rate, however more studies are needed to confirm this fact.

References


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Autochthonous Cryptosporidium cuniculus infection in Spain: First report in a symptomatic paediatric patient from Madrid

Infección autóctona por Cryptosporidium cuniculus en España: primer informe en un paciente pediátrico sintomático del área de Madrid

Human cryptosporidiosis, an infection caused by protozoan of the genus Cryptosporidium, is a major cause of gastrointestinal disease worldwide including Spain.1 Most human cases are attributed to C. hominis and C. parvum, although infections with zoonotic C. meleagrisidis, C. canis, C. felis, and a number of rare genotypes are also sporadically documented.2 Among these, Cryptosporidium cuniculus (previously known as the Cryptosporidium rabbit genotype) was first identified in an adult female rabbit in 19793 and fully re-described and characterized in 2010.4 Since then, very few cases of human infections by C. cuniculus have been identified in UK (n = 37),5 Nigeria (n = 5),6 Australia (n = 1),7 and France (n = 1).8

In May 2015 a 7-year-old female child complaining of gastrointestinal symptoms including acute, non-bloody watery diarrhoea and abdominal pain, was admitted to the outpatient clinic of the University Hospital Puerta de Hierro Majadahonda (Madrid) for routine coproparasitological examination. The patient had a normal immune status and no relevant record of recent travelling abroad. Information regarding contact with pet or wild rabbits was unavailable. A single, concentrated stool sample tested positive for the presence of Cryptosporidium oocysts by a commercial immunochromatographic test (Cer Test Biotec S.L., Zaragoza, Spain) and by microscopic examination of a fresh faecal smear stained with the modified Ziehl–Neelsen method.

A new aliquot of the faecal material was sent to the National Centre for Microbiology at Majadahonda (Madrid) for genotyping analyses. Total DNA was extracted and purified using the QIAamp® DNA stool mini test kit (Qiagen, Hilden, Germany). Identification and molecular characterization of the obtained isolate was conducted by multiplexing (involving PCR-based protocols and DNA sequence analyses) of the small-subunit ribosomal RNA (SSU rRNA)9 and the 60-kDa glycoprotein (GP60)10 genes of Cryptosporidium. Both SSU rRNA-PCR and GP60-PCR assays generated the expected amplicons of ~587 bp and ~870 bp, respectively. Sequence analysis of the corresponding PCR products confirmed the presence of C. cuniculus, allowing the assignment of this isolate to the sub-genotype VbA34 of the parasite. As expected, phylogenetic analysis at the GP60 locus between the sequence from this study and reference sequences previously deposited in GenBank placed our isolate into a well-defined allelic group (family Vb), while other representative sub-genotypes of C. cuniculus clustered together in a separate allelic group (family Va) (Fig. 1). The