parole, conscience_). Il n'y aucun substrat organique et une origine psycho-affective est fortement suspectée.

AIM DISCUSSION Un tableau de perte de fonction aiguë chez des patients pédiatriques amène les parents à consulter un médecin en urgences. Quels sont les éléments pouvant faire évoquer le diagnostic de trouble de conversion? Jusqu'où est-il nécessaire de pousser le bilan d'un point de vue strictement médical et somatique? Quand l'approche psychologique doit-elle être introduite et comment?

METHOD Nous allons passer en revue rétrospectivement quatre cas cliniques de patientes admis dans notre service de pédiatrie par le biais des urgences. Les cas concernent des patientes âgées de 10 à 14 ans ayant présenté des symptômes neurologiques aigus variant de la perte d'usage d'un membre à des céphalées avec troubles de la conscience fluctuants. Elles ont été hospitalisées pour la réalisation d'un bilan complémentaire. Au terme des bilans spécialisés négatifs, nous avons pu introduire de manière progressive l'origine psychosomatique de la symptomatologie et la nécessité d'une prise en charge psychologique. L'évolution à court terme montrait une amélioration spectaculaire dans deux des cas et plus contrastée dans les deux autres.

RESULTS Dans tous les cas, nous avons pu mettre en évidence que 1) L'examen neurologique répété échouait à définir un trouble organique systématisé. 2) L'hospitalisation était chaque fois nécessaire le temps de la mise au point. 3) Des examens complémentaires exhaustifs ont été réalisés biologies, toxicologies, EEG, imageries ; et se sont révélés négatifs 4) L'anamnèse rapportait des facteurs de stress psychoaffectifs importants. 5) Une discordance persistait entre l'inquiétude parentale face à la perte de fonction et l'indifférence de l'enfant.

CONCLUSION Le trouble de conversion existe chez l'enfant, il est peu courant mais très invalidant et concerne le plus souvent la perte d'usage d'un membre. Il reste un diagnostic d'exclusion. Devant une telle symptomatologie, l'hospitalisation est nécessaire avec mise au point neurologique complète. Après exclusion des causes organiques, l'origine psycho-affective doit être évoquée. Il est alors important d'arrêter les bilans complémentaires et la surenchère du traitement pour ne pas renforcer le symptôme. Il faut ensuite réorienter le patient vers un suivi pluridisciplinaire plus approprié. La prise en charge thérapeutique consiste en thérapies cognitivo-comportementales ou familiales qui ont prouvé leur efficacité.

O208 / Poster • Schizencephaly associated with a severe prothrombotic syndrome caused by antithombin III deficiency.

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INTRODUCTION A 12 year old boy presented with non syndromic mental retardation. Brain MRI showed schizencephaly with a deep right parietooccipital cleft extending from cortical surface to the occipital horn of cerebral ventricle. Moreover his family was known for antithrombin III deficiency linked to the homozygous c.391C>T (p.Leu131Phe) mutation in SERPINC1. Coagulation studies revealed in this young boy severe antithrombin III deficiency and molecular analysis confimed the mutation.

RESULTS Schizencephaly can be considered as cerebral malformation of neuronal migration, caused by mutations in several transcription factors, but most cases occur sporadically and are believed to be associated with a vascular disruptive mechanism. In the proband, analysis of the SHH, SIX3 and EMX2 genes showed no mutation. Porencephaly and schizencephaly have also been attributed to mutations of COL4A1, linking, as in our case, genetic vascular pathology with schizencephaly. Otherwise mutations of methylterahydrofolate reductase and factor V Leiden genes seems also responsible of cases of schizencephaly. We suggest that a similar encephaloclastic mechanism took place in our patient and hypothesize the occurrence of an early antenatal cerebral vascular injury.

CONCLUSION Extensive coagulation studies should be performed in patients with schizencephaly before molecular analysis.

case of a young girl who presented a Lyme neuroborreliosis manifested as meningoradiculitis, occurring one month after tick bites.

RESULTS An 8 year-old Caucasian girl with no previous medical history presented to the emergency department for disabling back pain since 10 days, causing restlessness. A thorough history pointed out three insect bites one month prior to presentation. Two of them fell on their own, while the third one was removed on the same day. No skin manifestations or fever were noticed following the bites. Clinical examination revealed neck stiffness with back pain. The latter was localised from C6 to T4 spinous processes, irradiating to both the left and the right axillary region. Laboratory workup showed no inflammation, but serologies revealed elevated Lyme total antibody by enzyme immunoassay (ELISA). Western blotting for Lyme disease confirmed the positive ELISA result. Cerebral and cervicothoracic magnetic resonance imaging performed prior to cerebrospinal fluid (CSF) sampling demonstrated a leptomeningeal enhancement from C4-C5 to T4-T5 as well as signs of meningitis. CSF analysis showed 435/µl white blood cells, with predominantly lymphocytes (91%), 45 mg/dl glucose (serum 84 mg/dl) and 0,98 g/l proteins. Moreover other CSF studies revealed elevated Borrelious antibody (IgG 171,8 UA/ml), no malignant cells, suspicion of borrelial DNA from polymerase chain reaction investigations and absence of tuberculous meningitidis. Rapid improvement of the clinical course was obtained after the introduction of intravascular ceftriaxone treatment. The follow-up was excellent with no neurological sequelae.

CONCLUSION Early CNS implication in Lyme disease could occur within 3 to 4 weeks after tick bites. It may be the lonely abnormality of Lyme disease without skin manifestations. Its rate varies from 3% in the USA up to 35% in the European countries. Differential diagnosis needs to be evoked in order to rule out other causes of aseptic meningitidis, especially tuberculosis, as well as oncological aetiologies. According to the literature, the risk of Lyme meningitidis is evaluated to 50% in case of headache lasting for ≥7 days and CSF mononuclear cells ≥70%. This rate rises up to more than 75% when cranial nerve involvement is present. In general, the disease's manifestations are similar in both children and adults, excepted for meningo-poly-radiculoneuritis that is rarely seen in children. This case report points out the fact that radiculoneuritis, although unusual, may be observed in children.

O235 / Poster • Diagnostic pitfall fever, vomiting and headache in an eleven-year old boy.

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INTRODUCTION Brain abscess is an uncommon infection in children with a high morbidity and mortality rate. We report a case of a boy with an epidural abscess and a cerebritis which converted into a brain abscess.

RESULTS Case description An 11-year old boy was hospitalized with following symptoms 10 days of frontotemporal headache, 7 days of fever, sporadic vomiting, anorexia and a weight-loss of 4 kg. Initial clinical examination showed nuchal rigidity. Repeated re-evaluation the same day showed no rigidity. Blood examination showed increased sedimentation (70 mm), a leucocytosis (14,9 10*9/l) with left shift, an increased CRP (307 mg/l) and transaminases (ALT 119 U/l, AST 126 U/l). Several differential diagnosis were proposed, but as the general condition remained stable an no definitive diagnosis was made, no treatment was started. The following days he developed fever and chills with increasing headache and vomiting. On day 5 we found nuchal rigidity. An ophthalmoscopy showed papilledema. A CT scan revealed an epidural abscess and cerebritis of the right frontal lobe with an accompanying frontal sinusitis. The boy underwent a trepanation with drainage and functional endoscopic sinus surgery (FESS). Pus culture isolated Streptococcus anginosus and Staphylococcus aureus. He was treated with oral metronidazole and intravenous cefotaxime and flucloxacillin during 6 weeks. Prophylactic treatment with levetiracetam was given during 10 weeks. On day 17 he developed symptoms of intracranial hypertension. A CT scan revealed a brain abscess in the right frontal lobe with a significant mass affect. A second trepanation and FESS occurred. 2,5 months after the diagnosis an NMR scan revealed an important regression of the infection. The boy recovered well there were no personality changes and 2,5 months after diagnosis he returned to school. Discussion A brain abscess can originate from a pericranial contiguous site infection, from a hematogenous spread of a distant focus of infection, after head trauma or neurosurgery or from cryptogenic sources. Streptococci are the most common pathogens in children. Multiple organisms are isolated in 1/3 of the patients. The clinical presentation of a brain abscess depends on the size and location of the collection, the multiplicity of lesions, the immune status and age of the patient. On early evaluation children can present with vague and nonspecific symptoms. Later on the course children can have symptoms of intracranial hypertension, focal neurological deficits or fever. Only 15% of the patients have nuchal rigidity. A lumbar puncture is never recommended. Both a CT scan with contrast and NMR scan can provide valuable information for the diagnosis and response to treatment. The management requires a multidisciplinary approach and might include antimicrobial therapy, surgical intervention, anticonvulsants and rehabilitation interventions.

CONCLUSION Brain abscess is an uncommon infection. Early diagnosis is important to reduce morbidity and mortality rates.