An unusual digestive onset of Henoch-Schonlein purpura in an adult

Une forme inhabituelle de Maladie de Henoch-Schonlein inaugurée par un tableau digestif chez l'adulte.

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Introduction

Henoch-Schonlein purpura (HSP) is a leucocytoclastic vasculitis involving small vessels with
IgA immune complexes deposit that targets multiple organs. Compared to children, HSP is less common in adults and although frequent, gastro intestinal symptoms rarely reveal the diagnosis (1). We report the case of a 25-year-old woman in whom HSP was revealed by an acute right-sided abdominal pain with fever mimicking ileal Crohn's disease (CD).

Observation

It's the case of a 25-year-old woman suffering from right-sided inferior abdominal quadrant pain and vomiting that held 72h ago. She reported an acute upper respiratory tract infection that spontaneously resolved three weeks later. Physical examination revealed right abdominal tenderness with fever 38°C. Body mass index was 16kg/m2. The patient had increased leucocytes (21130/mm3) and platelets counts (605000/mm3) as well as C reactive protein (CRP) level (171 mg/l). She also had microcytic anemia (hemoglobin=9.1 g/l, VGM=67.9u3) associates with other biological signs of malabsorption, hypoalbuminemia (22g/l), hypocalcemia (85mg/l) and hypocholesterolemia (0.57 g/l). Hepatic enzymes as well as renal function were normal. Quantiferon test was negative.
Abdominal tomodensitometry showed terminal ileitis with multiple adenomegalies in the right inferior abdominal quadrant as well as a localised mesenteric thickening. However colonoscopy was normal while terminal ileal could not been examinated. Upper endoscopy showed diffuse duodenal erythema and superficial ulcerations. Histological examination revealed intense inflammation of the chorion with no architectural alterations and could not been conclusive. Infectious terminal ileitis was suspected as well as a Crohn's disease with an acute onset. An intravenous antibiotics course (cefotaxim and metronidazole) was initiated with no clinical or biological significant improvement. One week later, the patient developed bilateral and symmetric erythema with purpuric lesions on the lower legs and feet. Urine testing (Labstix) revealed hematuria and proteinuria with a 24h proteinuria of 2.11 g/l but normal creatinine level. Cutaneous biopsies revealed leucocytoclastic vasculitis with intense IgA and IgM deposit on direct immunofluorescence and renal biopsies mesangial proliferative glomerulonephritis with mesangial IgA deposit on immunofluorescence examination (figure1), thus leading to HSP diagnosis. Clinical and biological evolution was spontaneously favorable within the two next weeks with normal tomodensitometry two months later. The patient remained asymptomatic during 2 years of follow up.

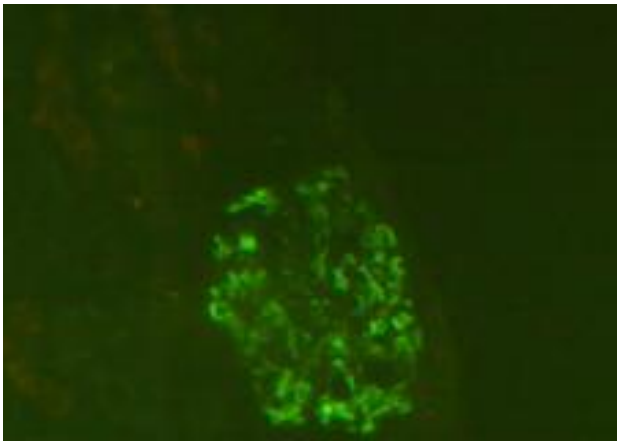

Figure 1: Mesangial Deposit of Ig A on electronic microscope.

Commentaries

Although frequent, gastro-intestinal manifestations rarely reveal HSP and an acute terminal ileitis presentation mimicking CD preceding cutaneous and/or renal manifestations is exceptional (1). We described the case of a young woman in whom the diagnosis was revealed by an acute febrile illness of ileo-ceacal associated to biological signs of malabsorption, terminal ileitis with mesenteric thickening on tomodensitometry and proximal duodenitis that preceded onset of cutaneous purpuric lesions and proteinuria. Renal biopsy led to HSP diagnosis.
SHP should be taken in to consideration as a differential diagnosis of Crohn's disease. Seldom
cases were already described enhacing diagnosis similarities between SHP and terminal ileal CD with cutaneous manifestations occurring after abdominal presentation (2-4). HSP has an unknown etiology so far but is often seen 2–3 weeks after an upper respiratory tract infection as it was the case of our patient. Infectious agents are among the common etiological suspected factors, as well as exposing to cold, vaccines, insect bite and drugs. HSP generally recovered spontaneously and doesn't require special treatment. In conclusion, HSP rarely presents with acute abdominal findings and can

mimic CD particularly when digestive manifestations precede onset of renal and/or skin expression.

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Penile foreskin trapped in a zipper: what to do?

Le prépuce coincé dans une fermeture à glissière: Quoi faire ?

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Introduction

Despite the fact that the penis is well hidden by trousers or pants, penile injuries may happen.

They remain relatively uncommon (1). This innocuous device may damage parts it is designed to protect. It is believed to account for the majority of penile injuries in children (2). During the process of zipping or unzipping, redundant skin of the penile foreskin can be caught in both the teeth of the zipper and the zipper slider (3) (Figure 1). Two types of zip entrapment were seen. In the most common type, the foreskin was caught by the slider body of the zipper. Less commonly, the foreskin was caught simply between the teeth of the zipper, the slider body having been pulled past and beyond (4).

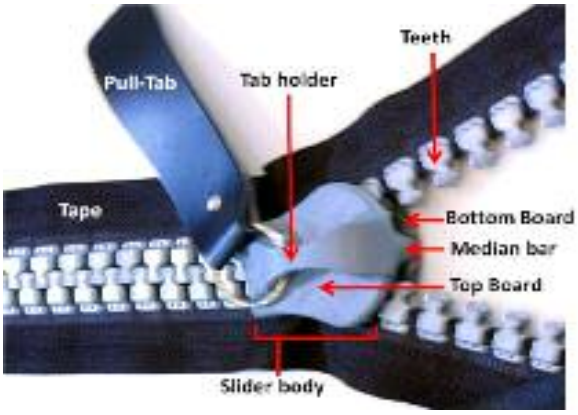

Figure 1: Structure of a zipper.

Entrapment of the prepuce or penis is a very distressing situation for the child and the parents obviously related to urinary, fertility and sexual function of the penis.

The approach to the zipper manipulation should be quick, simple and non-traumatic. It should be reproducible independently of the age of the child, entrapment mechanism, lesion site and zipper size or design (5). Any overzealous intervention might worsen the situation.

Several management strategies were reported in the literature, ranging from dismantling the zipper with bone or wire cutters to circumcision under general anesthesia (2,4). The most obvious disadvantage of this latter procedure is that is only suitable for the uncircumcised child with an injury to the prepuce. However, this may not be the best option as it necessitates a general anesthetic in addition to conferring its own operative risk (6). It may also be unacceptable for various social/cultural reasons but not in our context. Herein, we report a case of penile foreskin (prepuce) entrapment in the pant's zipper.

Through this case report; circumstances and treatment of children presenting with zipper injury to the penis were investigated.

Case report

A 5-year-old uncircumcised-boy presented to the emergency unit about 40 min after accidentally trapping the ventral surface of the penis in the zipper of his jeans. The incident happened whilst the mother's child was helping him to wear his trousers while standing. She was fastening the zipper upwards.

The child immediately experienced intense pain and discomfort in the penis. After a number attempts to free the penis, the parents had cut the pant off the zipper mechanism and attend the emergency.

The boy was very distressed and frightened, at presentation, due to several unsuccessful and painful attempts at extrication by the parents. Approximately 5 mm of penile foreskin was trapped in the zipper mechanism at the midpoint of the ventral prepuce portion (Figure 2).

Figure 2: A prepuce entrapment in the zipper

There was only very slight bleeding from the involved skin and minimal oedema. There was no injury to the scrotum nor other parts of the body and the chid passed urine freely.

Under local anesthesia (EMLA cream), numerous

attempts at removing the zipper with lubricant has failed. The patient was becoming increasingly anxious and understandably less cooperative. Thus he was surgically treated under sedation. After parent's agreement, he underwent circumcision. The penal glans and urethral meatus were normal.
He received anti-tetanus prophylaxis and analgesics and he was discharged few hours after.

Conclusion
Penile entrapment in a zipper may be seen in any emergency department. Medical personnel, especially in pediatric emergencies must be aware that zipper-entrapped penis is a problem that requires immediate, adequate care and avoid empirical treatments.

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Le fibrome chondromyxoïde : A propos d'une observation

Chondromyxoid fibroma : A case report

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Introduction
Initialement décrit par Jaffe et Lichtenstein en 1948, le fibrome chondromyxoide est une tumeur cartilagineuse bénigne rare représentant 1% de toutes les tumeurs osseuses [1,2]. Elle survient généralement chez l'adulte jeune à un âge médian de 31 ans avec une prédominance masculine (sexe ratio 2/1) néanmoins l'âge des malades peut varier de 3 à 70 ans [3, 4]. Il est généralement admis que cette tumeur se développe aux dépens de la face diaphysaire du cartilage de croissance. Nous rapportons l'observation d'un fibrome chondromyxoïde chez un jeune garçon de 13 ans.

Observation
Il s'agissait d'un patient âgé de 13 ans qui présente une tuméfaction douloureuse du genou, post-traumatique, rebelle aux antalgiques et aux anti-inflammatoires, répondant radiologiquement à une lésion lytique, bien limitée de l'extrémité supérieure du tibia. Une biopsie a été réalisée montrant à l'examen anatomopathologique, une prolifération tumorale d'allure bénigne associant trois contingents tissulaires, fibreux, myxoide et cartilagineux(Figure 1). La densité cellulaire était pour la plupart très lâche avec toutefois, des foyers plus denses en périphérie (Figure 2). Les territoires lâches étaient composés de cellules fusiformes, étoilées, à cytoplasme dense et à noyau allongé ou ovoïde, discrètement atypique, sans aucune figure de mitose. Ces éléments étaient disposés au sein d'une trame myxoide, (Figure 2) étroitement mêlés à un tissu cartilagineux avec des logettes uni ou bicellulaire comportant des chondrocytes sans atypies. En périphérie, dans les territoires densément cellulaires, les éléments tumoraux étaient arrondis, à limites cytoplasmiques non définies et à noyaux ronds, munis d'une chromatine fine sans mitoses. A ce niveau, il existait quelques remaniements inflammatoires et hémorragiques avec des sidérophages associés à de rares cellules géantes ostéoclastiques Le diagnostic de fibrome chondromyxoide a été retenu.

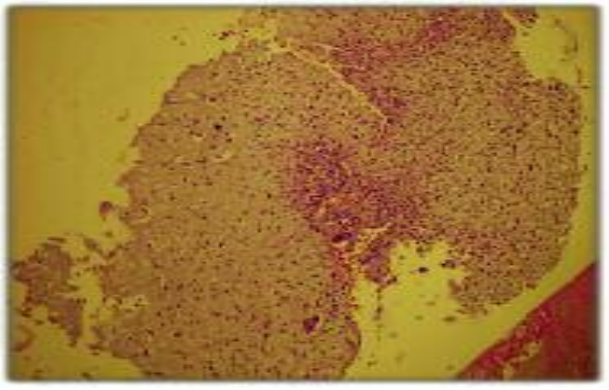

Figure 1 (HEx200):coexistence de contingent tissulaire myxoïde, fibroblastique et cartilagineuxfibroblastique et cartilagineux

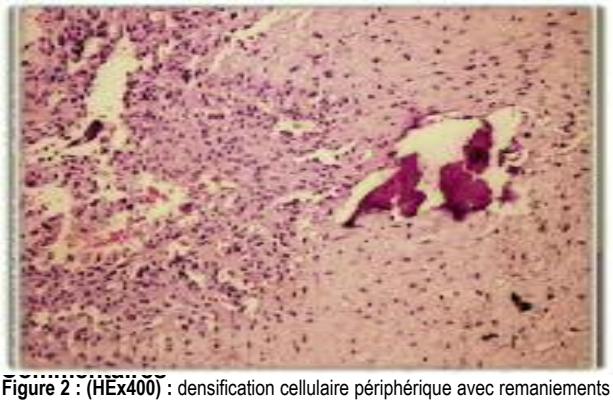

Figure 2 : (HEx400) : densification cellulaire périphérique avec remaniements hémorragiques et calcifications

Le fibrome chondromyxoïde est une tumeur bénigne des plus rares du tissu osseux, d'histogenèse cartilagineuse, caractérisée par l'association de lobules chondroïdes, de zones fibreuses et de plages myxoïdes. Cette tumeur peut être observée à tous les âges et dans tous les os, essentiellement dans les métaphyses des os longs (45 à 66%), surtout dans le tibia proximal et le fémur distal [3,5]. Le signe révélateur habituel est une douleur peu intense, parfois une tuméfaction [4]. La radiologie conventionnelle montre une image ostéolytique métaphysaire excentrique ovoïde à grand axe parallèle à l'axe de l'os et cernée par un fin liseré de sclérose [3,4]. Son diagnostic positif est essentiellement histologique, et doit être nécessairement corrélé aux données radio-cliniques. Il est généralement aisé par la présence de cette triple association tissulaire de territoires fibreux, myxoïdes et cartilagineux. Dans le cas contraire, il peut poser des difficultés diagnostiques avec le chondrosarcome myxoïde en présence d'un pléomorphisme cellulaire qui est assez fréquent surtout en périphérie des lobules myxoïdes, avec présence de noyaux hyperchromatiques, volumineux et irréguliers. Toutefois, la rareté des mitoses et l'absence de nécrose de liquéfaction sont en faveur d'un pronostic bénin, éliminant un éventuel chondrosarcome. La principale caractéristique évolutive du fibrome chondromyxoïde est le risque de récidive après traitement, et en particulier après curetage intra-lésionnel [1, 2, 3, 4, 5].

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An unusual localization of mediastinal hydatid cyst mimicking diaphragmatic tumour

Localisation inhabituelle d'un Kyste hydatique médiatisnal mimant une tumeur diaphragmatique

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Introduction:

Intrathoracic extrapulmonary hydatid cyst (HC) is very uncommon, but should be considered in the differential diagnosis of mediastinal lesions [1,2]. We report a case of an intrathoracic extrapulmonary HC mimicking diaphragmatic tumour which the precise location was confirmed during surgical intervention.

Case report:

A 40-year-old male smoker with a past medical history of right pulmonary hydatid cyst (HC) surgery 30 years ago was admitted with a 3 month history of chest pain. His physical examination was normal. Chest radiography revealed right-sided opacity (Figure 1).

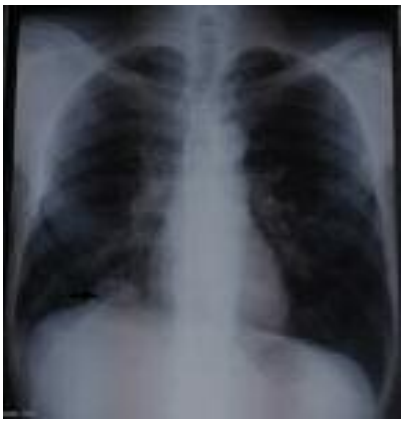

Figure 1: Anteroposterior chest radiography showing right sided opacity adjacent to diaphragm (black arrow).

The results of laboratory tests showed eosinophilia. Serum serology for hydatid disease by ELISA test was negative. Thoraco-abdominal computed tomography (CT) revealed an intrathoracic extra parenchymal mass lesion measuring 51x37x24 mm, neighboring the diaphragm with central calcifications and without enhancement after injection of contrast materiel (Figure 2). The appearance of the liver, spleen, gall bladder and pelvic organs was normal. Fiberoptic bronchoscopy was negative. Thoracic magnetic resonance imaging (MRI) showed a mass with heterogeneous hyposignal T1 and T2 with central calcifications and enhancement after injection of the gadolinium, it was separated from the diaphragm by a greasy and regular border (Figure 3). Right thoracotomy was performed. On exploration, a calcified extra parenchymal cystic lesion measuring 5 cm, located between lung parenchyma and diaphragm, adhered to the phrenic nerve was identified. Total cyst excision including a margin of normal diaphragm was performed. The histopathological examination was compatible with HC,

demonstrating an outer cyst wall composed of acellular hyalinised fibrous tissue with focal calcifications. Multiples ectocysts of the parasite echinoccocus granulosus in the form of laminated membranes were seen (figure 4). The patient received complementary medical treatment with albendazole for 6 months. On follow-up period (36 months), no complication or recurrence was seen.

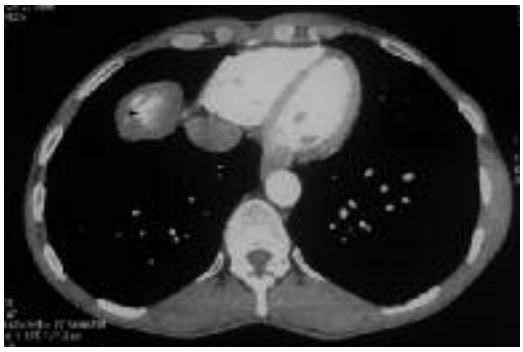

Figure 2: Axial Chest CT view with contrast material showing an intrathoracic extra parenchymal mass lesion neighboring the diaphragm with central coarse calcifications (arrowhead).

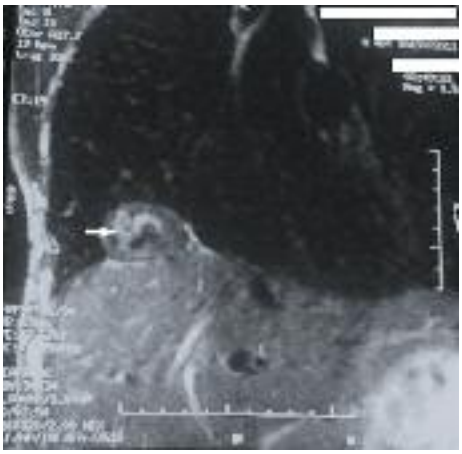

Figure 3: Coronal T1-weighted magnetic resonance image showing hypointense heterogeneous mass with central calcifications, independent of the diaphragm (white arrow).

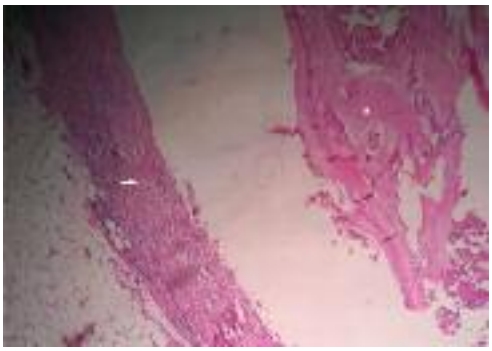

Figure 4: Histopathologic section showing cystic fibrous wall (white arrow) containing hydatid membrane (white star) (HEx200)

Commentaries

Intrathoracic extra pulmonary hydatid cysts are very uncommon (5-7%) even in endemic areas and are found in the chest wall, mediastinum, pericardium, myocardium, pleural space and diaphragm [1-4]. This rarity may cause difficulties in diagnosis which can be confusing with other diseases such as bronchogenic cyst, enteric cyst, and pleural, diaphragmatic and neurogenic tumors [5]. Since the lesion seems to be arising from diaphragm, a diaphragmatic tumour was suspected first in this case. The pathogenesis of mediastinal HC remains controversial. HC could come from fissuring hydatid liver or lung cyst into the systemic circulation, allowing the parasite to settle in the mediastinum or from transdiaphragmatic dissemination in the mediastinum or via lymphatic abdominal hydatidosis. In our case, postoperative dissemination in the mediastinum from the pulmonary cyst is the most plausible mechanism. CT is providing information about the morphologic characteristics and the localization of the cyst in the thorax, showing the water density in intact cysts [3, 6]. However, the increased density in complex cysts can be confused with mass lesions. It may also show multivesicular structure and calcifications characteristic of HC [1, 7]. MRI is superior to CT in localisation and relationship to adjacent structures, specifically spinal, cardiac and diaphragmatic involvement as well as characterization of the mediastinal HC [8]. The precise location of an intrathoracic extra pulmonary HC is usually confirmed during surgical intervention. Cystectomy and resection of the adjacent pericystic structures is the curative treatment of HC [2, 8-10]. Taking maximal precautions during the procedure can only reduce the risk of perforation and dissemination. Additional anthelmintic medical regimen postoperatively may avoid recurrence [3, 8].

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