Adult Henoch-Schonlein purpura and pulmonary tuberculosis

Purpura rhumatoide de Henoch-Schonlein de l'adulte et tuberculose pulmonaire

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Rheumatoid purpura named also Henoch-Schonlein Purpura (HSP) is a small vessel vasculitis characterized by immunoglobulin A (Ig A) deposition (1). It's a common childhood vasculitis but rarely occurs in adult age (2, 3). The causes of this disease still unclear. A past history of respiratory infection is often described (4). We report the case of HSP associated with pulmonary tuberculosis in adult and we discuss the mechanisms of this combined disease.

Case report

A 29-old man known case of type I diabetes mellitus on insulin since five years, admitted to pulmonology department because of one month history of mild recurrent hemoptysis with night sweats and loss of weight without any contact with TB patient . No extra-thoracic symptoms.

On physical examination, he is average body weight, afebrile, vitally stable. No lymph node enlargement. The chest was clear and the rest of examination is unremarkable.

Laboratory investigations revealed that hemoglobin was 12 g/dl, white blood cell count was 9600/mm3, the platelet count was 269.000/mm3 and creatinine was normal. Urine analysis showed no hematuria and proteinuria.

Chest radiography (Figure 1) showed a left para-hilar cavitary lesion.

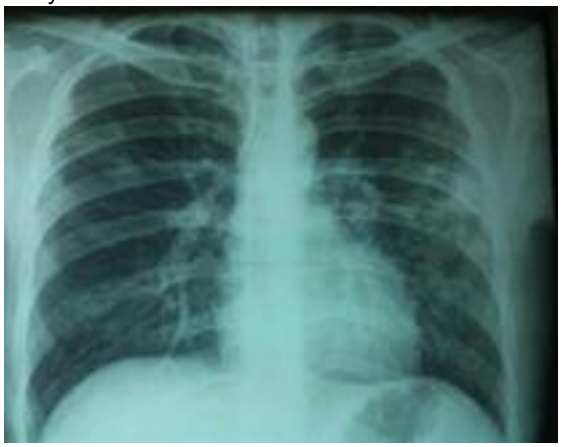

Figure 1: Chest radiography: left para-hilar cavitary lesion

Acid fast bacilli in the sputum was positive and the patient started on fixed dose combination anti- TB treatment according to body weight (4 tablets of HRZE).

Three days after taking the anti-TB treatment, the patient developed skin lesions with purpuric and necrotic rush coalesced resulting in bullous on his ankles and elbows (Figure 2) associated with arthralgia of the both knees.

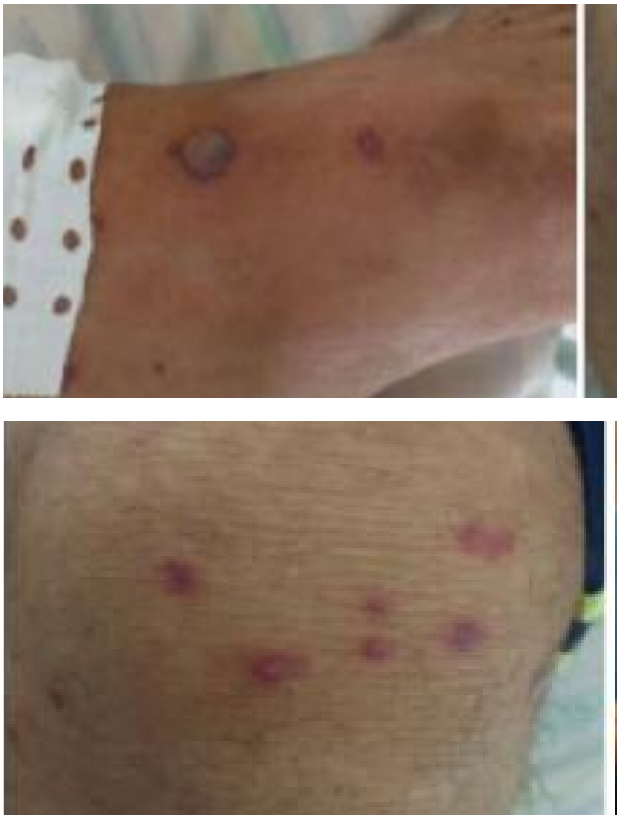

Figure 2: Purpuric and necrotic rush on ankles and elbows

Blood biochemistry and urine analysis were normal. A cutaneous reaction to anti-tuberculosis treatment was suspected and combined drug was stopped. A skin biopsy of the purpuric lesions was done and the pathological skin examination revealed leukocytoclasic vasculitis (LCV) and immunofluorescence showed Ig A deposition.
Anti-tuberculosis treatment was reintroduced with separated anti- TB drugs without any incident.
The diagnosis of combined HSP and pulmonary tuberculosis is retained after having eliminated other causes of rheumatoid purpura. Over the next month the skin eruption gradually disappeared and since this episode the patient still completely asymptomatic.

Conclusion
Our case is one of the rare adult cases associating HSP and pulmonary tuberculosis. This diagnosis was retained according to clinical findings (arthralgia and purpuric skin lesions) and LCV with IgA deposition in the skin biopsy, but neither abdominal pain nor renal manifestation has been observed with our patient.
The exact mechanism of this form is still unknown. Many hypothesis have been suggested like a direct invasion of vessel walls by tuberculosis, the deposit of immune complexes and Arthus reaction. This form is treated by treating the infection.

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An unusual case of primary colonic dedifferentiated liposarcoma with confusing presentation

Liposarcome dédifférencié du colon de présentation déroutante

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Liposarcoma is a common sarcoma derived from adipose tissue. Dedifferentiated liposarcoma (DDLPS) is defined as a well differentiated liposarcoma showing progression to non-lipogenic sarcoma of variable histological grade [1]. It occurs usually in the retroperitoneum [2,3]. It rarely presents in the gastrointestinal tract, and colon is an extremely uncommon site [4]. Only fourteen cases of primary liposarcomas of the colon have been found.
We report here a case of colonic liposarcoma and discuss diagnostic features and management.

Case presentation
A 50-year old man presented with epigastric pain and jaundice. Lipase and amylase were elevated. Abdominal ultrasound showed angiocholitis with two millimeter calculations of down choledoqie. Computed tomography (CT) revealed a pericholecystic abcess, and pancreas was normal. The diagnosis of pancreatitis stage A with angiocholitis was established and a cholecystectomy was performed after ten days of antibiotic therapy. Histological examination of the gallbladder showed lesions of acute ulcerated cholecystitis. The patient was discharged without any complication. Four months later, he presented with a 18-day history of abdominal pain without fever or jaundice. CT revealed intra-abdominal effusion and multiple parietal and intraperitoneal collections. The largest was under and next to the liver and measured 12 cm in larger axis [Figure 1].

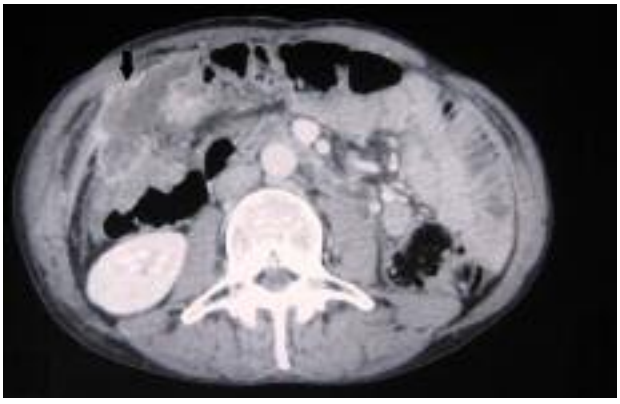

Figure 1: CT findings: A large collection under and next to the liver which measured 12 cm in larger axis (arrow).

This collection was associated with infiltration of adjacent parietal soft tissues and high densification of peritoneal fat of vicinity. Diagnoses discussed were: textiloma, residual stones of bilary duct, biloma and hematoma. A recovery of subcostal incision was performed. The hepatic flexure was perforated. An infiltrative tumor was found at ascending colon associated with anterior parietal invasion. Right hemicolectomy and ileostomy were performed. Macroscopic examination of the ascending colon revealed a nodular formation that measured 5.5 x 5 x 2.5 cm which infiltrated the adjacent small intestine and a second formation in the cecum that measured 1cm. Pathologic examination of both colic and cecal formations showed a proliferation which extended from the subserosa upward to the submucosa and dissociated muscular layer. The overlying mucosa was intact. The tumor cells were spindle-shaped and pleomorphic arranged in storiform and trabecular patterns [Figure 2].

Focal areas of adipose cells were present. Immunohistochemistry was done. Spindle-shaped cells showed nuclear reactivity for MDM2 [Figure 2] whereas pankeratin, CDK4, CD117, desmin, and caldesmon were negative. Fluorescence in situ hybridization with a MDM2-specific probe was uninterpretable. The diagnosis of DDLPS was established. The patient was lost to sight.

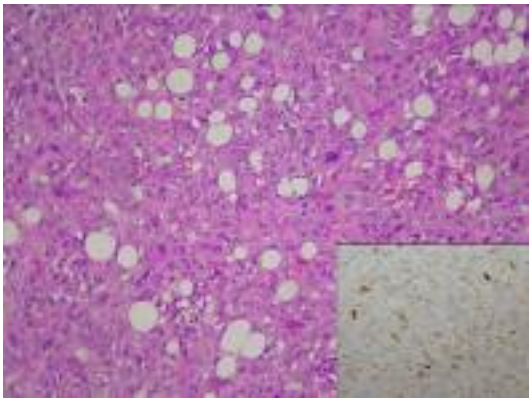

Figure 2: (HEx40): Tumor cells were spindle-shaped and pleomorphic. In the lower right, spindle-shaped cells showed focal nuclear reactivity for MDM2 (IHCx40).

Conclusion

In the present report the dedifferentiation component was large expanses which made diagnosis difficult and insite thinking about a stromal tumor or rhabdomyosarcoma. This fact demonstrates the role of immunohistochemistry in the exclusion of other tumor types. Diffuse nuclear expression of MDM2 and/or CDK4 made the diagnosis. The well-differentiated and dedifferentiated family of liposarcomas demonstrates both amplification of the chromosome subregion 12q13–q15 with resultant amplification of the MDM2 and CDK4 genes [5]. In our case, fluorescence in situ hybridization with a MDM2-specific probe was uninterpretable.

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Coronary-subclavian steal syndrome presenting with stable angina

Syndrome du vol sous-clavier symptomatique d'angor stable

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Case report

A 75-year-old male with multiple risk factors was referred to our department for worsening of exertional angina Canadian Cardiovascular Society Class III. Five years ago, he had an anterior myocardial infarction. Coronary angiography showed three-vessel disease. He underwent myocardial revascularization surgery (coronary artery bypass grafting CABG) with two aorta-coronary bypasses using right internal mammary artery (RIMA) to the left anterior descending (LAD), and left internal mammary artery (LIMA) sequentially to the diagonal branch and to the first marginal artery. Physical examination revealed absence of pulse in the left radial artery with a 50mmHg difference in blood pressure measurements between the two arms (140/80 and 90/50 mmHg in the right and left arms, respectively). Echocardiography revealed a mildly impaired left ejection fraction (45-50%) with hypokinesia of inferior and infero-lateral walls.

Cardiac catheterization via femoral approach showed the native coronary arteries with similar lesions to those found in the pre-CABG examination with patent RIMA and LIMA grafts. The left subclavian artery (LSCA) could not be catheterized, but angiography revealed 95% proximal stenosis of this artery (Figure 1).

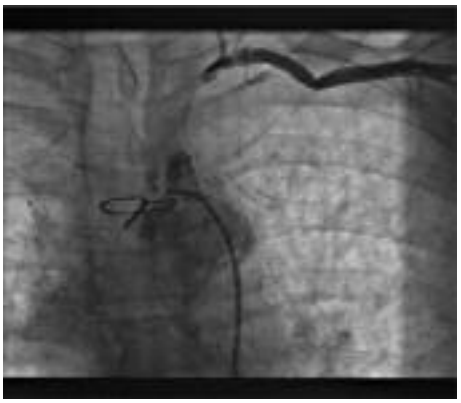

Figure 1: Angiography revealing a severe stenosis of left subclavian artery.

This helped to clarify the clinical setting: angina caused by ischemia of the left ventricular inferior wall due to the LIMA bypass 'stealing' from the native marginal artery circulation, as a consequence of occlusion of the proximal LSCA. We decide to proceed with a percutaneous transluminal angioplasty of LSCA. Using 0.035''

hydrophilic guidewire, LSCA stenosis was crossed, and then it was exchanged by a stiff wire with the same diameter. Predilation with balloon was performed (Figure 2) followed by the implantation of two overlapping auto-expandable stents with good angiographic result (Figure 3). Immediately, blood pressure became symmetric in the two arms. The patient remained asymptomatic on dual anti-platelet therapy.

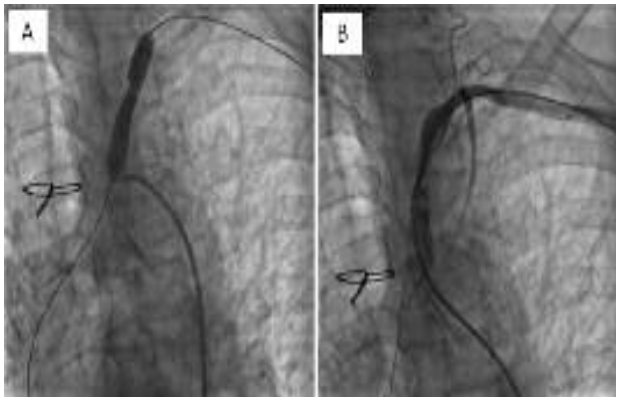

Figure 2: A) Balloon predilation with an 8/40mm Fox Plus balloon (Abbott Vascular., Switzerland).
B) Angiographic result after balloon predilation.

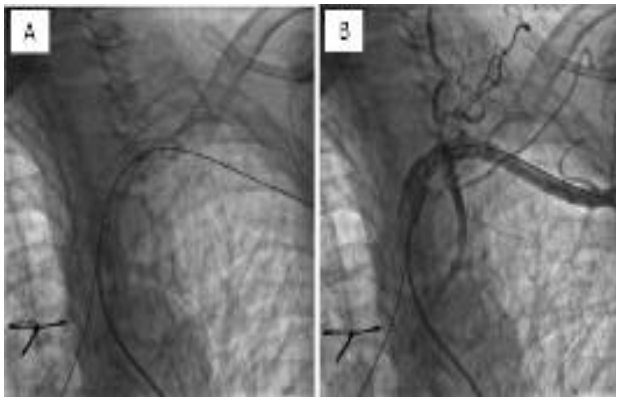

Figure 3 : A) Implantation of two overlapping autoexpandable stents (both SelfX 8/40 mm, [Abbott Vascular, Switzerland]).
B) Final angiographic result.

Relevant points

The coronary-subclavian steal syndrome (CSSS) is an unusual clinical syndrome, found in patients who underwent internal mammary coronary artery bypass grafting (1). Described for the first time in 1974 (2), it is relatively uncommon (incidence~0.4%) (3). It is caused by inversion of flow in the internal mammary artery used in a prior myocardial revascularization. It appears around three years after revascularization surgery (1) and might lead to exertional angina, silent ischemia or even myocardial infarction. In patients with previous CABG presenting symptoms or signs of ischemia, CSSS should be suspected in presence of difference in pulse and/or blood pressure between the two arms. Conventional or computed tomography angiography are recommended to confirm the diagnosis. Percutaneous treatment of CSSS is the standard treatment and it is a safe therapeutic option that avoids the risks of reintervention (4-7). Carotid-subclavian bypass is a useful option in case of total occlusion and inability to pass the guide wire through LSCA.

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