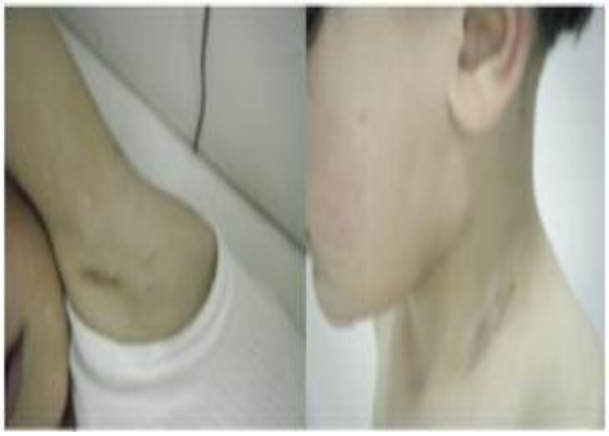
negative. Cervical lymph node biopsy showed recartive hyperplasia without mycobacterium. The serum salmonella agglutination test was positive as well as blood cultures for Salmonella enteritis.
The patient received a third generation cephalosporin intravenous for 10 days. Clinical signs and symptoms of vasculitis resolved after one week of treatment. The patient did not develop any signs of vasculitis as assessed in the last follow up visit at the age of five years without any recurrence. In conclusion, the diagnosis of CLV induced by Salmoenlla was confirmed after excluding a common cause of CLV sach as drugs, autoimmune disease, T cell deficiency or chronic granulomatosis, laboratory data and resolution of skin disease after removal of the inciting agent.

Figure 2 : Scar after axillary and cervical lymph nod biopsy



Conclusion

IL12-R β1 deficiency should be considered in patients with BCG infection, awareness should be increased in our country where BCG vaccination is performed in first day of life and consanguineous marriage is common. Patients with this genetic defect develop rarely CLV often induced by Salmonella and environmental Mycobacteria. Patients with chronic, recurrent, complicated or unusual localization salmonelosis should been screened for MSMD particularly for Il-12 deficiency. For these patients, Salmonella infection must be screening and prophylaxix antibiotherapy drug for salmonella is needed to prevent recurrence.

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Adenocarcinoma of the colon complicated by a psoas abscess: An exceptional mode of revelation

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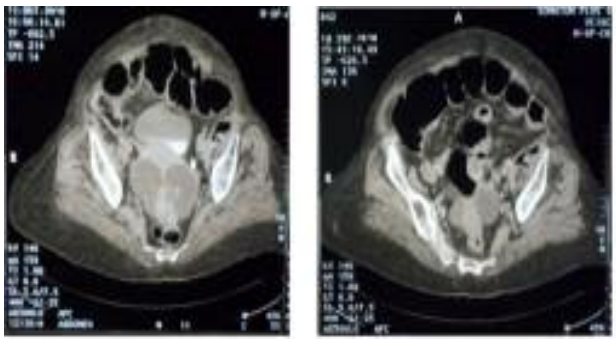
Infective spondylitis, tuberculosis, Crohn's disease, and diverticulitis are known causes of psoas abscess, unlikely to be, a perforating carcinoma of the colon [1, 2]. The abscess exceptionally reveals an underlying colon cancer [3]. Abdominal CT scan is the only preoperative test that can make the diagnosis of causality [3]. We report through this case an exceptional mode of revelation of adenocarcinoma of the sigmoid colon.

Case report

It is a 83-year-old woman, diabetic, hypertensive, who suffered from a deterioration of the general state and fever for about a month, associated with abdominal pain a few days before admission. Physical examination revealed a significant wheelbase of the left iliac fossa with a biology leukocytosis 16000/mm^3, glycemia at 20 mmol/L and urea at 15 mmol/L. An abdominal CT scan was done (Figure 1) showing an aeric-hydro collection with peripheral enhancement, interesting the left psoas muscle with a continuity with the wall of the sigmoid colon. The patient underwent operation with a midline incision and the discovery of a psoas abscess related to a perforated sigmoid colon tumor. The abscess was flattened and a sigmoidectomy completed by a Hartmann colostomy was then performed. The postoperative course was eventful by the occurrence of cerebral ischemic stroke, complicated by multi-organ failure and death of the patient at day-13. Histology concluded to a sigmoid colon adenocarcinoma.

Figure 1 : Collection of the left psoas muscle with peripheral enhancement coming into contact with the sigmoid colon



Conclusion

Perforation of the colon adenocarcinoma usually occurs intraperitoneally, whereas perforation in the retroperitoneal direction is relatively rare [4]. The extra peritoneal tissue does not react as fast as the peritoneal cavity to bacterial contamination [3]. Precise preoperative diagnosis is somewhat difficult, resulting in prolonged sepsis and associated high morbidity and mortality [4].

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Severe haemophilia B revealed by intracranial hemorrhage in a newborn

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Haemophilia is an X-linked congenital bleeding disorder caused by a deficiency of coagulation factor VIII (haemophilia A) or factor IX (haemophilia B). Hemophilia B is less common than hemophilia A, representing 15-20% of the total haemophilia population [1]. Intracranial haemorrhage(ICH) is the most serious bleeding symptom in haemophiliacs, resulting in high rates of mortality and disabling sequelae[2,3]. In observational cohort study in 12 European haemophilia treatment centres, Major bleeds occurred in 4-7%, head bleeds in 3-5% and ICH in 0,8% of neonates[3]. We report a new case of severe haemophilia B revealed by neonatal intracranial hemorrhage and discuss prolonged prophylaxis with factor IX replacement therapy in such patients.

Case report

The patient was born after a term pregnancy to a healthy mother, G1P1 via caesarean section after premature rupture of membranes, perinatal asphyxia and prolonged labour. Birth weight was 4400 g, length 53 cm and head perimeter 36.5 cm. Apgar scores were 8 at 1 min and 9 at 5 min. The patient received prophylactic intramuscular vitamin K at birth. Within 48 hours of birth, he presented seizures. In physical examination, he was noted to be pale, irritable with bulging anterior fontanel, and increase of head circumference (41cm). Computed tomography (CT) of the head showed intracranial hemorrhage with a right frontal intraparenchymal hematoma (figure 1). The family had no history of bleeding disorders. Laboratory tests showed a haemoglobin level of 7 g/dL (reference range: 14.5–22.0 g/dL) and a platelet count of 500. 10^9 (reference range: 150–500 .10^9). His prothrombin time (PT) was 100 %, and his activated partial thromboplastin time (APTT) was 103 s (reference range: 42,9±5,80).The patient received a transfusion of red blood cells and fresh frozen plasma pending the results of diagnostic coagulation tests. Factor VIII activity was 100 % (reference range: 55-121%) where as facteur IX activity was 0,5 % (reference range: 34-72%). Severe Hemophilia B was diagnosed. He was then given a 50 unit/kg /day of intravenous plasma derived factor IX concentrate during 14 days.

Figure 1 : Cranial computed tomography showing right frontal intraparenchymal hematoma

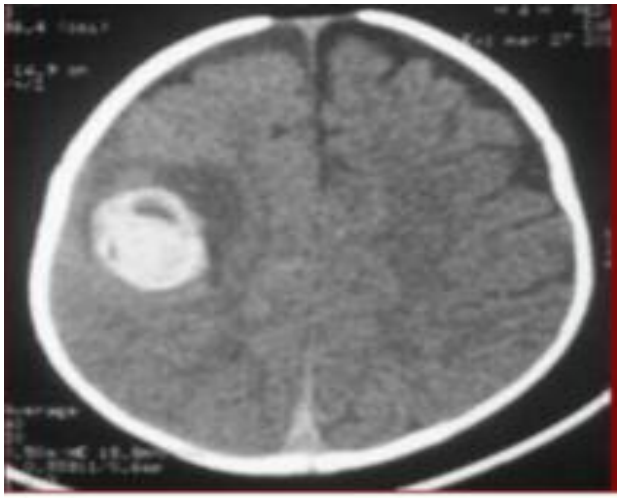
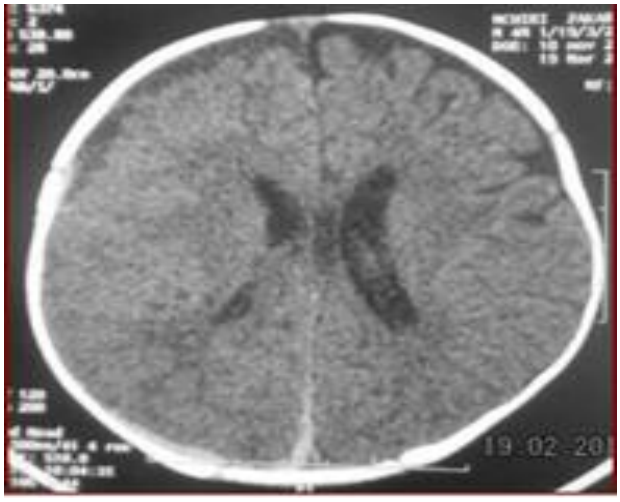


Figure 2 : Cranial computed tomography showing disappearance of the cerebral hematoma from the parenchymal tissue



The patient remained stable, so he was discharged home on the 20 hospital day without prophylaxis. The parents were informed of the diagnosis and presenting features of significant bleeding prior to discharge from hospital. Hemophilia carte was delivered. Scans performed 30 days after the event showed complete disappearance of the cerebral hematoma from the parenchymal tissue. At 3 months ages, the patient was readmitted for fever and seizure. A computed tomography scan of the brain revealed the recidive of the right frontal hematoma. View the recurrence of ICH, prolonged prophylactic factor IX replacement therapy was started. An implanted venous access was discussed with parents. However, because of the risks of surgery, local infection, and thrombosis associated, factor IX was given via venipuncture. During factor IX substitution, neither bleeding, nor inhibitor development were noted. The patient is currently 12 months old, he is neurodevelopmentally normal for his age. A follow up CT scan revealed frontal hypodense lesion (Figure 3).