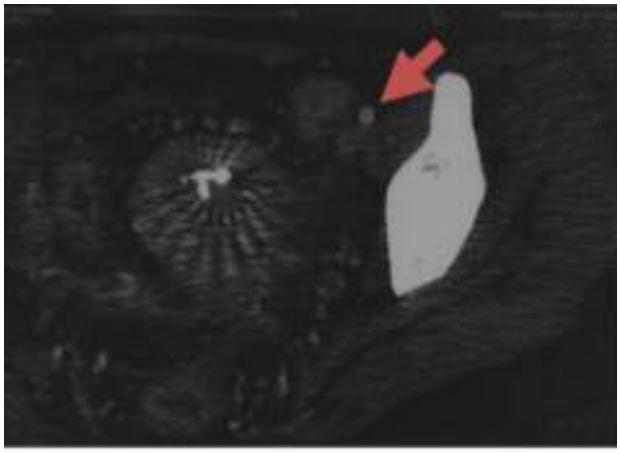
recommended, but possible use should be reviewed in an individual patient after evaluation of the risks, such as hemorrhage, compared to potential benefits (3).
We report here a case of 35-years old woman with Crohn's disease and internal iliac vein thrombosis and heparin induced thrombopenia.

Case report
A 35-years old woman, with no familial or personal history of inflammatory bowel disease, was hospitalized for diarrhea more than 6 bloody stools daily with abdominal pain which was predominant in pelvic region. She was not smoker and was not at any contraceptives. On presentation, the initial physical examination found an axillary's temperature of 38° C, tachycardia to 100/min, pallor conjunctiva, hypogatric sensitivity on abdominal palpation. Laboratory tests found an inflammatory process with sedimentation rate of 102 mm/h and C reactive protein 35mg/l. Complete blood count revealed anemia with hemoglobin of 8g/dl, platelet of 500000/mm^3 and normal white blood count. Albuminaemia was 24 g /l. Colonoscopic examination showed evidence of active colitis with ulcers and erosions in all colonic segments with patchy distribution of lesions. The ileum was normal. Histopathological examination of colonic biopsy specimens showed local irregularities in the crypts, cryptitis, lymphoplasmocytic and neutrophil leucocytic infiltrations in the lamina propria with feature of epitheloïd granuloma. Bacterial and viral superinfections were ruled out and initial abdominal CT not found any intra abdominal abces but found a regular thickening of the entire colon with infiltration of against fat and the presence of a left internal iliac vein thrombosis (Figure 1). I

Figure 1: CT-scan showing internal left iliac thrombosis



n the etiologic framework of pelvic thrombosis, the gynecological examination of the patient was normal and thrombophilia screen including protein S, protein C activity, antithrombin III activity, lupus anticoagulant and factor V Leiden mutation was negative. Homocysteine level was normal. We perform a lower-limb ultrasound to rule out a deep-vein thrombosis which was normal. The patient was treated with first line intensive treatment, based on absolute parenteral nutrition, corticosteroids intravenously at a dose of 1mg/Kg/day and low molecular weight heparin (LMWH) at an hypocoagulative dose. The evolution was marked by clinical and biological improvement in the seventh day of treatment allowing the switch to oral corticosteroids and the association, of warfarin. Otherwise, on the tenth day of treatment with LMWH (and the third day of warfarin), laboratory screening test found a thrombocytopenia at 98000/mm3 with NRI of 3. Heparin was, so, strongly incriminated and the diagnosis of heparin induced thrombopenia (HIT) was strongly suspected. Repeated doppler ultrasonography demonstrated no evidence of lower limb thrombus and normal blood flow in the left internal iliac vein. So we decide to stop LMWH but warfarin was continued and platelet count returned to normal on the fourth day of discontinuation of LMWH. Two months after the diagnosis, repeated colonoscopy showed completely normal macroscopic findings. Prednisolone was tapered slowly and azathioprine (1.5 mg/kg/day) was prescribed. Three month after the diagnosis patient was already on warfarin and azathioprine treatment with clinical remission.

Conclusion
Our patient developed a symptomatic form with combined risk factors for the development of thrombosis which were active Crohn's disease, treatment with high doses corticosteroid and heparin induced thrombopenia. The particularities of our case are the site of the thrombosis and the HIT developed after LMWH prophylactic therapy which constitutes an additive risk factor needing a specific management. Our patient developed initially iliac vein thrombosis before LMWH treatment and was successfully management with improving the inflammatory process.

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Concha bullosa pyocele

Concha bullosa, a pneumatized middle turbinate, is a common anatomic variant found in the paranasal sinuses. It incidence varies from 14 to 53% (1). When a concha bullosa becomes obstructed, it can form a mucocele and, eventually, a

mucopyocele if it becomes secondarily infected. We report a case of a secondarily infected mucocele arising from a concha bullosa.

Case report

A 22-year-old female was referred to us with nasal obstruction, acute loss of the sense of smell and intermittent postnasal drainage in the right side. The patient had been suffering from these complains for nearly 1year. She denied epistaxis, diplopia and loss of vision. Anterior rhinoscopy revealed a massively large middle turbinate on the right side. The nasal mucosa was congestive and no pus or crust was detected in the nasal passage. A giant middle turbinate displacing the septum was seen on endoscopic nasal examination. Computed tomography (CT) showed a homogenous fluid or soft tissue density lesion surrounded by a thin bony shell in the right nasal passage. The mass caused the septum to deviate to the contralateral side, without orbital displacement. Moreover, right ethmoidal sinus was partially filled by secretion. This lesion considered as middle turbinate (figures 1, 2).

Figure 1: Axial computed tomography shows a soft-tissue mass well circumscribed by a bony perimeter of the concha bullosa. The left middle turbinate seems normal.

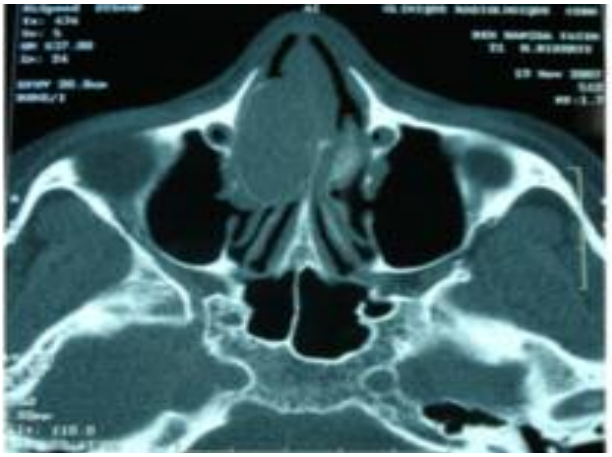
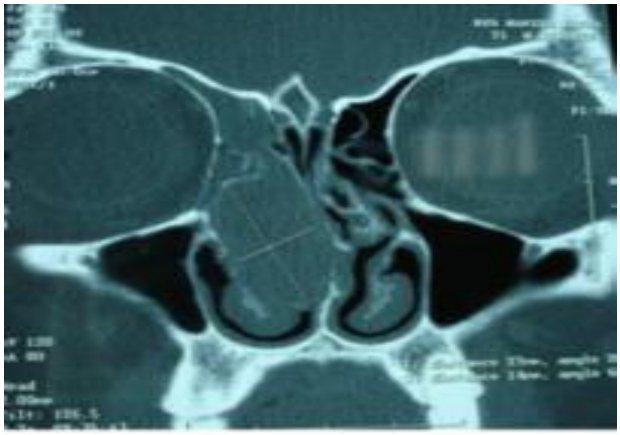


Figure 2: Coronal computed tomography demonstrates a right large intranasal mass



Endonasal microsurgery was performed. After freeing the mass from the superior margin, a bullous middle turbinate, with pus in it, was seen. Incision of the mucoperiosteum and underlying bone was followed by resection of the lateral and inferior walls of the middle turbinate. On histopathologic examination of the surgical specimen, respiratory epithelium and osseous particles with chronic inflammatory changes were seen. The final diagnosis was middle turbinate pyocele. No recurrence has been observed after 1 year.

Conclusion

Pyocele of concha bullosa may cause several pathological changes. The mucosal lining of pneumatized middle turbinate in a pyocele may become inflamed, leading to swelling of the anterior portion of the middle turbinate with obstruction of the middle meatus, then results in sinusitis. Other symptoms are possible in CBP such as headache, nasal discharge, postnasal drip, nasal obstruction, and loss of the sense of smell. In advanced cases, dacryocystocoele, orbital invasion, and subdural empyema have been reported. The diagnosis of concha bullosa pyocele is based on physical, radiological and histopathological examinations. Endoscopic appearance of concha bullosa pyocele reveals an enlarged head or body of the middle turbinate that is in contact medially with nasal septum and bulges laterally into the nasal wall of the nose. CT scan of facial bone shows a prominent soft-tissue mass well circumscribed by a bony perimeter (the bony framework of the concha bullosa). Magnetic resonance imaging (MRI), however, is considered superior to CT in diagnosing the intracranial and intra-orbital complications. Concha bullosa pyocele should be treated surgically. The recommended treatment for CBP is endoscopic surgery. Four methods are used to manage the CB surgically: lateral marsupialisation, medial marsupialisation, crushing, and transverse excision. Delay in the treatment may lead to permanent orbital sequelae or intracranial complication.

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