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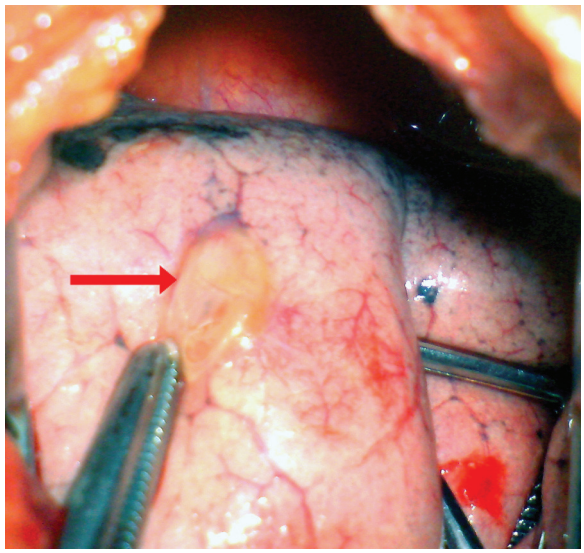

Figure 1: Intraoperative image of ruptured bullae on a metastatic lesion (red arrow).

Surgical treatment of gastrointestinal stromal tumour of jejunum

Dear Editor,

Gastrointestinal stromal tumours (GISTs) are rare mesenchymal tumours of the gastrointestinal tract acounting 5% of all sarcomas. GISTs originate from interstitial cells of Cajal, which regulate the peristalsis mechanism of the gastrointestinal tube. Treatment may be surgical, conservative or both[1]. Here we report the case of a female patient with upper abdominal pain, obscure intestinal bleeding and anaemia.

A 43-year-old Caucasian female was presented to Emergency Department with a 15-day history of upper abdominal pain and fatigue. Physical examination and blood biochemistry were normal. Haematological tests proved anaemia, thrombocytopenia and obscure intestinal bleeding. CEA and CA 19-9 were normal. Use of NSAID was not mentioned. Chest and upright abdominal X-rays, abdominal ultrasonography, upper gastrointestinal endoscopy and colonoscopy did not reveal any significant findings. The abdominal computed tomography indicated an extraluminal, well-delimited formation of jejunum, measuring 4×3×5 cm, possibly malignant. Wireless capsule endoscopy revealed a cancerous mass that partially obstructed the lumen of jejunum. Exploratory laparotomy confirmed a haemorrhagic tumour at the end of jejunum, without presence of adhesions to surrounding tissues. Local resection of the tumour was performed at 15 cm both proximally and distally, to have an oncologically safe margin. Histopathological examination confirmed a GIST of jejunum with mitotic activity >2 mitosis per 50 high-magnification optical fields (m ≤ 2 / 50 OPMM). Regions with cystic degeneration and haemorrhagic infiltration were recognized. The immunohistochemical study showed positive indicators for identification of tumour: C Kit + (CD 117), CD34 +, S100-, Actin +, Desmin-. The lymph nodes in adjacent mesenteric tissue were metastasis free. The patient was discharged in good condition on the 5th postoperative day and up to today is regularly followed up at the oncology outpatient clinic.

Primary surgical aim is to completely remove the tumour[2]. First-line adjuvant therapy is imatinib mesilate, a selective receptor tysorine kinase inhibitor that targets the KIT and platelet-Kinases. Another TKI, Sunitinib Malate is approved as second-line treatment for GIST patients, following failure of imatinib. Over the last decade patient management with GIST has greatly improved due to the introduction of TKI therapies, iimprovement of imaging modalities and surgical intervention.

A vast majority of patients can expect cure by expert surgical resection. As regards patients at higher risk of relapse, adjuvant imatinib may help prolong disease recurrence. Patients who suffer unresectable or metastatic GIST are treated successfully with imatinib[3]. For those who develop imatinib-resistance receiving imatinib in higher doses was tested successfully. For patients that imatinib can not control any further progression of the disease the only globally approved second-line TKI is sutinib[4] that has already given promising results. Research will probably have impressive results regarding the management of the disease in the future.

Conflict of interest statement

No conflict of interest is declared by any of the authors.

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Sweet's syndrome associated with upper respiratory tract streptococcal infection: "wait-and-see" strategy or anecdotal use of corticosteroids?

Dear Editor,

A 53-year old Caucasian male presented with fever and multiple painful erythematous papules and plaques located at face, chest, back and upper extremities. Physical examination revealed vesicles and pustules on top of lesions and large edematous nodules at dorsal surface of hands. Eruption's onset was reported 2 days before, followed by gradually appearing skin lesions. Few days before patient experienced upper respiratory tract infection symptoms (pharyngoamygdalitis), without receiving any medication. Detailed history revealed no co-morbidities and previous medications.

Laboratory investigation revealed leukocytosis with neutrophilia (WBC 20700/mm^3, neutrophils 85.6%); antistreptolysin O (ASTO): 4260 IU/ml (normal<166 IU/ml); ESR: 67 mm/h (normal <20 mm/h); CRP: 152 mg/L (normal <5 mg/L). Full biochemical and serological examination and laboratory investigation for autoimmune diseases were negative. Treatment with amoxicillin/clavulanic acid (500/125 mg every 8 hours) orally was initiated for treatment of streptococcal infection. Histological examination established Sweet syndrome diagnosis revealing marked dermal edema and infiltrate composed of neutrophils, some of which showed nuclear fragmentation (leucocytoclasis), along with vasodilation and vascular endothelium swelling with no true vasculitis findings.

Taken into consideration the risk of systemic corticosteroid use during streptococcal infection, a "wait-and-see" strategy was decided and no concomitant treatment was added. Skin lesions' remission started 4 days after initiation of treatment and complete remission was achieved after 10 days. Serum levels of WBC, ESR and CRP decreased after 3 days of treatment and reached normal levels within 10 days. Fever resolved after day four. ASTO levels continued to elevate for 3 days before gradually dropping and reaching normal levels after 6 months. Treatment with amoxicillin/clavulanic acid was continued for 15 days. Skin lesions did not recur after 7 months of follow-up.

Sweet's Syndrome has been associated with infections, malignancies, medications and autoimmune diseases[1]. Interestingly, is has been also associated with granulocyte-colony stimulating factor (G-CSF) treatment[1]. The initial hypothesis for streptococcal infection related Sweet Syndrome is hypersensitivity reaction to bacterial antigens. On the other hand, it is known that streptococcal infection induces GM-CSF. Increased G-CSF production in nonspecific acute phase response is key physiological component of host defense and enhances host resistance to bacterial infection[2]. Corticosteroids reduce GM-CSF protein synthesis[3], thus negatively influencing immunological response against streptococcus.

Although in our patient spontaneous remission of Sweet Syndrome can not be ruled out, we believe this is not the case, since remission of infection, ASTO, WBC and ESR levels and skin lesions moved in parallel. Taken into account the low morbidity of Sweet's syndrome, the negative influence of systemic corticosteroids on immunological response against streptococcus and evidence presented in this case report (Level of evidence:5), we suggest that when findings clearly demonstrate Sweet Syndrome associated with upper respiratory tract streptococcal infection, treatment of infection with antibiotics like amoxicillin/clavulanic acid should be considered before corticosteroid use and perhaps a "wait-and-see" strategy should be preferred.

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