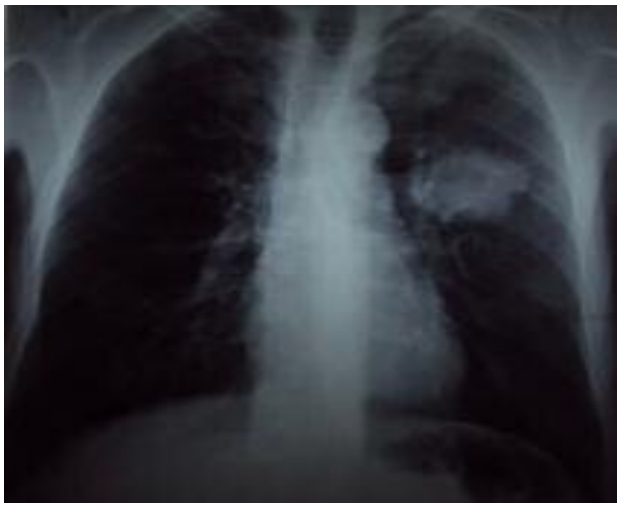
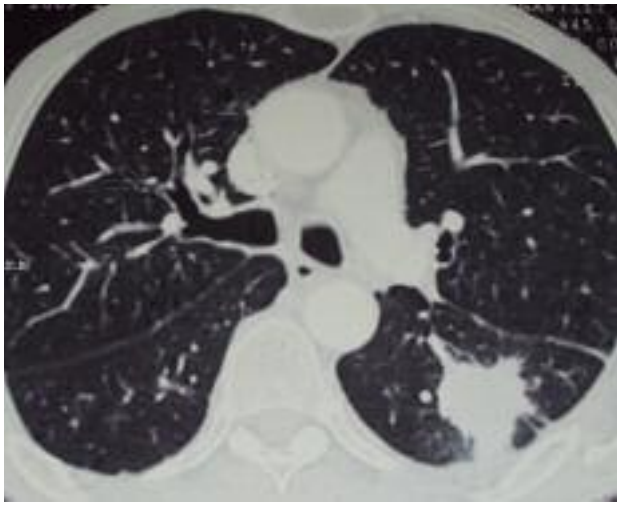
department of pneumology to have further explorations. On admission the patient reported a performance status at 1. He was not febrile. Cardiac and pulmonary auscultations were normal. His blood pressure was 110/80 mm Hg. Digital clubbing was present.

Figure 1 : Chest X-ray PA view showing a left parahilar, homogenous, speculated opacity which was 5 cm in its largest diameter.



The rest of physical examination was normal. Biology exams revealed a biologic inflammatory syndrom with accelerated sedimentation rate at 70 on the first hour and elevated C reactive protein at 76 mg/l. Bronchoscopy was without abnormality. Chest CT scan revealed a speculated mass of the left Fowler laying on the scissure and having contact with the parietal pleura. The mass was associated to a local lymphangitis carcinomatosis (Fig.2).

Figure 2 : An axial section of a CT scan of the chest revealed a speculated mass of the left Fowler laying on the scissure and having contact with the parietal pleura.



The patient had therefore a biopsy of the mass which was guided by CT scan. Pathologic examination of the mass showed tumoral proliferation consisting on rounded small cells with non abundant cytoplasm. Immunohistochemistry examination of the tumor revealed that the cells were positive for CD99 and EMA, which made the diagnosis of primary pulmonary neuroectodermic tumor. Extension of the lung cancer was looked for by abdominal, pelvian and cerebral CT scans that didn't show any metastasis. The patient was then treated by neo-adjuvant chemotherapy with etoposide, doxorubicine, vincristine, holoxan and uromitexan. CT scan made at the end of four chemotherapy cycles revealed a decrease of the size of the Fowler's mass and a regression of the lymphangitis carcinomatosis. Four additional cycles of chemotherapy were hence carried. However, the patient had very rapidly a profound alteration of his general condition and had bone metastasis on his right scapula and sterno-clavicular articlulations objectived by bone scan. The patient died few days later before undergoing surgery.

Conclusion

PNET of the lung are rare. In our knowledge, our patient is the tenth case of PNET reported in the literature. Their diagnosis is based on histological evidence. Treatment of these tumors is heavy and mainly depends on location, histologic grade and the presence or not of metastases. It includes, in the majority of cases, radical surgery, neo-adjuvant chemotherapy and/or adjuvant chemotherapy and radiotherapy. However, the prognosis is bad with a survival rate which remains poor.

References

1. A. Ammani, A. Janane, J. Sossa, H. En-Nouali, A. Albouzidi. Tumeur neuro-ectodermique primitive (PNET) de la voie excretrice supérieure. J Maroc Urol 2009 ; 13 : 13-17.
2. Lin Shi, Zhanlin Guo and Xinlin Wu. Primary pulmonary primitive neuroectodermal tumor metastasis to the pancreas: a rare case with seven-year follow-up. Diagnostic Pathology 2013, 8: 51.
3. C. Daniel, V. Laurence, J.-Y. Pierga. Tumeur neuroectodermique primitive périphérique du poumon chez un homme de 49ans. Revue des Maladies Respiratoires 2013; 30: 62-66.

About an unusual association Guillan Barre syndrome and pulmonary tuberculosis

Daghfous Hafaoua, Zaibi Haifa, Tritar Fatma.
Service de Pneumologie pavillon C, hôpital Abderrahmen Mami-Ariana

Guillan-Barré syndrome (GBS) is an acquired inflammatory peripheral neuropathy defined by acute onset, cerebrospinal fluid (CSF) albumin cytological dissociation and a clinical monophasic course with partial or total recovery (1). GBS has been associated with both infective (viral, mycoplasmal, bacterial and chlamydial infection) and non-infective aetiologies (1, 2). Association GBS and tuberculosis has been exceptionally reported and pathagenosis was unknown (3, 4).
We report an original case report of GBS in immunocompetent patient treated with pulmonary tuberculosis which simulating a drug polyneuroradiculopathy.

Case report:
A 19-year-old man, was initially presented with 6 months clinical history of chronic cough, scanty expectoration, prolonged fever and loss of appetite and weight. Physical examination was normal. Chest X ray revealed several confluent consolidations and cavity lung lesions in upper right pulmonary zone. The erythrocyte-sedimentation rate was 120 mm in the 1st hour and CRP was 102 mg/l. Sputum smears was positive for acid-fast-bacilli and culture for mycobacterium tuberculosis, with normal susceptibility to usual drugs. The diagnosis of pulmonary tuberculosis was confirmed and anti-tuberculosis fixed multi-dose combination H75R150Z400E275: 4 tablets/ day was instituted.Twenty one days after start of treatment, the patient suffered acute and progressive legs weakness that ascended to the arms. A progressive respiratory muscle weakness, respiratory failure, no traumatic paraplegia, paraperesis, bilateral motor neuron 7th cranial nerve pals were developed and patient was admitted in the intensive care unit and ventilator support was required during 14 days. It was initially thought to be isoniazid-neuropathy. However, stopping the drug did not improve this condition.
Cranial computer tomography and cerebro-spinal magnetic resonance imaging were normal. Lumber puncture showed acellular cerebrospinal fluid with 210 mg/dl of albumin and 75 mg/dl of sugar. Nerve conduction studies confirmed the diagnosis of GBS, revealing an acute sensitive-motor axonal and radicular neuropathy with denervation.
Human immunodeficiency virus, type A, B and C hepatitis virus and antibodies to nuclear antigens were negative.
Therefore, we concluded to the association of guillain Barre syndrome and pulmonary tuberculosis.
Immunoglobulin therapy (0,4g/Kg/day) over 3 days was prescribed. Anti-tuberculosis therapy and physiotherapy were instituted and continued during 6 months with recuperation of the motor failure after 3 weeks. Two months after, patient experienced partial clinical improvement and at the end of anti-tuberculosis treatment patient was recovered.

Conclusion:
Peripheral neuropathies in association with tuberculosis infection are uncommon, and mostly due to associated malnutrition, alcoholism, and the neuropathic effects of medication especially Isoniazid and Ethambutol (4-6). In our patient, peripheral neuropathy developed after the treatment of TB started, let's to suspect drug toxicity, but aggravation of neurologic syndrom despite interruption of anti-tuberculosis drug deviates this hypothesis.
The pathogenesis of SGB associated with TB remains obscure. Disease can be a result of meningeal involvement, direct nerve root involvement, and granuloma affecting peripheral nerves (4-8). In our case this hypothesis seems the most likely but what the method that we can used to confirm this hypothosis and can nerve biopsy help to diagnosis? Immunocompetent status and no any medical history in our case underlines the originality of this case report. Those factors, can explain the favorable out-come and the recovering of this patient.

References:

1. De la Torre RG, Morís G, Martínez DP, Montes IC. Guillain-Barré syndrome, tuberculosis and inflammatory bowel disease: a multiple association. Int Arch Med. 2010; 16: 3:15.
2. Hughes RA, Cornblath DR: Guillain-Barré syndrome. Lancet 2005 ; 366:1653-1666.
3. Peiris Eiris JB, Wickrasinghe H R, Chandrasekera MA. Tuberculous polyradiculitis. BMJ 1974; 4: 107.
4. Taha AA, Tee KH. Guillain Barre syndrom associated with pulmonary tuberculosis. BMJ Case Resp 2012; 13: 012.
5. Fernández-Fúnez A, Gómez Garrido J, Alamillo A, Sáez L: Demyelinating polyneuropathy as the Honest form of lymph node tuberculosis. Paradoxn response in an immunocompetent patient. Med Clin 2007; 129:78-79.
6. Shin SS, Hyson AM, Castañeda C and all. Peripheral neuropathy associated with treatment for multidrug-resistant tuberculosis. Int JTuberc Lung Dis 2003; 7: 347-353.
7. Sabriá Mestres J, Alijotas Reig J, Ordi Ros J, Biosca Gómez de Tejada M, Sumalla Suñé J. Guillain-Barré syndrome and tuberculosis. A chance association? Rev Clin Esp 1985; 176: 320-1.
8. Chong VH, Joseph TP, Telisinghe PU, Jalihal A: Chronic Inflammatory demyelininating polineuropathy associated with intestinal tuberculosis. J Microbiol Immunol Infect 2007; 40:377-80.

Association maladie de Crohn et tumeur carcinoïde appendiculaire.

Olfa Hellara[1], Aya Hammami[1],Rim Hadhri[2] ,Ali Abdelmoula[1], Mayada Trimech[1], Hichem Loghmari[1], W Ben Mansour[1], Wissem Melki[1], Fethia Bdioui[1], Leila Safer[1],Abdelfattah Zakhama[2], Hammouda Saffar[1].
[1]: service de Gastro-entérologie de Monastir.
[2] : Laboratoire d'anatomo-pathologie de Monastir

Il est actuellement reconnu que les patients atteints d'une maladie de Crohn sont plus à risque de développer des cancers digestifs (1). Il s'agit essentiellement d'adénocarcinome grêliques et coliques (2). L'association à des tumeurs carcinoïdes est exceptionnelle et controversée (3,4). A notre connaissance, uniquement douze cas ont été rapportés dans la littérature. La localisation appendiculaire semble être prédominante, suivie par la localisation iléale. Nous rapportons deux observations de tumeur carcinoïde appendiculaire associée à une maladie de Crohn.

Observation 1 :
Patient MH.B âgé de 18 ans, aux antécédents de maladie de Crohn chez son frère, suivi pour une maladie de Crohn depuis l'âge de 8 ans. Il s'agit d'une maladie iléo-colique avec des poussées fréquentes ayant nécessité plusieurs cures de corticothérapie avec un retentissement sur le développement staturo-pondéral et pubertaire. Devant le profil évolutif, le patient a été mis sous Azathioprine à la dose de 2.5mg/kg/jour. Deux ans après, le patient a développé une poussée sévère avec une atteinte sténosante traitée endoscopiquement, avec échec de celui-ci. Ainsi, le malade a été opéré avec une résection iléo-caecale. A l'examen anatomo-pathologique, le grêle réséqué mesurait 20 cm. L'appendice avait un aspect macroscopiquement normal. A l'histologie, on notait une iléite ulcéreuse et granulomateuse panpariétale et segmentaire compatible avec une maladie de Crohn en poussée (Fig 1,2). Une prolifération tumorale occupant la sous-muqueuse et mesurant 2 mm a été notée au niveau de la pointe appendiculaire. Il s'agissait d'une tumeur neuroendocrine bien différenciée, classée grade 2 de l'OMS(Fig 3,4). Les suites étaient simples. Le malade était mis sous anti-TNF alpha , en traitement d'entretien ,étant donné l'échec de l'Azathioprine. Concernant sa tumeur endocrine, et en se référant au consensus de