major difference between CD and lymphoma is the *clonality* and *loss of architecture* in the latter.

Clinically, most CD are unicentric (UCD) and so clinically benign without B symptoms. On the other hand, multicentric CD (MCD) might be clinically indistinguishable from lymphoma. So far, CD has been mostly found in second to seventh decade of life and young patients mostly present with UCD; but our case is clinically MCD in a teenage which is unusual.

Another *unusual* presentation is the back pain. Pain from retroperitoneal structures may refer to back (e.g., pain from pancreas or kidney), but intraabdominal lymph node enlargement causing back pain is quite unusual, that compelled neurosurgeons to go for MRI of lumbosacral spine.

Morphologically CD is classified into hyaline vascular variants (HVV) and plasma cell variants (PCV); some authors preferred another class of mixed variants. When combining clinical and morphological features, CD can be classified in 4 major types: unicentric HVV (72%), unicentric PCV (18%), multicentric PCV (10%, *like this case*) (Figure 1) and rare multicentric HVV (1%)¹.

Pathogenesis of UCD is inconclusive, but it is said that IL-6 has a strong role in the pathogenesis of MCD and deletion of IL-6 gene prevents developing Castleman's disease. It is postulated that HHV-8 produces interleukin 6 and is responsible for lymphoplasmacytic proliferation². That is why anti-IL6 (suramin) and IL-6 receptor antagonist antibody (atlizumab) both are tried for treatment of MCD³.

From the above discussion it is clear that in our case many things should have been done but for unavailability and financial restraint we couldn't do many tests, namely, PET to see true disease involvement; and HHV-8 PCR and CD20 which guide therapeutic measures where HHV-8 positive cases respond to valgancilovir, and in CD20-positive cases anti-CD20 monoclonal antibody (rituximab) is considered⁴.

At present, there is no consensus as to the optimal management strategy for MCD. Successful treatment of MCD has been achieved using chemotherapy, with or without prednisone, given at the time of initial diagnosis⁵. That is why, considering the age toxicity and chemotherapeutics, he was treated with 4 cycles of ABVD which was completed by December 2010. On follow up for 6 months, there was significant clinical improvement and disappearance abdominal lymphadenopathy evident ultrasonography.

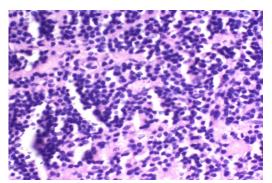


Fig. 1: It presents follicular hyperplasia with prominent germinal centres. Marked proliferation of germinal centres are seen. In some areas the blood vessels have entered into the follicles forming so called lollypop appearance. Angiofollicular lymph node hyperplasia (Castleman disease).

Conclusion: Though rare, Castleman's disease should be considered in patients presenting with single or multiple lymph node enlargement. Due to malignant potential and late complications, patients with multicentric CD should be offered chemotherapy early with adequate explanation and counselling, which might prevent reaching a point of no return.

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References

- Jun bean Park, et al. Multicentric Castleman disease presenting as jaundice. The Korean Journal of Internal Medicine 2007; 22: 113-117.
- Dupin N, Diss TL, Kellam P, Tulliez M, Du MQ, Sicard D, Weiss RA, Isaacson PG and Boshoff C. HHV-8 is associated with a plasmablastic variant of Castleman disease that is linked to HHV-8-positive plasmablastic lymphoma. Blood 2000; 95(4):1406-12.
- Beck JT, Hsu SM, Wijdenes J, Bataille R, Klein B, Vesole D, Hayden K, Jagannath S, Barlogie B. Brief report: alleviation of systemic manifestations of Castleman's disease by monoclonal anti-interleukin-6 antibody. N Engl J Med. 1994 Mar 3; 330(9): 602-5.
- Casper C.The actiology and management of Castleman disease at 50 years: translating pathophysiology to patient care. Br J Haematol 2005; 129: 3-17.
- Weisenburger DD, Nathwani BN, Winberg CD, Rappaport H: Multicentric angiofollicular lymph node hyperplasia: A clinicopathologic study of 16 cases. Hum Pathol 1985; 16: 162.

Constipation – presenting compliant and clinical marker of Parkinson's disease

To date a large portion of existing literature has reported the high incident of constipation present in Parkinson's disease patients. Some studies have reported prevalence rates of constipation in Parkinson's disease (PD) patients as high as 90%¹. The disease causes a loss of peristalsis,

immobilization, and rectal-anal reflex¹. Typically constipation progresses as the Parkinson's disease progresses still, there have been cases of patients who have a history of mild constipation before the diagnosis of Parkinson's disease¹. However, acute onset of constipation before the onset of motor symptoms is rather unusual and was seen in our patient which definitely led to the diagnosis of PD. Our assessment of this patient thus highlights that acute constipation could be a presenting symptom of PD.

The patient was a 59-year old male was referred for evaluation of constination. He was unable to have bowel movements for 5 to 6 days. Prior to this, he had regular bowel movements of once daily. After multiple investigations including gastrointestinal endoscopy, abdominal X-rays and CT scans, no cause was found. He had a history of intermitted mild lower back pain. After a neurology assessment a MRI of the spine was requested. An MRI examination of the spine was done and showed multi-level degenerative disease with spinal stenosis at T10-11 accompanied flattening of the spinal cord. Still no cause for constipation could be found. He was referred to spine surgeon, and after assessment he was not deemed to be a candidate for spine surgery as spinal stenosis was not marked. For the management of pain due to spinal stenosis he was referred back to a neurologist. During this assessment, he was noted to have decreased facial expression and blink rate. His speech was also soft. Motor examination revealed mild rigidity of upper limbs, left greater than right. He had mild bradykinesia of left side, and his speed of walking was slight slow. He was eventually diagnosed with PD. Introduction of dopaminergic medications helped to resolve his constipation.

Presentation of this patient with acute constipation in the absence of other typical causes of constipation and development of Parkinson's disease is interesting. This indeed highlights the importance of screening patients with acute constipation in the absence of other causes for signs and symptoms of PD. Attention should be focused on medical history, duration, and frequency of the stools and bowel movements. Abdominal examination for any sign of hardened mass and digital rectal exam, and other investigations if necessarily should be checked early on

A diagnostic criterion of constipation has to meet a minimum of two of the following conditions for at least three months^{3,4}

- 1. Incomplete evacuation, more than 25% of time
- 2. Straining during bowel movement, more than 25% of the time spent in toilet

- 3. Two or less bowel movements per week
- 4. Hard stool, more than 25% of the time

An accurate and detailed medical history from a patient with constipation is vital in order to define the type of constipation they are afflicted with. This, in turn, directs a proper diagnosis and treatment.

An array of investigations can be performed to evaluate constipation. However, most patients require only a few basic diagnostic tests. Extensive investigations are reserved for individuals who have severe constipation or whose condition does not respond easily to treatment.

This case report urges a comprehensive appraisal of autonomic symptomatology in elderly PD patients and affirms that autonomic nervous system dysfunction is a pervasive problem in PD^{5,6}. Most patients with PD suffer from constipation-related symptoms. However, severe constipation does arise as the disease advances. In Parkinson's disease, aggressive treatment of constipation is required as it may impair the absorption of dopaminergic medications. Improving gastrointestinal motility can increase the absorption of medication leading better efficacy of medication. This leads to better motor control⁷.

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References

- Huntley R, Editor IH .The Surprising Link Between Constipation and Parkinson's. Insider's Health Articles 2010.
- Mathers S E, Kempster P A, Law P J, et al. Anal sphincter dysfunction in Parkinson's disease. Arch Neurol. 1989; 46: 1061-4.
- Thompson W G, Longstreth G F, Drossman DA, et al. Functional bowel disorders and functional abdominal pain. Gut. 1999; 47: 2222-30.
- Longstreth GF, Thompson WG, Chey WD, et al. Functional medical therapies for chronic constipation: systematic review. Bowel disorders. Gastroenterology. 2006; 130: 1480-91.
- Koller WC., Tse W. Unmet medical needs in Parkinson's disease. Neurology. 2004; 62:S1-S8.
- Micieli G, Tosi P, Marcheselli S, Cavallini A. Autonomic dysfunction in Parkinson's disease. Neurol Sci. 2003; 24(Suppl 1): S32–34.
- Levine RR. Factors affecting gastrointestinal absorption of drugs. Am. J. Dig. Dis. 1970; 10: 171-188.