Acute Proptosis as an Initial Presentation of Bronchogenic Carcinoma

Sir,

A 47 years non-smoker male presented with acute onset painless, rapidly progressive left ocular bulging for last seven to ten days. Patient also had difficulty in left lateral gaze that caused double vision. He learnt to adapt to this by turning his head to the left. There was no other ocular symptom or symptoms pertaining to other body system. His past, personal and family history was non-contributory.

On examination, the patient had moderate pallor and tender, firm hepatomegaly. Lymph nodes were not palpable. Most striking clinical sign was proptosis of the left eye. Detailed examination revealed lateral, superior and inferior rectus palsy along with conjunctival congestion. However, pupillary reaction was present and forced duction test was positive- excluding both primary neurologic or muscular disease. Ophthalmoscopy showed no significant abnormality. Examination of other systems was non-contributory.

Investigations showed Hb= 8.6 gm%, ESR=106 mm at 1st hour, and alkaline phosphatase 482 U/L. All other routine investigations and thyroid function tests were normal including a negative anti-thyroperoxidase antibody. Ultrasound examination of the left eye revealed destruction of the lateral orbital wall by a heterogeneous mass with bony spicules. CT scan of orbit confirmed the mass as an extraconal lesion. Chest X-ray revealed a 3 cm diameter mass in the right upper and mid zones with blunting of right costophrenic angle. CT scan of thorax, showed enlarged lymphnodes in pretracheal, precarinal, right paratracheal and subcarinal region. An inhomogeneously enhancing soft tissue density lesion was seen involving the right upper lobe extending to the pleural surface. Ultrasound of the abdomen revealed enlarged liver with multiple hypoechoic metastatic deposit. The lung mass on FNAC was diagnosed as non-large cell bronchogenic carcinoma. CT-guided FNAC from both the orbital mass and hepatic deposits corroborated with the diagnosis of metastatic large cell lung cancer. A whole body scintigram revealed multiple bony metastases involving left orbital wall, vault of the skull, lower cervical spine, and lumbosacral spine. The patient was given systemic chemotherapy (cisplatin and etoposide) and irradiation of left orbital tumor. A favourable response was achieved in ocular movements. Patient died one month later because of progression of the primary lesion.

Metastatic orbital tumours represent 1.5% of all orbital tumours and tumour-like conditions. The primary sites are breast (51-3%), prostate (12-17%), lungs (6-8%), gut (3-6%), kidney (3-5%), adrenal (1%), and unknown (10-11). The most common presenting signs and symptoms include diplopia with non-comitant strabismus (54%), proptosis (50%), and a palpable mass (43%). In 19-26% cases, the orbital metastasis may be the initial presentation.1 The average patient survival after the diagnosis is 13 months. Treatment includes chemotherapy, irradiation, hormone therapy, surgical excision, or observation depending on the clinical circumstances.1

Lung cancers tend to metastasize to the bone (25%), liver (20%), lymph nodes (20%), and brain (5-0%). Ocular and orbital involvement occurs in less than 1% of cases.2 The presence of distant metastasis at the time of initial diagnosis is significantly higher in small cell lung cancer (84%), while brain and orbital metastases are more common with adenocarcinomas (56%).3 There are case reports of non-large cell bronchogenic carcinomas presenting as proptosis, often acutely.3 Extensive literature survey does not reveal any report of large cell bronchogenic carcinoma with orbital metastasis and that too as the initial presentation. This makes our case a unique one.

In conclusion, physicians should consider the possibility of metastatic malignancy in patients presenting with acute onset proptosis particularly if it is unilateral.

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REFERENCES

Autoimmune Haemolytic Anaemia in a Patient with Rheumatoid Arthritis- A Rare Association

Sir,

The concept of rheumatoid arthritis (RA) over the years has witnessed a paradigm shift from an ill-defined...
joint disorder to a clinically well established multi-system disease. A variety of haematological disorders have also been reported in patients with rheumatoid arthritis.\(^1\) However, the association of RA with autoimmune hemolytic anaemia (AIHA) is extremely uncommon. We report here a patient with rheumatoid arthritis who developed a cold antibody type AIHA.

NK, a 38 years lady presented with fever for one month, jaundice since three days and altered sensorium eighteen hours prior to admission. She was diagnosed to have rheumatoid arthritis by a private practitioner and was on non-steroidal anti-inflammatory drugs and glucocortico steroid therapy for the past 5 months. She was also given anti-tubercular treatment since one month for fever and a chest X-ray that was suggestive of tuberculosis. On examination, she was pale, icteric and was in circulatory shock (blood pressure- 90/50 mmHg). Conducted breath sounds were heard in the right infra-mammary area. A non-tender hepatomegaly 4 cms was present (liver span-14 cms). There was no splenomegaly. She responded partially to painful stimuli. Deep tendon reflexes were depressed with extensor plantars response. She fulfilled four ACR criteria out of seven for rheumatoid arthritis (duration >6 weeks, synovitis, polyarticular symmetrical joint involvement and positive rheumatoid factor). Haematological investigations showed anaemia with bizarre red cell indices (Table 1). The peripheral blood smear revealed marked auto-agglutination of red blood cells (Fig. 1) with occasional nucleated red blood cell (04/100 WBC). The reticulocyte count was 0.4%. The agglutination and hematological parameters (Table 1) reverted back to normal after the blood sample was incubated at 37\(^\circ\) C.

Biochemical investigations showed a total bilirubin of 16.1 mg/dl (indirect bilirubin-11.5 mg/dl). Liver function tests revealed an ALT of 7000 u/l, AST-4120 u/l and LDH-8700 u/l. Serum creatinine was 5.0 mg/dl.

Table 1 : Hematological parameters of the patient at room temperature and at 37\(^\circ\) C. Note that the bizarre red cell indices seen at room temperature revert to normal values at 37\(^\circ\) C.

<table>
<thead>
<tr>
<th>Hematological parameters</th>
<th>Test value (Room temperature)</th>
<th>Test value (37(^\circ) C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin x (10^9g/l)</td>
<td>40</td>
<td>43</td>
</tr>
<tr>
<td>Hematocrit (l/l)</td>
<td>7.4</td>
<td>12.6</td>
</tr>
<tr>
<td>RBC count (x 10^12/l)</td>
<td>0.74</td>
<td>1.73</td>
</tr>
<tr>
<td>MCV (fl)</td>
<td>100</td>
<td>73</td>
</tr>
<tr>
<td>MCH (pg)</td>
<td>54.4</td>
<td>24.8</td>
</tr>
<tr>
<td>MCHC (g/l)</td>
<td>54.6</td>
<td>34.1</td>
</tr>
<tr>
<td>RDW (%CV)</td>
<td>12.7</td>
<td>15.7</td>
</tr>
<tr>
<td>TLC (x 10^9/l)</td>
<td>33.8</td>
<td>32.7</td>
</tr>
<tr>
<td>Platelets (x 10^9/l)</td>
<td>320</td>
<td>342</td>
</tr>
</tbody>
</table>

MCV-mean corpuscular hemoglobin, MCH-mean corpuscular hemoglobin, MCHC-mean corpuscular hemoglobin concentration, RDW-red cell distribution width, TLC-total leukocyte count

Both direct and indirect Coomb’s tests were positive. A diagnosis of RA with AIHA (cold antibody type), drug induced hepatitis with hepatic encephalopathy (ATT induced) was made. In spite of supportive management, the patient expired on the day of admission. Cold antibody titres were not done.

Haematological manifestations of rheumatoid arthritis include anaemia of chronic disease, iron deficiency anaemia, neutropenia, drug induced or autoimmune thrombocytopenia and rarely lymphomas, leukaemias and multiple myeloma.\(^1\) There have been very few case reports of auto-immune haemolytic anaemia (AIHA) occurring in patients with rheumatoid arthritis.\(^2\)

The prevalence of AIHA in patients with rheumatoid arthritis is similar to that of the general population. A chance occurrence cannot entirely be ruled out and it is also possible that both diseases may be part of a complex immunological dysregulation leading to emergence of multi-autoreactive clones. Majority of AIHAs in collagen diseases is of warm antibody type although cold antibody type anaemia has also been reported.

Coexistence of RA and AIHA has also been seen in patients on methotrexate therapy.\(^3\) Knowledge of the rare occurrence of AIHA in patients with RA may be helpful in early diagnosis and management of this condition.

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REFERENCES

Hepatitis E Infection with Bell’s Palsy

Sir,

Hepatitis E virus has been implicated in epidemics of viral hepatitis in India, South East Asia and Africa. An association of hepatitis E virus with Bell’s palsy has not been documented till date. Their co-existence may be a cause-effect relationship (hepatitis E virus causing Bell’s palsy) or it may represent a chance co-existence. A literature search did not reveal such co-existence between the two.

We had a 32 years young man who presented with yellowness of eyes and urine of 3 weeks duration and concomitant weakness of right side of face of 2 weeks duration. There were symptoms suggestive of viral prodrome three weeks back, which subsided with onset of jaundice. In the last 2 weeks he developed deviation of angle of mouth to left with loss of nasolabial fold and forehead wrinkles on the right side of face. The patient did not report hearing loss or loss of taste or visual blurring. There was no history of trauma or otologic disease.

General examination revealed icterus and confirmed the presence of right-sided facial weakness. Neurological examination revealed right-sided lower motor neuron facial palsy (Bell’s palsy) with a House-Brackmann facial nerve grading scale of 3. Abdominal examination was unremarkable except for a reduced liver span of 9 cm.

Viral hepatitis could be established by elevated liver enzymes (ALT and AST values of 1000 IU) and increased bilirubin levels. IgM antibodies against hepatitis E virus were positive. IgM antibodies against hepatitis A virus was negative. HBsAg, VDRL and HIV tests were negative. Chest radiograph was normal.

He was started on treatment for viral hepatitis with liver supportive measures. Steroids were withheld keeping in mind the possibility of exacerbation of hepatitis. Moreover, he presented to us with Bell’s palsy, which showed features of recovery. He fully recovered from viral hepatitis and Bell’s palsy in 3 weeks.

Bell’s palsy is an acute, unilateral, peripheral, LMN facial-nerve paralysis that gradually resolves over time in 80-90% of cases. Its cause is unknown, though it appears to be a polynuernititis. Possible etiologies include infections (herpetic, Lyme disease, syphilis, Epstein-Barr viral infection, HIV infection), inflammation, and microvascular disease (diabetes mellitus and hypertension). Hepatitis E virus has not yet been implicated as an inciting agent in Bell’s palsy. Researchers have investigated for serologic evidence of cytomegalovirus, rubella virus, and hepatitis A, B, and C viruses in patients with Bell’s palsy. They found an association between hepatitis B and idiopathic facial paralysis. Bell’s palsy in Hepatitis E virus infection has not been documented till date. The association may be a cause-effect relationship or it may represent a chance co-existence. Methods to detect Hepatitis E virus or antibodies within facial nerve are not available. Electrophysiological studies are indicated in patients with a House-Brackmann facial nerve grading scale of 4 or more so as to plan surgical decompression. Management of Bell’s palsy with Hepatitis E virus infection is not different from that of Bell’s palsy alone.

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