# PLASMA CELL DISORDERS DENGAN SINDROM HIPERVISKOSITAS

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## ABSTRAK

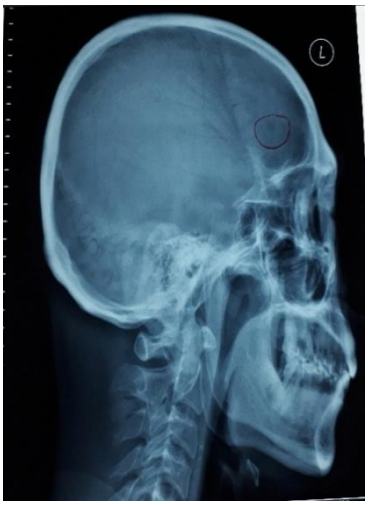
Plasma Cell Disorders adalah kelompok penyakit yang termasuk dalam garis sel B. Monoclonal gammopathy undetermined significance (MGUS), multiple myeloma (MM), Waldenstrom's macroglobulinemia (WM), primary amyloidosis, dan heavy chain disease (HCD) semua termasuk kelompok gangguan ini.Sindrom hiperviskositas merupakan kombinasi dari tanda – tanda dan gejala klinis yang terkait dengan peningkatan viskositas darah.Gejala klasik pada sindrom hiperviskositas ada tiga yaitu, kelainan neurologis, perubahan penglihatan, dan perdarahan mukosa. Dilaporkan kasus laki – laki berusia 55 tahun dengan keluhan lemas, pandangan kabur, pusing, pendengaran menurun, pendarahan pada gigi, dan buang air besar bercampur darah sejak 1 tahun yang lalu. Pemeriksaan fisik adanya konjungtiva anemis, paru dan jantung dalam batas normal, dan vital sign dalam batas normal. Pemeriksaan darah rutin didapatkan total protein 13.5 g/dL, albumin 2.9 g/dL, dan globulin 10.6 g/dL.Pemeriksan darah tepi ditemukan sel darah merah membentuk formasi rouleux, clumping/aglutinasi eritrosit, dan sel plasmasitoid.Pemeriksaan BMP didapatkan trombosit banyak dan sebagian clumping.Pemeriksaan SPE sulit dilakukan karena darah sangat pekat dan serum tidak bisa didapatkan.Hasil elektrolit tidak valid karena darah yang mengental. Pasien di diagnosa plasma cell disorders dengan sindrom hiperviskositas.

## Introduction

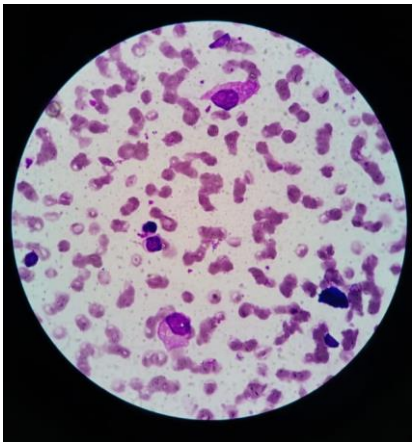
Plasma cell disorders are a group of related diseases arising from a common progenitor belonging to the B-cell lineage. They are characterized by the expansion of plasma cells in the bone marrow (BM) and nearly always accompanied by the presence of a monoclonal immunoglobulin (Ig) or Ig fragment in the serum and/or urine of patients. In this case, the patient examination of electrolytes results from hypokalemia, hyponatremia and hypocalcemia, with special note regarding its validity, make this case interesting.

**Case Presentation**

Our patients, a men 55 year old complaints of weakness, fleeing vision, pushing, decreased hearing, bleeding in the teeth, and defecating mixed with blood since 1 year ago. Physical examination of the presence of anemic conjunctivae, lungs and heart within normal limits, and vital signs within normal limits. Blood tests received hemoglobin 6.9 g /dL, platelet 115 $10^3$/ul, erythrocytes 2.00 jt/ul, hematocrit 19.5% anemia thrombocytopenia, protein 13.5 g/dL hyperproteinemia, albumin 2.9 g/dL hypoalbuminemia, hyperglobulinemia, 10.6 g/dL globulin, and 5.80 mg/Dl hypokalemiaoccasionally. Blood examination found red blood cells to make rouleux formation, clotting / agglutination of erythrocytes, and plasmacytoid cells. On examination of BMP the plasma cell yield is around 12%. SPE examination is difficult because blood is very concentrated and serum cannot be obtained. Examination of electrolytes results from hypokalemia, hyponatremia and hypocalcemia, with special note regarding its validity. Patients were diagnosed with plasma cell abnormalities with hyperviscosity syndrome.



Picture 1. firm round lucency in the left frontal os, suspicious of lytic lesions



Picture 2. Blood examination found red blood cells to make rouleux formation, clotting / agglutination of erythrocytes, and plasmacytoid cells

**Discussion**

Plasma cell disorders are a heterogeneous group of blood disorders characterized bythe detection of a monoclonal paraprotein in the serum or urine and/or the presence ofmonoclonal plasma cells in the bone marrow space or, rarely, in other tissues.Monoclonal gammopathy of undetermined significance (MGUS), multiple myeloma (MM), Waldenstrom'smacroglobulinemia (WM), primary amyloidosis, and heavy chain diseases (HCD) all belong to this group of disorders.

Hyperviscosity syndrome is a combination of clinical signs and symptoms associated with increased blood viscosity. The classic triad of symptoms includes neurologic abnormalities, vision changes, and mucocal bleeding.

**References :**

Jorge J. Castillo. Plasma Cell Disorders. 2016; 677–91