Table 1. Demographic data of the reported patients on dialysis with *Chryseobacterium meningosepticum* infection

Reference	Gender/ age	Dialysis modality	Access for dialysis	Source of infection	Consequence	Initial antibiotics	Outcome
[2]	F/33	CAPD[a]	Tenckhoff catheter	Dialysate effluent	Peritonitis	Not respond (TOB + VAN)	Not remove access, survival
[3]	F/76	CAPD	Tenckhoff catheter	Dialysate effluent Blood	Peritonitis; bacteraemia	Not respond (CTZ + GM + VAN)	Remove access, died
[3]	UA/14	CAPD	Tenckhoff catheter	Blood	Sepsis	Not respond (NAF + GM)	Remove access, died
[3]	F/63	CAPD	Tenckhoff catheter	Dialysate effluent	Peritonitis	Not respond (AZT+ PIP)	Remove access, survival
[3]	F/45	CAPD	Tenckhoff catheter	Dialysate effluent	Peritonitis	Not respond (IMI and TOB)	Not remove access, survival
[4]	M/54	CAPD	Tenckhoff catheter	Dialysate effluent	Peritonitis	Not respond (CZL + GM)	Remove access, survival
[2]	33 cases	CAPD (30 episodes) HD[b] (4 episodes)	Tenckhoff catheter NA	Dialysate effluent Blood	Peritonitis bacteraemia	NA	Remove access: NA 1 died, 31 survival.
[2]	M/74	HD	Arteriovenous graft	Blood	Bacteraemia; purulent pericarditis	Not respond (MER)	Remove access, died
[5]	M/78	CAPD	Tenckhoff catheter	Dialysate effluent	Peritonitis	Respond (CIP + RIF)	Not remove access, survival
Our case	F/77	HD	Femoral vein catheter	Tip of the catheter	Bacteraemia	Not respond (VAN + RIF)	Remove access, survival

[a]CAPD; continuous ambulatory peritoneal dialysis.
[b]HD: haemodialysis.
NA: not available; TOB: tobramycin; VAN: vancomycin; CTZ: ceftazidime; NAF: nafcillin; AZT: aztreonam; PIP: piperacillin; IMI: imipenem; CZL: cefazolin; GM: gentamicin; MER: meropenem; RIF: rifampin; CIP: ciprofloxacin.

3. Korzets Z, Maayan MC, Bernheim J. Flavobacterial peritonitis in patients treated by peritoneal dialysis. *Nephrol Dial Transplant* 1995; 10: 280–283
4. Aki ZA, Stern L, Romagnoli MF *et al*. Flavobacterium group IIb peritonitis in a patient on chronic ambulatory peritoneal dialysis. *Perit Dial Int* 1996; 16: 331–332
5. Wu VC, Tsai TJ, Wang R *et al*. Peritonitis caused by *Chryseobacterium meningosepticum* in a patient undergoing continuous ambulatory peritoneal dialysis. *J Formos Med Assoc* 2003; 102: 270–272
6. Chang JC, Hsueh PR, Wu JJ *et al*. Antimicrobial susceptibility of flavobacteria as determined by agar dilution and disk diffusion methods. *Antimicrob Agents Chemother* 1997; 41: 1301–1306

doi: 10.1093/ndtplus/sfp080

Advance Access publication 8 July 2009

Altered mental status in a case of multiple myeloma not related to a metabolic cause

Sir,
Altered mental status (AMS) in a patient with multiple myeloma (MM) is generally attributed to uremia, hypercalcemia, hyperviscosity and/or increased serum ammonia. We present an unusual case of altered mental status that could not be attributed to metabolic encephalopathy.

Our patient was a 68-year-old African American male who was admitted for AMS. The patient was asymptomatic 1 week prior to admission. On examination, no focal neurologic deficit other than altered sensorium was found. The rest of his physical examination was normal. Routine laboratory analysis revealed elevated BUN of 58 mg/dl (7–25 mg/dl), creatinine of 4.9 mg/dl (0.7–1.4 mg/dl), calcium of 12.1 mg/dl (8.5–10.3 mg/dl), total protein of 9.6 g/dl (5.5–9 g/dl) and serum ammonia of 65 mcg/dl (35–65 mcg/dl) with normal liver function tests. A toxicology screen was negative. Intravenous hydration with normal saline was initiated. Magnetic resonance imaging (MRI) of brain showed chronic microvascular ischaemic changes with no acute infarct. On cerebrospinal fluid (CSF) analysis, he was found to have elevated protein of 172 g/dl (15–45 g/dl), no pleocytosis and a negative gram stain. Polymerase chain reaction on CSF for herpes simplex was negative. Electroencephalogram (EEG) showed no seizure activity. Though all metabolic parameters normalized by the third day (creatinine of 1.3 mg/dl and calcium of 9.3 mg/dl), there was no improvement in his sensorium. To rule out paraneoplastic syndrome of unknown aetiology, a whole body CT scan was done. It showed a soft tissue mass in the pre-sacral area with multiple diffuse lytic bone lesions. The bone marrow was diagnostic for plasma cell myeloma. Serum immunofixation revealed 5050 mg/dl (700–1600 mg/dl) of monoclonal IgG. The serum viscosity was normal. A repeat lumbar puncture revealed a CSF with negative cytology, but abnormal bands of high intensity in the immunoglobulin region identical to the serum electrophoresis pattern. Four days after the normalization of all his metabolic parameters, there was still no improvement in his sensorium. The patient was started on intravenous dexamethasone for MM. After the first cycle, his sensorium returned to normal.

The most common cause of AMS in a patient with MM and acute renal failure (ARF) is metabolic encephalopathy.

Though rare, direct invasion of CNS by the myeloma cells has been reported. Currently, 70 cases of leptomeningeal myelomatosis (LMM) and intraparenchymal plasmacytoma have been published [1,2]. Schluterman *et.al*. published a case series of 23 patients. They were diagnosed up to 29 months (median, 13 months) after the initial diagnosis of MM. The presenting symptom in 65% of the patients was AMS. The CSF analysis revealed pleocytosis and/or increased protein (generally >100 g/dl) similar to our case. CSF cytology showed myeloma cells; however, it was negative in 4 out of the 23 patients at initial presentation. Unlike our patient, cranial leptomeningeal contrast enhancement was seen on MRI in as many as 70% of the cases. Our patient did not fulfil all the diagnostic criteria of LMM but did have a dramatic recovery of his sensorium after the first cycle of dexamethasone therapy. His altered mental sensorium may represent a paraneoplastic manifestation of MM or alternatively, the patient falls into the spectrum of disease activity before fully evolved LMM. He was discharged from the hospital and was referred to an oncologist for further management of MM.

In conclusion, altered mental status in a patient with multiple myeloma may be due to a paraneoplastic manifestation of MM or due to direct invasion of the CNS by myeloma cells. Intrathecal chemotherapy should be used for patients that meet the diagnostic criteria of LMM. However, even those who do not may still show a significant improvement in their neurologic status after treatment with intravenous dexamethasone.

Conflict of interest statement. None declared.

[1]Division of Nephrology
Department of Medicine
[2]Department of Pathology
St Luke's Roosevelt Hospital Center
Columbia University College of
Physicians & Surgeons, New York
USA
E-mail: gsandhu@chpnet.org

Gagangeet Sandhu[1]
Antony A. Farias[1]
Aditi Ranade[2]
Ira Meisels[1]

1. Schluterman KO, Athanasios B. Multiple myeloma invasion of the central nervous system. *Arch Neurol* 2004; 61: 1423–1429
2. Patriarca F, Zaja F, Silvestri F *et al*. Meningeal and cerebral involvement in multiple myeloma patients. *Ann Hematol* 2001; 80: 758–762

doi: 10.1093/ndtplus/sfp083

Advance Access publication 13 July 2009

Peripheral CD19+ B cells are increased in children with active steroid-sensitive nephrotic syndrome

Sir,

Pathogenesis of steroid-sensitive nephrotic syndrome (SSNS) is thought to be related mainly to T-cell dysfunction [1]. However, the beneficial use of rituximab in cases of frequently relapsing SSNS provided evidence of B cell involvement [2–4]. Our aim was, thus, to investigate prospectively the levels of the circulating CD19+ B cells in children with a first episode of SSNS in sequential stages (presentation, remission on steroids and remission off steroids).

Twenty-three children (M/F = 13/10, age = 2.5–14 years, median = 4.32 years) with a first episode of SSNS were studied; 23/23 both at presentation (before steroids initiation) and in remission on steroids (40 mg/m^2 on alternate day); 15/23 were tested as well in remission off steroids for at least 6 months. Twenty-five age-matched children who had come to the outpatient haematology clinic in order to be tested for b-thalassaemia trait were found to be negative and served as healthy controls (controls 1). Considering that the presentation of SSNS may be associated with a recent infection, mainly a respiratory tract infection, twenty age-matched children with an upper respiratory tract infection acted as a second control group (controls 2).

The percentages of CD3± T cells, as a pan T-cell marker, and the percentages of CD19+ and CD20± B cells were evaluated in all children. The above-mentioned parameters were determined in each sample by flow cytometry using the lysed whole blood method. The duochrome phycoerythrin-cyanin5 (PE-Cy5) conjugated CD3± monoclonal antibody (MoAb) and phycoerythrin (PE) conjugated CD19+ and CD20± MoAbs purchased from Beckman Coulter were used. The samples were analysed with a EPICS-XL flow cytometer. The results were expressed as percentages (%) of fluorescence-positive cells as well as actual numbers (cells/μL) of the circulating CD19+ and CD20+ B cells, based on the white blood cell count.

Statistical analysis was performed using the Statistical Package for the Social Sciences for Windows (SPSS version 11.5). The paired *t*-test and independent-samples *t*-test were used to compare differences between study groups with and without paired data, respectively. Pearson's coefficient of correlation (r) was used to determine the correlations. A $P \leq 0.05$ was considered to be statistically significant.

In 5 of 23 children with a first episode of SSNS, there was a recent history of an upper respiratory tract infection. Remission was achieved in all children within 6–15 days after steroid initiation. During remission, all patients presented normoalbuminaemia and were free of proteinuria and albuminuria. Percentages of CD3± T cells were found to be within normal limits in all patients (at presentation of SSNS, in remission still on steroids and in remission off steroids) compared with the two control groups ($P \geq 0.05$). As depicted in Figure 1, the circulating CD19+ B cells were significantly higher at presentation of SSNS (mean percentage = 18.13 ± 5.43, mean actual number = 695.34 ± 258.29) compared with remission on steroids (mean percentage = 13.57 ± 4.22 and $P < 0.0001$, mean actual number = 468.05 ± 164.69 and $P < 0.0001$), remission off steroids (mean percentage = 13.25 ± 2.32 and $P < 0.0001$, mean actual number = 414.88 ± 140.76 and $P < 0.0001$), controls 1 (mean = 13.96 ± 3.29 and $P = 0.008$, mean actual number = 442.75 ± 99.78 and $P = 0.009$) and controls 2 (mean percentage = 14.18 ± 3.6 and $P = 0.01$, mean actual number = 508.05 ± 148.9 and $P = 0.015$). During remission stages, on and off steroids, CD19+ B cells were