Infection and AIDS

**PP1144**

**Multiphasic disseminated encephalomyelitis associated with Campylobacter jejuni infection**

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**Introduction:** Acute disseminated encephalomyelitis (ADEM) typically occurs as an isolated post-infectious phenomenon. If a relapse occurs shortly after the ADEM presentation in association with steroid withdrawal, the term multiphasic disseminated encephalomyelitis (MDEM) is used.

**Case study:** Female, 26-years-old, presented with progressive paraparesis developing within a week. She also reported diarrhoea three weeks before. Neurological examination showed dysarthria, tetraparesis, ataxia and pyramidal signs, without encephalopathy. Cranial MRI showed multiple bi-hemispheric white matter lesions with enhancement. Spinal MRI, CSF and neurophysiological studies were unremarkable. The titer of anti-Campylobacter jejuni antibodies in serum was extremely high and anti-GM2 antibodies were positive, while other infectious and autoimmune studies were negative. Oral corticotherapy with gradual tapering was started with complete remission of neurological signs. After 3 months, when the patient was completing weaning of steroids, a clinical relapse occurred. MRI revealed absence of new lesions but an area of restricted diffusion in one of the previous lesions, without abnormal enhancement. Corticotherapy was restarted with slower tapering protocol. At ten month follow up the patient remains asymptomatic on 5mg prednisolone. MRI showed pronounced regression of prior lesions.

**Conclusions:** Difficulties still exist in distinguishing ADEM and MS at the initial clinical episode. C. jejuni infection has been related to different neurological syndromes linked with antiganglioside antibodies. To our knowledge, only four cases of ADEM associated with Campylobacter were previously described. We report a case of MDEM associated with C. Jejuni and anti-GM2 antibodies that extends the spectrum of neuroimmunologic complications of C. jejuni infection.

**Disclosure:** Nothing to disclose

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**PP1145**

**Brain abscess with Listeria monocytogenes following Rituximab infusion for Pemphigus vulgaris**

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**Introduction:** Immunoocompramized patients have more risk to develop meningitis or rarely brain abscess due to Listeria monocytogenes.

**Objective:** To report an immunocompramized patient who had brain abscess following two doses of Rituximab infusion. Up to our knowledge, such case has not been reported in the literatures.

**Case report:** A 50-year-old, lady, diagnosed with pemphigus vulgaris and diabetes who had been on prednisolone and azathioprine for about four years. She presented with headache, low grade fever and left sided weakness, two weeks after receiving the second dose of Rituximab infusion. Her MRI revealed enhancing space occupying lesion with multiple small vacuoles and vasogenic oedema at the right temporo-parietal area. Blood culture yielded Listeria Monocytogenes. Brain biopsy resulted in necrotic tissues with pus and inflammatory cells. She recovered after 6 weeks course of empirical antibiotics with Ampicillin and Gentamycin.

**Conclusion:** Brain abscess with Listeria is a risk that should be considered when adding Rituximab therapy to a patient who is already immunocompramized.

**Disclosure:** Nothing to disclose
PP1146

A sinister cause of cavernous sinus thrombosis

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Presentation: A 44-year-old lady with poorly controlled type 1 diabetes mellitus presented with left-sided facial pain. She was treated with metronidazole for a tooth abscess but represented one week with headache and facial drooping.

Examination: On examination she had chemosis and complete ophthalmoplegia of her left eye with an unreactive pupil. She had reduced sensation in all branches of the trigeminal nerve and had a left lower motor neurone facial palsy. There were no abnormal findings in the limbs.

Results: Blood tests revealed elevated inflammatory markers. An MRI brain revealed expansion of the left cavernous sinus with no enhancement with contrast. She was diagnosed with cavernous sinus thrombosis and treated with anticoagulation and antibiotics. A CT head revealed non-enhancing left-sided turbinates consistent with the “black turbinate sign” associated with invasive mucormycosis.

Management: Antifungal therapy was commenced. She underwent surgical debridement and biopsy which revealed necrotic nasopharyngeal tissue and fungal culture confirmed mucormycosis. She developed right-sided limb weakness and a repeat CT head revealed a right-sided pontine infarct and a new left cerebellar artery aneurysm. 9 days after initial presentation she developed newly diagnosticated focal motor right seizure and slightly right hemiparesis. MRI showed multifocal T2-high lesions mainly in the cerebral white matter, in the left hemisphere, and partly in the cerebral cortex. No gadolinium enhancement was found. CSF examination revealed that the cell count was slightly increased (8/mm³), protein level (41mg/dl), and IgG index (0.4) were normal. Encephalitis was evoked but the MR spectroscopy raise the possibility of cerebral low grade glioma. Than, a brain biopsy was necessary and revealed demyelinating pathology: demyelinating plaques involving the subcortical U-fibers with sparing of the cortex and deep gray matter. These findings were consistent with progressive multifocal leukoencephalopathy. Her symptoms were subacutely progressive, and she developed akinetic mutism two month after seizure onset. Polymerase chain reaction (PCR) was positive for JC virus.

Disclosure: Nothing to disclose

PP1147

Migraine headache and hemispheric cerebral, progressive oedema in a young woman - a challenging case

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Introduction: We report a 32-year-old woman with progressive migraine headache evolving from 9 months with no other medical complaints. Repeated neurological examination, fundoscopies was normal and NSAID just ameliorate for short time the headache. Progressive evolution determined neuroimaging investigation. Repeated (3 weekly) dynamic computed tomography revealed an increasing left hemispheric oedema. Patient developed newly diagnosticated focal motor right seizure and slightly right hemiparesis. MRI showed multifocal T2-high lesions mainly in the cerebral white matter, in the left hemisphere, and partly in the cerebral cortex. No gadolinium enhancement was found. CSF examination revealed that the cell count was slightly increased (8/mm³), protein level (41mg/dl), and IgG index (0.4) were normal. Encephalitis was evoked but the MR spectroscopy raise the possibility of cerebral low grade glioma. Than, a brain biopsy was necessary and revealed demyelinating pathology: demyelinating plaques involving the subcortical U-fibers with sparing of the cortex and deep gray matter. These findings were consistent with progressive multifocal leukoencephalopathy. Her symptoms were subacutely progressive, and she developed akinetic mutism two month after seizure onset. Polymerase chain reaction (PCR) was positive for JC virus.

Disclosure: Nothing to disclose
**PP1148**

**Progressive multifocal leukoencephalopathy in a patient with adrenal cortical tumor**

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**Introduction:** Adrenal cortical tumors can present with Cushing’s syndrome accompanied with central obesity, buffalo hump, moon face, striae, weight gain, hypertension and diabetes mellitus but also immunosuppression due to hypercortisolism. We report about a man from Bangladesh with neurofibromatosis type 1 who developed dysarthria, confusion, central facial paresis and right-sided limb weakness over a one-month period during the workup for Cushing’s syndrome.

**Methods:** CT abdomen showed an 80x60x80mm contrast enhancing tumor in the right adrenal gland with compression of the vena cava and the right liver lobe consistent with an adrenal cortical carcinoma. An initial cranial CT suggested cerebral infarctions lacking clinical correlation. A following MRI of the brain showed bilateral high signaling diffuse T2 and FLAIR white matter changes with involvement of U-fibers that corresponded to Progressive Multifocal Leukoencephalopathy (PML). The cerebrospinal fluid analysis revealed no cell elevation but there was a significantly raised albumin quote. Only 200 JC virus copies/ml were found. An initially low CD4⁺ and CD8⁺ count rose after the total resection of the right adrenal gland to normal levels. An attempt to treat with mefloquine was discontinued due to rapid deterioration.

**Results:** After an initial post-operative neurological deterioration, the patient’s condition stabilized two months post-operative, now being wheelchair dependent, in the need of 24 hour assistance, percutaneous endoscopic gastrostomy tube and using monosyllabic communication. The patient suffers of epilepsy.

**Conclusions:** This presented case is the first reported association of an adrenal cortical tumor induced hypercortisolism and PML.

**Disclosure:** K. Fink received travel compensation for a consulting assignment from BiogenIdec.

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**PP1149**

**Cerebellar form of progressive multifocal leukoencephalopathy in a patient with pulmonary sarcoidosis**

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**Introduction:** Progressive multifocal leukoencephalopathy (PML) is a demyelinating disease of the central nervous system caused by JC virus reactivation that occurs nearly exclusively in immunocompromised patients, particularly in those who are HIV positive. We report a rare case of cerebellar form PML in a patient with pulmonary sarcoidosis.

**Case report:** A 25-year-old man was admitted with gait and speech disturbances that developed over 2 months as well as pulmonary sarcoidosis from 4 years prior without therapy. Neurological examination findings revealed ataxic gait, scanning speech, horizontal nystagmus and incoordination in all extremities. Blood tests showed elevation of angiotensin-converting enzyme and lysozyme, while the percentage of T lymphocytes, especially the CD4 subset, was decreased with a normal CD4/CD8 ratio. HIV-1, and -2 antibodies and a tuberculin skin test were negative. Chest CT revealed bilateral hilar lymphadenopathy. Magnetic resonance imaging (MRI) of the brain demonstrated fluid-attenuated inversion recovery high signal intensity in the bilateral middle cerebellar peduncles extending into white matter. Despite steroid and immunoglobulin therapy, tetraplegia and eye movement impairment developed, and he became lethargic and locked-in syndrome. PML was diagnosed following PCR detection of JC virus DNA in cerebrospinal fluid. Abnormal lesions shown by MRI gradually spread to the pons, medulla, thalamus, and finally subcortical white matter of the frontal lobe after 6 months.

**Conclusions:** This case supports the hypothesis that a CD4/CD8 ratio shift in sarcoidosis with lack of JC virus-specific cytotoxic cells facilitates PML development. PML should be considered in sarcoidosis patients with white matter lesions.

**Disclosure:** Nothing to disclose
PP1150
The chameleon and master of all mimics: a case of neurosyphilis presenting as an HSV encephalitis
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Introduction: Neurosyphilis is thought to be almost extinct in the non-HIV population. It has been reported infrequently in the literature as a clinical and radiological mimic of HSV encephalitis.
Methods: A highly illustrative case of neurosyphilis presenting as a presumed viral/HSV encephalitis is described with a review of the literature.
Results: A 67-year-old white British gentleman presented with a week’s history of drowsiness, confusion, sweats and olfactory hallucinations with associated poor appetite, nausea and vomiting. He was sweaty and confused with poor attention and perseveration. There was visible focal and complex partial seizure activity. An MRI brain showed right medial temporal lobe signal change. A CSF showed 90 white cells, 90% lymphocytes with a raised protein of 0.90. He was treated with aciclovir for presumed HSV encephalitis. The CSF viral PCR was negative. A repeat CSF showed 51 white cells, 80% lymphocytes and protein 1.12 and negative PCR. He developed an erythematous maculopapular rash over his trunk. A screen for autoimmune and paraneoplastic encephalitis and was negative. A serum VDRL result was later received and was positive with an RPR of 1:128 and a positive TPPA with a titre of 1:1280, consistent with a diagnosis of recent treponemal infection. CSF VDRL serology was positive. He was treated for 21 days with high dose IV penicillin.
Conclusions: Neurosyphilis has been identified as presenting as HSV-like temporal encephalitis. We recommend VDRL testing as standard in such PCR-negative presentations, regardless of retroviral status as all the reports are in HIV-negative individuals.
Disclosure: Nothing to disclose

PP1151
Early progressive multifocal leukoencephalopathy in a patient with common variable immunodeficiency syndrome reversed under mirtazapine and mefloquine treatment
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Introduction: Demonstration of reversal of progressive multifocal leukoencephalopathy (PML) in a patient with common variable immunodeficiency syndrome (CVID), consisting of severe hypogammaglobulinemia and CD4+ T lymphocytopenia, during treatment with mirtazapine (30mg/day) and mefloquine (250mg/week) over a 12 months period.
Methods: Regular clinical examinations including Rankin scale and Barthel index, nine hole peg and box and block tests, Berg balance and 10m walking tests, and Montreal Cognitive Assessment (MOCA). Laboratory diagnostics included complete blood count and JC virus (JCV) count in cerebrospinal fluid (CSF). The non-coding control region (NCCR) of JCV, important for neurotropism and neurovirulence, was sequenced. Repetitive high-resolution MRI was performed to investigate brain lesion load.
Results: Barthel (60 to 100 points) and Rankin (4 to 2) scores, performances in nine hole peg (300 s to 52 s) and box and block (7 to 35 pieces) tests, and gait stability and walking speed improved. MOCA showed a slight but stable cognitive impairment. JCV disappeared over 3-5 months from 2,568 to 0 copies/ml. The NCCR showed genomic rearrangement. Over time, JCV with NCCR rearrangements were detected that rather resembled the archetype sequence. Cerebral MRI lesion load decreased (8.54 cm3 to 6.31 cm3) and brain atrophy became apparent.
Conclusions: The patient with congenital CVID who contracted PML with rapidly worsening neurologic deficits at age of 56 years showed improvement followed by stabilization under continuous mirtazapine and mefloquine treatment over the course of one year. This was paralleled by JCV clones with lower replication capability before JCV could not be detected anymore.
Disclosure: Nothing to disclose
**PP1152**

**Encephalic complications of sinusitis: about 13 cases**

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**Introduction:** Sinusitis can cause multiple encephalic complications, which although they are exceptional, can be threatening for life and functional prognosis.

**Observations:** We report 13 cases of adolescents aged from 13 to 19 years, who presented a not treated sinusitis, complicated by cerebral abscesses in 6 cases, cerebral empyema in 4 cases, 2 cases of encephalitis and 1 case of cerebral thrombophlebitis. The germ highlighted in four cases was Haemophilus influenzae. For other cases cultures were negative (probably caused by antibiotic taken earlier). All patients were HIV negative, and patients who presented suppurative collections received neurosurgical treatment with good outcome for 8 of them, and 5 patients died. Patients with encephalitis and cerebral thrombophlebitis received antibiotics for 45 days with good outcome.

**Results:** Intracranial suppurations from ORL origine are caused in 87% of cases to sinusitis, they are certainly caused to the late arrival of patients in our hospitals because they often come at the stage of complications. Our patients come an average 14 days after the onset of symptoms, which is consistent with the literature where some come after 1 month.

**Conclusions:** Sinusitis is a disease requiring treatment early, thus avoiding serious complications which are frequent in our country because of the late arrival of patients in hospitals.

**Disclosure:** Nothing to disclose

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**PP1153**

**Flaccid paraplegia - a consequence of medula spinal compression lymphoma with an HIV patient**

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A case of a 56-year-old HIV-positive patient with ALCL who had abnormal spinal cord compression syndrome is presented. One of the major late manifestations of the underlying disease in patients with infection by the human Immunodeficiency (HIV) is the occurrence of lymphoma. Two main types of lymphoma are Hodgkin’s lymphoma HL and non – Hodgkin’s lymphoma NHL. 56-year-old man, family-healthy, asked for doctor’s help because of subfebrile conditions that lasted for twenty days, constant pain in the muscles of arms and legs. After a routine examination by internist, performed laboratory blood tests, he was treated as likely flu situation. Fifteen days later, he felt a general weakness and severe pain in the lumbar region of the spine. Due to continuous subfebrility hospitalized at the Clinic of Infectious Diseases. During hospitalization gradually leg weakness and numbness occured that progressed to flaccid paraplegia. All laboratory test repeated. Among other tests, ELISA and “Western Blot” were done and were positive. Antiviral therapy was included. RTG of thoracolumbosacral spine was normal. Magnetic resonances imaging (MRI) of the thoracolumbar spine showed heterogeneous ventilation on spine vertebrae at different levels of the thoracolumbar spine and extradural soft tissues of T10 – L1 which made cord compression. The patient undergone surgery and laminectomy and extradural mass was removed. It was histologically confirmed that extradural mass corresponds ALCL. After that, therapy was conducted (cyclophosphamide, doxorubicin, vincristine and prednisolone) and followed by radio-therapy. Currently, patient is in generally stable condition, with flaccid paraplegia.

**Disclosure:** Nothing to disclose
PP1154

**Invasive brain aspergillosis following alemtuzumab therapy**

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**Introduction:** Despite its remarkable efficacy, alemtuzumab therapy can be accompanied by serious complications, including cytopenia and subsequent infections. Fungal infections are mainly evident during the post-treatment phase. These infections are also common within patients suffering from hematologic malignancies.

**Case report:** A 74-year-old female in remission of B cell chronic lymphocytic leukemia, submitted to alemtuzumab and intravenous steroids developed apathy and functional dependence progressing over five months. She was admitted to the hospital due to a subacute right hemiparesis. Neurological examination revealed a drowsy patient, right oral myoclonic movements, right homonymous hemianopia, and severe right hemiparesis. Analytic work-up and head CT were unremarkable, with the exception of mild elevation of D-dimers. Electroencephalogram was compatible with grade 2-3 encephalopathy. CSF samples were normal, with no identifiable bacteria, fungi or neoplastic cells, normal immunophenotyping and negative JC virus PCR. She continued worsening presenting fluctuant mental status. A second head CT showed a non-enhancing hypodensity in the right occipital lobe. Brain MRI revealed multiple hyperintense T2-signal lesions. She entered in septic shock due to ischemic colitis. Post-surgery, the patient suffered an extensive fatal left temporo-parieto-occipital hemorrhage. At postmortem, fungal hyphae were identified in some of the brain lesions and Aspergillus was also identified in lungs.

**Conclusions:** Invasive pulmonary aspergillosis and cytomegalovirus reactivation are the two most commonly seen opportunistic infections in alemtuzumab therapy. Intracranial Aspergillus infection corresponds to 10-20% of all cases of invasive aspergillosis and remains a challenging diagnosis, especially due to its non-specific clinical presentation and uncommon fungus growth in CSF.

**Disclosure:** Nothing to disclose

PP1155

**Listerial brainstem encephalitis – a case report**

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**Introduction:** Listeria monocytogenes is a common cause of meningitis in well defined risk groups including newborns, elderly people and patients with immunosupression. Brainstem encephalitis accounts for only 5-10% of listerial central nervous system infections and is observed prevalingly in middle-aged healthy adults. We report the case of a 57-year-old woman with Listerial encephalitis.

**Methods:** Case report.

**Results:** A 57-year-old woman was admitted with headache, fever, diplopia, hypesthesia on the right side of the face, slight cerebellar ataxia, mild dysarthria and dysphagia. Magnetic resonance imaging (MRI) revealed T2-hyperintense lesions and small areas of nodular enhancement reflecting microabscesses predominantly in brainstem and cerebellum, furthermore thalamus and basal ganglia. Cerebrospinal fluid (CSF) analysis showed pleocytosis of 58 leukocytes/µl with lymphocytic predominance. With isolating Listeria monocytogenes from blood culture the diagnosis could be confirmed. The patient was treated with ampicillin and trimethoprim/sulfametrol intravenous for 3 weeks, followed by oral treatment with amoxicillin/clavulanic acid for 2 weeks. The patient’s neurological condition subsequently improved and she could be discharged with full recovery. MRI controls revealed a noticeable decrease of the lesions.

**Conclusions:** Listerial encephalitis is a rare and severe infection of the brainstem with a high mortality. Diagnosis can be difficult, because the CSF analyses often show mild, nonspecific abnormalities and CSF cultures have a low sensitivity, misleading to diagnosis of viral or autoimmune encephalitis. As an early treatment is crucial for a favorable outcome, empirical treatment with appropriate antibiotics for Listeria should be administered in every patient with brainstem encephalitis.

**Disclosure:** Nothing to disclose
PP1156

Acute encephalomyelitis revealing a neurotoxocariasis

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**Background:** Toxocariasis is a parasitic zoonosis caused by larvae of Toxocara canis or Toxocara cati. Most human infections are thought to be subclinical or self-limited. Clinical involvement of the central nervous system is exceptional.

**Observation:** A 62-year-old woman with a history of hereditary multiple exostosis has presented with acute left side weakness with facial palsy. Examination noticed left flaccid hemiparesis, left peripheral facial palsy, left trigeminal nerve impairment and a thoracic sensory level (T8). Blood count revealed hypereosinophilia. Brain and spine MRI showed multiple spinal lesions (T5-T7 and T10-T12) with central nodular contrast enhancement, a pontine lacune and periventricular hyperintense lesions. CSF analysis showed 6 cells per millimeter and normal proteins. Toxocariasis serologies were positive in plasma and CSF. Intravenous Albendazol treatment (15mg/kg/d) was initiated associated to corticoids with clinical improvement and spinal lesions regression on control MRI.

**Discussion:** Neurotoxocariasis can result in varying neurological manifestations: encephalitis, strokes, meningitis and myelitis. The diagnosis of neurotoxocariasis is based on several findings: high serum titers of T-canis antibodies, eosinophilia in blood and/or CSF, clinical and radiologic improvement, as well as the normalization of the CSF parameters during antihelminthic therapy. In the literature, angiographically documented reports describe cerebral vasculitis involving small vessels occlusion and resulting in brain infarcts. Albendazol is considered as the treatment of choice. Corticosteroids are sometimes added to reduce the acute inflammatory and immunologic manifestations.

**Conclusion:** To our knowledge, this is the second reported case of toxocaral encephalomyelitis. Neurotoxocariasis should be considered in every central neurological syndrome associated with eosinophilia.

**Disclosure:** Nothing to disclose

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PP1157

Bilateral syphilitic optic neuritis: an alert to human immunodeficiency virus co-infection

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**Introduction:** Syphilis is an infection with high incidence and prevalence worldwide. The involvement of the central nervous system (CNS) can occur at any time after the initial infection with Treponema pallidum. Despite the broad spectrum of CNS manifestations described in syphilitic infection, the ocular involvement, especially if bilateral, is uncommon.

**Methods:** Case report.

**Results:** A 52-year-old man noticed decreased central vision in his left eye (LE). Three days later he noticed a similar event in the right eye (RE). He reported weight loss of 10Kg in 5 months and previous maculopapular eruption on the soles. On evaluation, he had visual acuity 1/10 in LE and 6/10 in RE, optic disc oedema and relative afferent pupillary defect in LE, bilateral central scotoma (more pronounced in LE) and normal brain magnetic resonance imaging. Laboratory tests showed positive serum Venereal Disease Research Laboratory (VDRL) and Treponema pallidum Haemagglutination as well as HIV-1 infection. Cerebrospinal fluid (CSF) showed lymphocytic pleocytosis, elevated protein levels and positive VDRL. These results confirmed the diagnosis of neurosyphilis with bilateral involvement of the optic nerves and HIV-1 co-infection. He was treated with intravenous penicillin G (14 days), with improvement of visual field defects, visual acuity and CSF changes, keeping follow-up in Infectiology.

**Conclusion:** Optic neuritis is a rare ocular manifestation of neurosyphilis. Accordingly, its occurrence, especially if bilateral and in accordance with previous case reports, should lead to the prompt investigation of concomitant infections, in particular, HIV co-infection.

**Disclosure:** Nothing to disclose
PP1158
Unusual MRI findings in an HIV positive adult with Epstein-Barr virus meningoencephalitis

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Background: Epstein-Barr virus (EBV) is ubiquitous within the general population. EBV infection is associated with multiple neurological complications, usually meningoencephalitis, being the most prevalent opportunistic viral infection among HIV infected patients.

Clinical case: A 40-year-old HIV1-positive female patient, undergoing antiretroviral therapy (ART) and poorly controlled due to treatment non-compliance (detectable viral load and CD4+ lymphocytes below 200cells/µL), was admitted for progressive headache and cognitive and behavioral changes for the previous month. Apart from confusion and mild neck stiffness, the neurological examination was otherwise normal. Cerebrospinal fluid (CSF) analysis showed mild lymphocytic pleocytosis and increased protein content. MRI revealed prominent bilateral calcifications of basal ganglia and dentate nucleus (already seen in a previous CT-scan from three months prior to admission), and de novo diffuse brain swelling and abnormal T2 hyperintensity of the caudate nucleus’ head. EBV serology was compatible with remote or reactivated infection and CSF polymerase chain reaction for EBV was highly positive, therefore establishing the diagnosis of EBV meningoencephalitis. Calcium-phosphorus abnormalities and other infectious causes were ruled out. There was a gradual recovery after reintroducing ART.

Conclusion: This case highlights atypical imaging aspects of EBV meningoencephalitis in adults: basal ganglia T2 hypersignal – observed in EBV encephalitis in children – and noticeable bilateral grey nuclei calcifications – common in pediatric acquired AIDS and described in children with chronic active EBV infection. We wonder if these unusual findings can be attributed to EBV infection alone or if they might be related to the coexistence of EBV and HIV.

Disclosure: Nothing to disclose

PP1159
Fulminant neurosyphilis and concurrent acute HIV infection presenting with new onset seizures

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Syphilis can coexist with HIV infection. Rarely both concurrent infections are diagnosed in their acute phase. Syphilis can extend to the central nervous system usually after longer period of time. It can present with seizures and focal neurological findings. We report a rare case of hyperacute neurosyphilis, with central nervous involvement in less than three weeks from infection, and concurrent HIV presenting as a fulminant picture. 24-year-old man without past medical history presented with abdominal pain, fever, vomiting and diarrhea for the past week. On exam had splenomegaly and inguinal adenopathy, later confirmed by CT abdomen – retroperitoneal and inguinal areas. Abdominal lymphadenopathy, massively elevated LDH and fever raised the suspicion of a non-Hodgkin lymphoma. Biopsy of an inguinal node and labs reveal instead an acute HIV and concurrent syphilis infection. Patient reported an unprotected sexual encounter three weeks prior. HIV-1 RNA copies/ml was over seven million. RPR high titer was positive in the blood. HIV had B genotype. Lymphocytic panel was consistent with an acute HIV infection. Early during the hospital stay had a generalized seizure. On Levitiracetam were no recurrent seizures nor abnormal electroencephalogram. MRI brain with contrast had no areas of abnormal enhancement. Spinal tap revealed elevated CSF protein and positive VDRL. Negative toxoplasma antibodies were found in blood and CSF, as well as Cryptococcus antigen. Central nervous involvement with syphilis usually takes longer from the inoculation, however massive infection can shorten the interval. Concurrent HIV in the acute phase contributed to the clinical lymphadenopathy.

Disclosure: Nothing to disclose
**PP1160**

**Dementia screening using the dementia screening profile in 12 Barangays of San Miguel Manila by Barangay health workers: a prospective study**

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**Objective:** The aim of this study is to determine the prevalence of dementia using the Dementia Screening Profile by Non Health professionals among 55-years-old and above from San Miguel, Manila.

**Methods:** This is a prospective study done in San Miguel, Manila from January 2013 up to the present. The Barangay Health Workers (BHW) conducted household screening for dwellers 55-years-old and above using Dementia Screening Profile by Non Health Professionals, Mini Mental Status Examination (MMSE-P) and the Clock Drawing Test (CDT).

**Results:** A total of 194 subjects from 12 barangays were included in the study. There were seventy four males and 120 females. Using the Dementia Screening Profile a ten item questionnaire with a cut off score of >2, the prevalence of cognitive impairment is 60%. 58 out of the 116 or 50% were screened using the Mini Mental Status Examination and the Clock Drawing Test. Out of the 58, 64% were cognitively impaired, 29% had a score below the cut-off for both MMSE and the CDT. Using the Clock Drawing Test, 34 % had a score of 3 and below.

**Conclusion:** Preliminary data showed that the prevalence of cognitive impairment was quite high at 64%. Using the Dementia Screening Profile, non health professionals may now help us determine who have signs and symptoms of cognitive impairment.

**Disclosure:** Nothing to disclose

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**PP1161**

**“Idiopathic” facial nerve palsy in hepatitis C infection**

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**Objective:** Acute idiopathic facial nerve palsy (Bell’s palsy) is the most common cause of facial paralysis, caused by inflammation of the facial nerve, not seldom after a viral prodrome. Viruses associated with Bell’s palsy are for example: HSV, mumps, EBV, CMV, HIV, Influenza, Coxsackie. Until fairly recently, acute or chronic hepatitis C infection (HCV) had not been implicated in Bell’s palsy. We present a patient with chronic hepatitis C, who developed Bell’s palsy during treatment with peginterferon-alfa (PEG-IFN-alfa).

**Case report:** A 49-year-old man presented with rightsided facial palsy since three days, which begun after he noticed pain behind his right ear. He had no recent illnesses, no headache or fever. His medical history revealed chronic HCV, for which he used PEG-IFN-alfa and Ribavirin since 6 weeks. Examination showed a facial palsy, House-Brackmann grade IV. He had no skin rash, vesicles or erythema. Medication was discontinued and three weeks later he had improved to House-Brackmann grade II.

**Results:** Quantitative HCV RNA:<12 IE/ml (2-2013: 2.9x10^6 IE/ml); genotype 3A Serology for Lyme, HIV, syphilis was negative.

**Conclusion:** PEG-IFN-alfa, together with Ribavirin, is the current treatment of choice for chronic HCV. Our patient presented with a peripheral facial nerve palsy while on peginterferon-alfa for chronic HCV. More than ten similar cases have been reported in the literature, so there seems to be an association between peripheral facial nerve palsy and chronic hepatitis C virus infection treated with interferon therapy. The underlying mechanism, however, is still poorly understood.

**Disclosure:** Nothing to disclose
**PP1162**

Aphasia, somnolence and recurring fever episodes caused by tuberculous encephalitis

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**Introduction:** A 79-year-old woman with recurring fever episodes, aphasia and somnolence was admitted. The lady reported about some cardiac problems and lung tuberculosis in the childhood and a history of several recent hospital stays because of the symptoms.

**Methods:** Case report.

**Results:** The preceding examinations included X-ray and computed tomographic scans of diverse body parts, a scintigram, transthoracal and transoesophageal echocardiography, pleura puncture, bronchoscopy, gastroscopy, bone marrow biopsy and laboratory tests without any relevant result. Nevertheless, in a magnetic resonance imaging of the cerebrum multiple contrast medium enhancing lesions could be seen. The analysis of the cerebrospinal fluid showed a increased cell count (195/3 per µl), lactat (4.4mmol/l) and total protein (171mg/dl) an a discrete decrease of glucose levels (38mg/dl) without an evidence of malignant cells. A polymerase chain reaction and culture of the cerebrospinal fluid finally detected mycobacterium bovis ssp bovis. The gamma interferon test was positive. Mycobacterium bovis ssp bovis was also found in the urine. Initially, we started an antibiotic therapy according to the guidelines with Isoniazid, Ethambutol, Pyrazinamid and Rifampicin and later changed to Moxifloxacin according to the antibiogramm because of a primary resistance of Pyrazinamid. We added Vitamin B6 and steroids. The vigilance and speech improved lightly but stayed bedridden. Finally, she died due to complications after four months.

**Conclusions:** In summary we report about a lady with encephalitis by bovine tubercle bacilli as a post primary reactivation with prolonged detection. This was caused by the rarity of this disease in Western Europe these days.

**Disclosure:** Nothing to disclose

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**PP1163**

An aggressive case of PCR negative varicella zoster virus induced transverse myelitis

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**Introduction:** Varicella zoster virus (VZV) infection is usually a mild and self-limiting disease. It has been reported that a primary infection can have a more severe clinical course in adulthood. In temperate regions such as Europe the vast majority of the population is seroconverted to VZV by adolescence. However tropical regions experience different age related VZV seroprevalence patterns.

**Case report:** We report a case of an immune competent 42 year-old female from Surinam who developed a complete tetraplegia with respiratory failure several days after a varicella virus primo infection. MRI of the spine depicted no abnormalities at first. Analysis of the cerebrospinal fluid showed a mild pleiocytosis; VZV DNA was not detected by PCR. However VZV IgG antibodies were positive. Repeat MRI of the spine ten days after admission showed a diffuse increased signal intensity throughout the spinal cord from C2 to T4 level with cord expansion and swelling. A treatment with intravenous acyclovir and steroids was started eventually followed by five cycles of plasmapheresis without neurological improvement.

**Conclusion:** It is important to stress that VZV induced myelitis as a primary infection can have an extremely aggressive course in immune competent patients. A negative VZV-PCR in the CSF does not exclude the diagnosis, and anti-VZV IgG antibodies should be routinely examined as well.

**Disclosure:** Nothing to disclose
PP1164

Acute human T-lymphotropic virus type associated myelopathy: a rare case successfully treated with intravenous pulse methylprednisolone

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PP1165

Abstract withdrawn

PP1166

Paradoxical tuberculosis-associated immune reconstitution inflammatory syndrome of central nervous system

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PP1167

Cerebral toxoplasmosis as first manifestation of AIDS

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PP1168

Herpes encephalitis: a case report

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