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Chapter 1 : Magnetic Resonance Imaging (MRI) findings in White Matter Disease of Brain

Progressive multifocal leukoencephalopathy is a rare disease caused by the reactivation of an opportunistic agent, JC virus almost in every cases in immunodeficient conditions. The disease is characterized by multifocal demyelinating lesions of the central nervous system and causes death within a few months.

Read PDF Abstract Demyelinating and dysmyelinating white matter diseases are important components of neurological problems. Therefore, the purpose of the current study is to evaluate the usefulness of MRI in determining the type and frequency of white matter disease. The MRI helped identify the sites and types of the lesion precisely and thereby. The current study demonstrated the effective use of the imaging and clinical presentation for arriving at the correct diagnosis. Introduction Demyelinating and dysmyelinating white matter diseases are important components of neurological problems. MS is the commonest of all the white matter diseases. The advent of Magnetic Resonance Imaging MRI has revolutionized the concept of diagnosing and understanding of white matter diseases. CT does not detect subtle lesions especially in stages of clinical inactivity and is not ideal in posterior fossa imaging due to the beam hardening artifacts. Moreover, with the advent of multi-echo sequences of MR, even subtle lesions of demyelination can be detected. Further the study will also look into use of MR imaging features in diagnosis of white matter disease and relate it with clinical findings. Forty eight patients visited the department during the study period. Thirty five patients were finally included in the study. The MRI examination features of these patients were highly suggestive of demyelinating or dysmyelinating diseases. All the patients were examined with 1. Head coil was used in all the patients. In case of difference of opinion, consensus was developed through discussion between the two radiologists. Among cases of acute disseminated encephalomyelitis and leukodystrophies, the diagnosis was established through typical imaging findings and clinical course without any relapse. MRI features of all the patients are shown in Table. MRI revealed multiple lesions confined to the white matter appearing hypo-intense on T1W and hyper-intense on T2W images. In all the patients at least three or more lesions were seen. The majority of the lesions were less than one cm. The commonest sites of involvement were the periventricular and pericallosal area Figures1a and b. The other sites were centrum semi-ovale, deep white matter and sub cortical white matter of left frontal lobe, parietal lobe, parieto-occipital region, left temporal lobe, cervical spinal cord, right pons and posterior part of left optic nerve. The results showed that 10 Each type of leukodystrophy presented with different frequencies and MRI findings. Three patients, all males with an average age of 4 years, suffered from metachromatic leukodystrophy. The commonest site was periventricular white matter. On the other hand, adrenoleukodystrophy was seen in three patients. The average age was three years. The abnormal signals of deep periventricular white matter specifically in trigonal area with ventriculomegaly were observed in two out of three patients. Leigh disease was diagnosed in two cases. The MR showed hyperintense signal on T2 weighted images in deep grey matter nuclei, tegmentum thalami, periventricular white matter, periequiductal region and dentate nuclei. The MRI showed periventricular hyperintensity in the frontal white matter with a cyst in the third ventricle and post contrast periventricular enhancement. Similarly, one patient had Niemen Pick Disease. MRI showed Gliosis in the frontal and temporal lobes The Central Pontine Myelinosis was ranked third among the white matter diseases identified. T2 signal changes were seen in the pons in all cases on MRI. One male patient suffered from Progressive Multifocal Leukoencephalopathy. MRI showed abnormal signal intensity in deep white matter of brain in right frontal regions, parietooccipital regions, putamen and midbrain. The signals were isointense on T1 and hyperintense on T2 weighed images. Acute Disseminated Encephalomyelitis was identified in four patients. T2 weighted and FLAIR images showed asymmetrical hyperintense signals in the subcortical white matter of frontal, parietal and occipital lobe and cerebellum. Conclusion The current study presents the proportion of patients suffering from each type of white matter disease. It also demonstrates the effective use of the imaging and clinical presentation in making the right diagnosis. White matter diseases are a heterogeneous group of

disorders, where the main imaging finding is the abnormal white matter. Although a clinical diagnosis is usually present in these disorders, imaging reinforces the diagnosis in many cases. Thereby, MRI imaging could play a pivotal role in prompt treatment of patients with white matter disease. Ind J Radiol Imag Delayed MR imaging changes in acute disseminated encephalomyelitis. Central pontine myelinolysis and its imitators: J Comput Assist Tomogr; Progressive multifocal leukoencephalopathy in 47 HIV-seropositive patients: Am J Roentgenol ; Neuroimaging in multiple sclerosis. Neurol Clin ; 13 Multiple sclerosis in North West India. Acta Neurol Scand ; MRI in the diagnosis of MS:

Chapter 2 : ICDCM Diagnosis Code : Progressive multifocal leukoencephalopathy

Progressive multifocal leukoencephalopathy is a central nervous system infection of oligodendrocytes by a human papovavirus resulting in large lesions and demyelination. The virus has been named as JC virus after John Cunningham, from whom it was first isolated.

Niemann-Pick, Fabry disease Peroxysomal e. The description of hereditary neuropathies, storage diseases and leukodystrophies can be found in chapter 1. Disorders of cortical neuronal degeneration 1. It can be caused by many different diseases. Dementias can be divided according to several criteria. The primary dementias caused by unexplained neuron loss, in secondary dementias a known central nervous system disease leading to cognitive decline. Among secondary dementias there are treatable, reversible disorders, so in each case, a detailed investigation of the patient is necessary, including laboratory and imaging tests to identify the type of dementia and the treatable forms. Based on neuropathological studies the mixed pathology is frequent, that is might be overlap between different diseases, such co-occurrence of vascular and AD pathology is not uncommon Taipa et al. Therefore, close follow-up and in case of progression early treatment is recommended Whitwell et al. The incidence of the disease increases with age. According to surveys conducted in the U. In the familial cases the genetic abnormality affects 3 genes: In view of the pathomechanism, all three lead to disease development via -amyloid formation. The presenilin is the part of -secretase. Under this hypothesis attempts were made to remove the amyloid. Neuropathology The AD belongs to tauopathies. In the background, the accumulation of abnormally phosphorilated tau protein in neurons and the formation of neurofibrillary tangles NFT are probable. The neuropathological picture shows a typical temporal development, which is paralell with the development of clinical symptoms. The Braak couple Braak and Braak identified 6 stages using a silver impregnation. In stage I-II neurofibrillary tangles is found in the entorhinal cortex, in CA1 region of hippocampus and subiculum, in stage III-IV in the limbic cortical regions cingular gyrus, orbitofrontal cortex, parahippocampal cortex, and a part of the insular cortex , in stages V-VI NFTs can be observed in the neocortical association cortical regions. Typical histological finding in AD the amyloid deposition primarily in the wall of arteries and arterioles, but veins and capillaries may be affected as well. Characteristic pathological finding in AD is the damage of the forebrain cholinergic cell groups, in particular the basal nucleus of Meynert, which provides cholinergic innervation of the cerebral cortex. The acetylcholine deficit is still one of the most important neurochemical alterations in AD, and the currently available drugs aimed to increase the level of acetylcholine. According to some data, low educational attainment, repeated head trauma, high blood cholesterol, cerebrovascular disease is a risk factor. Long-term use of non-steroidal anti-inflammatory drugs and low cholesterol levels may be protective. It is usually difficult to determine the disease onset, as the initial cognitive decline seems to be the part of the normal aging for the patient and for the family also. In the early stage of AD episodic memory impairment dominates. Loss of ability to recall of names, memorizing trivial daily events, dates, however, recall the old memories can be intact for many years. A terminally ill patient may be completely unable to be contacted and incontinent. In the clinical practice beyond the characteristic signs above, MMSE, clock drawing test might help to establish the diagnosis of AD, imaging brain MRI shows diffuse cortical atrophy, but occurrence of cerebrovascular lesions e. High level of CSF hyperphosphorilated tau protein was proved with experimental studies in AD the -amyloid is reduced , this test may help in thediagnosis. One group is the approved drugs aims to increase the level of acetylcholine in the central nervous system acetylcholinesterase inhibitors: In more severe cases, memantine NMDA receptor antagonist is suggested, also might result in a temporary improvement. The main characteristic sign is the fluctuating cognitive decline, visual hallucinations appearing in the early stage of the disease. Patients may be highly susceptible to neuroleptic treatment, which may cause akinetic-rigid symptoms, cardiac arrhythmias, neuroleptic malignant syndrome. The name of the disease was originated from neuropathology, which demonstrates Lewy bodies LB in autopsy material. The early years of DLB in a

minority of cases, but in the advanced stage of the disease in almost all patients develop symmetrical hypokinesis, rigidity, which not responds to dopaminergic treatment, even the dopaminergic treatment promotes visual hallucinations. The average survival from the onset is years. The most common and characteristic clinical presentation is the change in behavior and personality of a patient with relative good intelligence behavior variant FTD, bvFTD. Patients become uncritical, aggressive or apatic, eating disorders, such as bulimia can occur. Imaging studies show the atrophy of frontal lobe and the medial paralimbic region. The second clinical type is the primary progressive motor predominant aphasia progressive non-fluent aphasia, PNFA. In this form the patient experiences difficulty in speech initialisation, the speed is slowed down, characterized by grammatical errors, word retrieval, reading and writing is difficult. The intellect can be held relatively long time. Morphological characteristics are the atrophy of the left frontal operculum, prefrontal area and anterior insula. Third clinical manifestation is the semantic dementia SD. Behavior changes is followed by cognitive decline: Brain MRI shows asymmetric atrophy of the temporal lobes. In advanced stages loss of memory, loss of initiative, general disturbance of cognitive functions develops, patients became socially isolated clinicopathological summaries: According to neuropathological studies this name represents a heterogeneous group of diseases. Currently, the classification is based on the recently discovered pathological alterations. The ubiquitinopathy group can be subdivided into further diseases according to the immunhistochemical feature of neuronal inclusions: Secondary, potentially treatable dementias It is important to notice these forms because of differential diagnostics. We have to consider the further forms of dementias in each case. Uremia, hepatic encephalopathy, endocrine-disease hypothyreosis can be followed by dementia. Paraneopastic limbic encephalitis Long term hypoxia COPD , anema, malnutrition deficiency of B12vitamin, niacin , toxic damage e. Primary nervous system tumors astrocytoma, oligodendroglioma , primary brain lymphoma Chronic subdural hematoma, or cerebral contusion after head injurycan cause dementia as well. Rare, but treatable disease is the normal pressure hydrocephalus NPH. It probable develops due to the disturbance of CSF reabsorption. NPH is suspected, if clinical signs, especially the gait disturbance improves after lumbar drainage of ml CSF. The routine CSF pressure measurement usually is normal. Implantation of ventriculo-peritoneal shunt results in significant and long-lasting improvement of the clinical condition. Other, rare diseases with neuronal degeneration This group consists of many, usually rare, infantile and childhood-onset degenerative diseases. Generally, the diseases are characterized by an enzym defect leading to accumulation of a deleteriosus metabolite and finally to neuronal degeneration. In a part of the diseases the genetic background and metabolic dysfunction is identified, in some types are still unknown. The clinical symptoms in the majority of cases are non-specific: The diseases caused by lysosomal enzyme defects are described in section of storage diseases. Leukodystrophies, degenerative diseases of white matter The background of leukodystrophies is the disturbance in building up and degradation of central and peripheral myelin. Each form shows a similar progressive clinical course. The infantile-onset is followed by mental retardation, pyramidal signs, epilepsy, visual impairment. Less commonly, childhood and adult onset can occur with milder symptoms. Brain MRI shows extensive periventricular white matter lesions. CSF total protein is increased. Differential diagnostically important to separate them from acquired diseases, such as multiple sclerosis MS , progressive multifocal leukoencephalopathy PML , toxic encephalopathies e. The disease usually begins before half years of age. Symptoms include muscle rigidity, vomiting, somnolence, and later increased muscle tone with predilection distribution, opistotonus, pyramidal symptoms, and in advanced stage blindness, hearing loss, neuropathy areflexia. The patients usually do not survive the 1st year of age. Histology demonstrates myelin degeneration and Globoid cells giant histiocytes in the white matter. In the Schwann cells inclusions are present. Diagnosis is based on the determination of blood enzymatic activity,in a positive occasion genetic test is possible. Clinically infantile, juvenile and early adult forms are known. The infantile form starts between months of age, characterized by the retardation of psychomotor development gait disturbance, cognitive impairment, speech disorder , epilepsy may occur. The juvenile form occurs between years of age in, and adult type is known, as well. These latter are characterised by ataxia, gait disturbance,

dementia, behavioral changes, psychosis, visual impairment optic atrophy. In suspected cases determination of enzyme activity from blood is necessary, in positive cases, genetic test is recommended. Nerve biopsy is not routinely required, in positive cases, myelin damage and lysosomal inclusions in Schwann cells are shownwith metachromatic appearance. In GLD this is more effective, especially in presymptomatic stage. ERT is not applicable because of peripherally administered protein enzyme does not cross the blood-brain barrier. Non lysosomal leukodystrophies 2. A classic childhood form ALD in boys startsat years age and central nervous system involvement develops. In patients it begins with attention deficit disorder and hyperactivity. This is followed by rapid progression with spasticity, blindness, deafness, seizures, and adrenal insufficiency. Brain MRI shows a large white matter damage most pronounced in the occipital region. The adult form AMN , starts at years of age and is characterized by slow progression, spastic paraparesis, urinary, fecal incontinence and polyneuropathy. In the background the lesion of pyramidal tract, posterior column of spinal cord and axonal damage in peripheral nerves can be found. Cerebral involvement is rare. If the disease is suspected, cranial and spinal imaging and CSF test is carried out to exclude acquired lesions. ENG shows multifocal axonal damage and demyelination. The only proven therapy is HSCT in the early cases.

Chapter 3 : [Progressive multifocal leukoencephalopathy] | Read by QxMD

To report an exceptional association between X-linked hyper-IgM syndrome and progressive multifocal leukoencephalopathy. Clinical, immunological, and histological analysis. Patient A year-old male patient with X-linked hyper-IgM syndrome developed typical signs and symptoms of progressive multifocal leukoencephalopathy.

Progressive lens Save View through a progressive lens at some distance. In normal use, a much smaller section of the glass is used, so that the distortion is much smaller. Progressive lenses, also called multifocal lenses, progressive addition lenses PAL , varifocal lenses, progressive power lenses, graduated prescription lenses, or progressive spectacle lenses are corrective lenses used in eyeglasses to correct presbyopia and other disorders of accommodation. The length of the progressive power gradient on the lens surface depends on the design of the lens, with a final addition power between 0. The addition value prescribed depends on the level of presbyopia of the patient. In general the older the patient, the higher the addition. This patent included the manufacturing process and design which was however never commercialized. Unlike modern PALs, it consisted of a conical back surface and a cylindrical front with opposing axis in order to create a power progression. This was based on an arrangement of aspherical surfaces. Irving Rips at Younger Optics developed the first commercially viable blended lens in called the Younger Seamless Bifocal. The breakthrough in user adaptation and comfort, as well as peripheral and dynamic vision however occurred in with the introduction of Varilux 2, for which Maitenaz created a totally aspheric design and manufacturing process. Right and left were identical variable power lenses with distance and reading power centers in the upper and lower part of the lens, respectively. The glazing was made to accommodate eye position changes from distance viewing to reading. By tilting the reading power towards the nasal side in perfect symmetry, appropriate reading power was given to the wearer. The symmetric design, however, was difficult to accept for patients, because the eyes in general work asymmetrically. When you look right, your right eye view distal and left nasal. Modern sophisticated progressive lenses are designed asymmetrically for greater patient acceptance and include special designs to cater to many separate types of wearer application: The typical progressive lens is produced from a so-called semi-finished lens. The semi-finished lens is molded with an asymmetrical power pattern on the front. On the back side a custom surfacing is made to adjust the power for each patient. This method is however problematic, especially for astigmatic prescriptions. The reason being that the semi-finished front pattern is designed for a spherical prescription. Freeform designs are tailored to each prescription and do not have this problem. In short, the price is based on the technology used and the year the lens came to market. Advantages and use Compared to single vision lenses , progressive lenses have the power required for a presbyopic patient to have clear vision at all viewing distances, typically adjusted by tilting the head slightly. Progressive addition lenses avoid the discontinuities image-jumps sometimes found with bifocal and trifocal lenses Some people find them more cosmetically attractive. Because bifocals and related designs are associated with old age, proponents have suggested the lack of visible lines makes a progressive lens appear similar to the single vision lenses worn before the onset of presbyopia. Progressive lenses suffer regions of aberrations and geometric distortions in the periphery, leading to poor vision when turning the eyes down and to the sides. Different brands of progressive lenses have more or less of this distortion. Incorrect specification of the fitting location can cause problems for the wearer including depending on the design of the lens narrow fields of view, clear vision in one eye only, on-axis blur, and the need to alter the natural head position in order to see clearly. Progressive lenses are pricier than bifocal and single-vision lenses due to higher manufacturing and fitting costs. Some research has been conducted recently to reduce the fabrication cost by precision injection molding. Different lenses have different glazing restrictions, lens material availabilities, maximum and minimum fitting heights, prescription ranges and as such the variation in quality between higher and lower end varifocal lenses is considerable. It is advised that, when these symptoms set in, the progressive lenses be removed for a short period and replaced after symptoms have subsided.

Returning to an older prescription or different type of lens design bifocal, trifocal only serves to increase the adaptation period to the progressive lenses. Some wearers find the visual discomfort caused by these distortions outweigh the benefits of wearing PALs; this is known as progressive non-tolerance. Depth perception and distance estimation can be influenced during the adaptation period. Retrieved 24 September Refractive Eyecare for Ophthalmologists.

Chapter 4 : Progressive Multifocal Leukoencephalopathy or PML | blog.quintoapp.com

Magnetic resonance imaging and pathology of the brain in a patient with rapidly progressing progressive multifocal leukoencephalopathy. A, Magnetic resonance imaging of the brain shows large confluent lesions in the right hemisphere with a slight compression of the third ventricle on T2-weighted images.

Advanced Search Abstract Progressive multifocal leukoencephalopathy is a currently untreatable infection of the brain. Here, we demonstrate in 2 patients that treatment with interleukin 7, JC polyomavirus JCV capsid protein VP1, and a Toll-like receptor 7 agonist used as adjuvant, was well tolerated, and showed a very favorable safety profile and unexpected efficacy that warrant further investigation. The role of antibodies is less clear, as they are frequently present before and at onset of PML [5]. Currently, there is no specific antiviral or other drug to treat PML, and the only option is to restore protective immunity. The hematopoietic growth factor interleukin 7 IL-7 is crucial for homeostatic T-cell proliferation [6] and restores T-cell function, including virus-specific immunity [7]. We have treated 2 PML patients, who suffered from hereditary or acquired immunocompromise, with 3 subcutaneous injections of recombinant human IL-7 rhIL-7 [CYT] and a therapeutic vaccine consisting of JCV VP1 protein in combination with a topically administered Toll-like receptor 7 TLR7 agonist as adjuvant [8], and report the results herein. When using a nonapproved medication, the patient must be adequately informed and special authorization at the local Agency for Therapeutic Products obtained. Both patients were treated in full compliance with regulatory requirements in Germany and Switzerland. Patients and relatives were informed of the risks of the treatment, including death from PML immune reconstitution inflammatory syndrome IRIS , and signed an informed consent. Patients Clinical information and neuroimaging magnetic resonance imaging [MRI] , virological, and immunological findings are summarized in Supplementary Table 1. Both patients were uninfected with human immunodeficiency virus HIV. The treatment protocol is summarized in Figure 1. Samples were preadsorbed with soluble BK virus VP1 to compete potentially cross-reactive antibodies. Virus-specific antibody indices AI were calculated as previously described [12]. Statistical Analysis Statistical analyses were performed with Prism 5. No hematological or blood chemistry abnormalities were observed, and all compounds were tolerated well. JCV viral load testing remained negative during follow-up 12 months. Contrast-enhancing lesions never occurred in either patient before treatment over a month period. After treatment, we observed a subtle gadolinium-enhancing MRI lesion in patient 1, and clear enhancement in patient 2 indicative of an immune response in PML lesions Figure 1 B. Patient 1 significantly deteriorated during the 12 months between diagnosis and treatment SNRS score dropped from 78 to At the time of treatment, he showed bilateral cerebellar signs of the lower limbs with gait ataxia and severe aphasia with leading comprehension deficits. Following treatment, he stabilized and perceptibly improved regarding cerebellar signs, speech, and cognitive functions while remaining stable during follow-up SNRS score Patient 2, who had steadily deteriorated before treatment, developing left-sided hemiplegia and becoming largely bedridden SNRS score 49 , stabilized clinically after treatment with signs of mild neuropsychological improvement regarding alertness SNRS score T-cell responses to the recall antigen TT were normal before treatment in patient 1 and remained unchanged at the end of treatment. Intrathecal VP1-specific antibody responses were elevated before treatment in both patients and did not change after treatment Supplementary Figure 1 C. View large Download slide Treatment protocol upper scheme. Dotted gray lines represent time points of recombinant human interleukin 7 rhIL-7 injection patient 1: Day 0 is the day of the first VP1 injection. Subtle differences in schedule between the 2 patients were due to an intercurrent urinary tract infection in patient 2. A, JC polyomavirus JCV load in cerebrospinal fluid from patient 1 left and patient 2 right , before and at different time points during and after treatment. B, T2 magnetic resonance imaging MRI from patient 1 left top row and patient 2 right top row before treatment and 12 or 14 months after treatment. Contrast-enhanced T1W MRI in patient 1 left bottom row and in patient 2 right bottom row performed before and 40 days patient 1 or 17 days patient 2 after first

VP1 injection. White arrows point at gadolinium contrast enhancement indicative of neuroinflammation in the areas of progressive multifocal leukoencephalopathic lesions in both patients. The inset in the image of patient 1 focuses on the bandlike gadolinium-contrast-enhancing lesion. C, Scripps neurological rating scale before treatment and 3 months and 12 or 14 months after treatment. Proliferative responses were measured by 3H-thymidine incorporation assay.

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Chapter 5 : Publications Authored by Aschermann Zsuzsanna | PubFacts

Progressive multifocal leukoencephalopathy is a rare disease caused by the reactivation of an opportunistic agent, JC virus almost in every cases in immunodeficient conditions.

Progressive scan Save Progressive scanning alternatively referred to as noninterlaced scanning is a way of displaying, storing, or transmitting moving images in which all the lines of each frame are drawn in sequence. This is in contrast to interlaced video used in traditional analog television systems where only the odd lines, then the even lines of each frame each image called a video field are drawn alternately, so that only half the number of actual image frames are used to produce video. This rough animation compares progressive scan with interlace scan, also demonstrating the interline twitter effect associated with interlacing. On the left there are two progressive scan images. In the middle there are two interlaced images and on the right there are two images with line doublers. The original resolutions are above and the ones with spatial anti-aliasing are below. The interlaced images use half the bandwidth of the progressive ones. The images in the center column precisely duplicate the pixels of the ones on the left, but interlacing causes details to twitter. Real interlaced video blurs such details to prevent twittering, but as seen in the pictures of the lower row, such softening or anti-aliasing comes at the cost of image clarity. A line doubler shown in the bottom right picture cannot restore the previously interlaced image in the center to the full quality of the progressive image shown in the top left. Because the refresh rate has been slowed down by a factor of three, and the resolution is less than half a resolution of a typical interlaced video, the flicker in the simulated interlaced portions and also the visibility of the black lines in these examples are exaggerated. Also, the images above are based on what it would look like on a monitor that does not support interlaced scan, such as a PC monitor or an LCD or plasma-based television set, with the interlaced images displayed using the same mode as the progressive images. Usage in storing or transmitting Progressive scan is used for scanning and storing film-based material on DVDs , for example, as p 24 or p 25 formats. Some TVs and most video projectors have one or more progressive scan inputs. Before HDTV became common, few displays supported progressive-scan input. This allowed these displays to take advantage of formats like PALPlus , progressive scan DVD players , and certain video game consoles. HDTVs support the progressively scanned resolutions of p and p. The p displays are usually more expensive than the comparable lower resolution HDTV models. Computer monitors can use even greater display resolutions. The disadvantage of progressive scan is that it requires higher bandwidth than interlaced video that has the same frame size and vertical refresh rate. Because of this p is not used for broadcast. Advantages The main advantage with progressive scan is that motion appears smoother and more realistic. Frames have no interlace artifacts and can be captured for use as still photos. With progressive scan there is no necessity in intentional blurring sometimes referred to as anti-aliasing of video to reduce interline twitter and eye strain. In the case of most media, such as DVD movies and video games, the video is blurred during the authoring process itself to subdue interline twitter when played back on interlace displays. As a consequence, recovering the sharpness of the original video is impossible when the video is viewed progressively. A user-intuitive solution to this is when display hardware and video games come equipped with options to blur the video at will, or to keep it at its original sharpness. This allows the viewer to achieve the desired image sharpness with both interlaced and progressive displays. An example of a video game with this feature is Super Smash Bros. Brawl , where a "Deflicker" option exists. Ideally, "Deflicker" would be turned on when played on an interlaced display to reduce interline twitter, and off when played on a progressive display for maximum image clarity. It also offers clearer and faster results for scaling to higher resolutions than its equivalent interlaced video, such as upconverting p to display on a p HDTV. HDTVs not based on CRT technology cannot natively display interlaced video, therefore interlaced video must be deinterlaced before it is scaled and displayed.

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Chapter 6 : Progressive scan | Revolvy

Progressive multifocal leukoencephalopathy (PML) is a devastating and often fatal demyelinating disease of the central nervous system (CNS) for which effective therapies are lacking. It is caused by the replication of polyomavirus JC (JCV) in the oligodendrocytes and astrocytes leading to their.

The expression of CD40 ligand on T cells was markedly reduced in the patient. Magnetic resonance imaging indicated confluent lesions involving the majority of the right hemisphere with a mass effect. The patient died after 6 weeks despite combined antiviral treatment. Progressive multifocal leukoencephalopathy PML is a rare demyelinating disease of the central nervous system. The virus resides in the kidney in a latent form and can be reactivated when the immune system becomes compromised. B cells may transmit the virus to oligodendrocytes in the brain. Lysis of oligodendrocytes results in multiple and progressive central nervous system symptoms. Progressive multifocal leukoencephalopathy affecting patients with congenital immunodeficiencies is exceptional. Case report A year-old right-handed male patient was admitted to our neurology department because of progressive left faciobrachial paresis. His brother died of pneumonia at 9 years of age after several recurrent episodes of aphthous stomatitis, upper and lower respiratory tract infections, purulent otitis media, mastoiditis, and bacterial pneumonia. This mutation resulted in stop codon at amino acid 72 p. Expression of CD40L was tested by flow cytometry as previously described. Anti-CD69 staining was performed as a positive control to T cell activation. The patient was treated with regular monthly intravenous immunoglobulin infusions and occasionally with recombinant human granulocyte colony-stimulating factor because of recurrent neutropenia. He developed severe opportunistic infections, including Cryptococcus laurentii meningitis and esophageal candidiasis. On admission, the patient complained of dizziness and an unsteady gait, followed by weakness of his left extremities and apathy after 4 days. Neurologic examination revealed left central facial and hypoglossus nerve palsy, visual neglect, left-sided hemiparesis with increased deep tendon reflexes, the Babinski sign, and spasticity. Decreased proprioceptive sensation was found on the left side of the body. His movements, speech, and attention were slowed, but memory and body perception were maintained; neither aphasia nor apraxia was detected. Electroencephalography showed a nonspecific diffuse slowing of background activity above the right frontotemporal regions. Cerebrospinal fluid examination revealed normal protein levels 0. No intrathecal immunoglobulin synthesis or damage of blood-brain barrier were found IgG index, 0. Cultures were negative for bacteria and fungi. Antiviral antibodies for cytomegalovirus, varicella-zoster virus, herpes simplex virus 1, and herpes simplex virus 2 were not found; herpes simplex virus type 1 DNA could not be detected by polymerase chain reaction in the cerebrospinal fluid. Serology tests were negative for the human immunodeficiency virus HIV. Immunoglobulin levels in the serum were measured twice. IgG levels were normal 6. Serial cranial magnetic resonance imaging showed 2 large lesions with a high signal on T2-weighted images and a low signal on T1-weighted images in the white matter of the right frontal and temporal lobes. The lesions progressively extended into the right side of the brainstem and into the left hemisphere through the corpus callosum Figure , A. None of the lesions showed contrast enhancement, and the ipsilateral third ventricle was slightly compressed. View Large Download Magnetic resonance imaging and pathology of the brain in a patient with rapidly progressing progressive multifocal leukoencephalopathy. A, Magnetic resonance imaging of the brain shows large confluent lesions in the right hemisphere with a slight compression of the third ventricle on T2-weighted images. E and F, Numerous small and large confluent foci of the hemispheric white matter and the mesencephalon were detected arrows. Some of the foci are partly cavitated. Brain biopsy revealed hypertrophic giant astrocytes, lipid-laden macrophages with vacuoles, and scattered oligodendrocytes loaded with nuclear inclusions Figure , B and C. The oligodendrocytic inclusions were positively stained by a cross-reactive, polyclonal antibody against simian virus 40 SV40 polyoma subgroup dilution, 1: Based on the clinical, histological, and immunohistochemistry findings, PML was

diagnosed, and thus polymerase chain reaction for JCV in the cerebrospinal fluid or in situ hybridization was not performed. The patient was treated with a combination of interferon alpha 3 million IU, subcutaneously, 3 times a week and ribavirin mg daily , but he died without improvement after 6 weeks. Autopsy was performed and the formalin-fixed brain was cut 4 weeks after death. Coronal brain slices confirmed asymmetrically confluent areas of abnormal parenchyma with cavitation of the white matter in the brain and the brainstem Figure , E and F. Comment The remarkably rapid progression of PML and the unusually large lesions involving the major part of the right hemisphere suggested an unlimited propagation of JCV. While absence of contrast enhancement was typical and helpful in differentiation of PML from central nervous system lymphoma, the involvement of the frontal lobe and the mass effect were somewhat atypical. Progressive multifocal leukoencephalopathy has been increasingly detected in patients with AIDS and other secondary immunodeficiency conditions. Whether the hepatitis C virus could play a role in reactivation of JCV remains obscure. Efficient therapies have not been established for patients with PML. Antiviral agents, highly active antiretroviral treatment in AIDS, and immunotherapies like interferon alpha may be beneficial in acquired and iatrogenic immunodeficiency. Back to top Article Information Correspondence: Drs Illes and Marodi contributed equally to this work. Study concept and design: Aschermann, Gomori, Kovacs, Simon, and Illes. Analysis and interpretation of data: Pal, Komoly, Marodi, and Illes. Drafting of the manuscript: Kovacs, Pal, Simon, Marodi, and Illes. Critical revision of the manuscript for important intellectual content: Aschermann, Gomori, Komoly, Marodi, and Illes. Administrative, technical, and material support: Gomori, Kovacs, and Simon.

Chapter 7 : Publications Authored by Endre Pal | PubFacts

Progressive multifocal leukoencephalopathy (PML) is a deadly demyelinating brain disease caused by JC virus (JCV). Genomic analysis of viral isolates in these cases often shows prototype-like JCV and its variants, which is a virulent strain compared to the latent archetype virions mostly found in the kidney.

Chapter 8 : pml | Search | blog.quintoapp.com

Progressive multifocal leukoencephalopathy (PML) is a life-threatening opportunistic infection of the brain caused by JC polyomavirus (JCV) [] that occurs in various states of immunosuppression.

Chapter 9 : Endre PÃ¡l | University of Pecs - blog.quintoapp.com

We report here, HCB in 2 patients of human immunodeficiency virus (HIV)-related progressive multifocal leukoencephalopathy (PML). A year-old woman and a year-old man presented to us with asymmetrical cerebellar syndrome with pyramidal and bulbar dysfunction of subacute onset of 2 and 3 months duration, respectively.