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ORL for the family of MÄ % I say frequent pathologies for the MÄ % I say vÄf ä é ° rtigo periphered Ä é ä ~ ä "maroÄ é ä ~ is a very frequent symptom in the primary consultation care, at the same time, which I have not specifically incooked with which it was presented in most cases. The most important thing is to make a differential diagnosis with serious reality causes that require immediate benefits, such as metabolism, cardiologic and central neurological. Once discarded, we arise if it is or not of vestibular origin. The peripheral va © ä © is a satellite decoder in which different symptoms converge. The predominant symptom is an intense sensation and generally sudden of Ä é ä ~ Ä "Give of Objectsa ä ~ around us. Everything is Ä é ä ~ Ä "muevéÄ é ä ~ fictitiously from side to side. The person responsible for this symptom is the Nistagmo Horizontortortory that is always present in the acute phase of painting. This spasmodic eye movement (to the right or left, depending on the altered vestibular system) will be sitting that the feeling that everything around us is turning cap. Nystagmo will be able to observe in 40 percent of cases, fundamentally because when the patient goes to us he has already passed the acute phase and also because the Nystagmo disappears with the setting of the gaze and therefore in the presence of light. It is also accompanied by vegetative courtship (nausea, vomiting, sweat), nosta symptoms such as Otaglia or Acoufenes and shift feeling to the right or left coinciding with the altered vestibular system. The most intense and safest flowering is the image we usually have that its origin is peripheral. As a NemoThel rule remember that the peripheral vehicle is a syndrome: complete + supplied + harmonic. Ä é ä ~ ä é Complete: all the symptoms that characterize it a bit of dialogue: sensation twist of objects, nausea, acoufenes, Nystagmo. Ä é ä ~ ä é Supplied: the greater intensity of VÄf © Rtig, greater intensity of the rest of the symptoms. Ä é ä ~ ä é Harmonica: body deviations are towards affection and nystagmus towards the opposite side. Exploration of vestibulospinal reflexes to exclude a central origin. Ä é ä ~ ä é Static balance the test. Romberg Test: We imagine the cerebellum as a Ä é ä ~ Ä Ä "Thole tabueteÄ é ä ~, the three legs are their sources of information: the vision, proprioceptive sensitivity and vestibular system. If there is a vestibular irritation and we are closing your eyes, we're also canceling visual information, with which a ä ~ Ä "The Stool remains with two Legs é ä ~ and the patient falls to the side affection. To perform this test the patient will rise with the heels united and open arms (figure 12). In a first phase with open eyes and then closed for 30 seconds. The Romberg test is considered positive if the patient goes to one side when closing his eyes. Indicates vestibular pathology and / or proprioceptive. If the pathology is exclusively cerebely, cadrÄly with the open eyes that closed. Similarly, suspected central origin if the patient falls forward when you run. Ä é ä ~ ä é Test of dynamic balance. Unterberger test (Figure 12): With its arms outstretched and the eyes closed are indicated that without moving from the place that occupies, 30-40 steps on the same site in about 30 seconds. If there is a peripheral lesion, a rotation of the body axis will observe to the side of the lesion. Only deviations greater than 30 Ä ° have diagnostic value. Ä é ä ~ ä é Cerebellar coordination test. Ä é ä ~ ä "dedo-nosea ä ~, (figure 13) The patient with open eyes extends the arm and is asked to touch the tip of the nose with the yolk finger index and then the browser finger , which therefore needs to be performed but no. If this test is altered, we talk about alteration of coordination or dispequance, indicating homolateral cerebellar lesions. VÄf © Rtigo PerifÄ © Rich Benigna ParoxySitus (Vppb) is the most frequent periphery VÄf © RT. It is produced by the presence in the semicircular ducts of particles that are otoliths, which in some cephalic movements are moved from the semicircular duct causing vestibular irritation that causes an intense but short vehicle. The patient usually say that it happens when he gets up or lying on the bed, and he can also specify if he happens with the head head to the right or left. It is diagnosed with a simple maneuver, but the most interesting thing is that its treatment is based on another maneuver that aims to deposit the alleged otoliths in the specifications, where it is reabsorbed. Therefore, in this case, the use of vestibular sedatives is not necessary for treatment. The most frequent VPPB is that caused by Channelltiiasis of the subsequent semicircular conduct, responsible for 90 percent of cases. For the diagnosis, the side flexion test is executed (figure 14). He sits on the patient on the stretcher. If we want to explore the next semicircular left conduct, turn the head slightly to the right and fell the patient to the left leaving the head 30 Ä ° under the body tree. We are waiting for you in that position up to 30 seconds. In the case of VPPB of the rear rear channel, an intense but self-limiting vehicle is reproduced in a seful way with a clear Nystagmus. The maneuver does not have to be abrupt and it is important that we feel the patient we can do the VÄf © RT, but in this case we are going to be able to resolve definitively with another simple maneuver. The Transfer channel maneuver (Figure 15) starts the diagnostic test. We will set the example of a VPPB of the left rear channel. The patient is in left side decline. The head appears from the edge of the stretcher, leaving 30Ä° under the body axis. After 15-30 seconds, turn the head at the same time as the rest of the body so that one is the research, leaving the patient in a supine decline. Then turn the head to the right and then is integrated, sitting on the stretcher with your head with discretion turned on the left. Each position is maintained for at least 30 seconds. During the different phases of the maneuver, it goes © Rtig can be reproduced. In this case, reassure the patient and wait self-limiting. This maneuver must be repeated in 2 times. The patient is quoted for the following week without prescribing any kind of vestibular sedative. So only relative rest will be avoided abrupt cephalic rides. In general, improvement is usually spectacular. It is true that VPPB tends to improve spontaneously in a few months, but with relocation maneuver the recovery is almost immediate and the patient is very grateful. In the rest of the peripherals VÄf Ä © Boxes Rtigic, a vestibular sedative receives (Tientil,pezzine or Sulpiride) no more than 3-4 days, the dose is progressively reduced up to suspension. A very common mistake is to maintain this treatment for long periods of time. First of all, because we have increased the risk of extrapyramidal effects as side effects, and secondly because its prolonged use can explain unstable stability paintings after the table of VÄf Ä © Oth this end during the Orl consultation , in which the only one who indicate is to suspend pharmacological treatment. There is no rich © Rte perifÄ © VÄf which lasts uninterruptedly for months. If there may be frequent crisis or reagudizations. Once the acute picture is to overcome we have to return our vestibular system to recover the normal activity as soon as possible. This indicates a series of exercises to run at home. On some occasions, oltrichines use as trimetacidine maintenance or betaistin treatment. The discussion of the clinical case returns. | presentation | Our case refers to a 67-year-old patient with a history of hierarchy that presents an acute ataxia start, with DysmethrÄa and a sign of Romberg, after a low-energy trauma falling from a lower height, without trauma of the neo Cra and that It presents an associate a feverish syndrome of a week of evolution, which is why the discussion gifts two problems to be addressed. Goals. Ä é ä ~ ä é Describe the approach, general concepts and ataxia classification. Ä é ä ~ ä é The assessment of the probable causes of Ataxia and the feverish syndrome in our patient. Ä é ä ~ ä é Final considerations. Ataxia, derives from the Greek, a = sin and taxia = order, anomalies in the coordination of the movement. So we can say that it is a disease characterized by errors in the speed, range, direction, duration, time and force of motor activity. As regards its origin and classification, as this is a quantious quantity, we are only going to refer to the most likely pathologies, always focusing on our patient. We can classify as follows: Ä ° Ä ° Hereditary / Ä ° inherited, Ä ° Evolution time: Acute, subacuta and chronic. Ä ° Ä ° According to neurological structures involved: cerebely, vestibular and sensory. Cerebellum must be analyzed from a phylogenetic and anatom-functional point of view, so as to be able to understand the clinical expression before affecting different areas. In this way, we are with the paleorbel that functionally corresponds to the front lobe and an anatomical point of view to the splyiner in charge of the axial and extremitous motion functions as well as muscle tone. Thus his alterations will be reflected as ataxia and rigidity. Arquiceberel, involves the flappulonodular and VestTulocerbellum lobe, in association with eye movements, posture and balance, therefore, Nistgamus and ataxia can be highlighted on any affection on this level. Regarding the Neocerobro, corresponds to the next lobe and the brain, relating to voluntary movements and their pathology expressed as Dislilation, activities and dysmehous atamethrous syndromes can be grouped according to the structures concerned: 1) cerebely. Ä ° Ä ° Vermis: static Ataxia, who hits the march and the trunk. Ataxia of the trunk muscles, disartriar and hypotonia. Little or nothing affection of extremity mobility. Ä ° Ä ° Cereobellar hemisphere. Atassia Clinical, homolateral end,
DysmethrÄa, Dydoclineisy, Disinergy. Ataxia of the limbs with low alteration of muscular tone and balance. 2) Vestibular. Ä ° Ä ° Rich perifÄ © Lityrith level injuries and vestibular nerve. VÄ © Rtigo Acute Autonomic Disorders. Ä ° Ä ° Central: core and vestibular cerebral trunk stretch. 3) Ataxia sensory: Lesion of the rear ropes of the Medulus. Romberg, Tabal March, decrease in deep tendon reflections. Ataxies from lesion of the cerebellar living room manifest themselves differently according to the lesion is in the hemispheres or the worm. In the first case, there is homolateral expression (in members), which can be determined with coordination tests (DysmethrÄa, adiadocccases, intentional tremor, decomposition of movement). When Verminian, axial coordination (of the trunk) is influenced, altering the march and posture. Diandria and Nistagmus complete this syndrome, and its presence rule outside a cordless cause of the syndrome. Sensitive ataxia worsens when visual stimulus suppression (positive sign of Romberg) is associated with superficial or deep sensitive disorders and an increase in the base of As well as DysmethrÄa. It is characterized by the T-Foot type (due to lack of proprioception). We will begin to describe the most likely pathologies of the clinical case below. Ä ° Ä ° Post-traumatic ataxia. Most post-traumatic Ataxies appear immediately after trauma, which is part of post-control syndrome, thus combating vomiting and drowsiness. It may seem deferred by the development of intracranial hematoma with the presence of signs of neurological and clinical fire of endocranial hypertension. After a cervical trauma, ataxia can appear from the dissection of the vertebral artery. Ä ° Ä ° Cardiovascular. Cerebrovascular attack (ACV) is the third cause of death in our population and is associated with a high rate to invalidate neurological sequel. Most of the Ischual type (80%) for thrombotic causes (45%) and a relief (20%). In a lower percentage (20%) are hemorrhagic. The myocardial cerebellum block, without being associated with trunk cerebral ischemia, is very rare (2% of ACV ischual). The clinical picture is the abrupt installation. It is characterized by the predominance of vertiginous symptomatology to be able to simulate vestibular peripheral pathology, which are added vomiting, disartriarus, cerebellar and nistagmus ataxia. The association with a brain trunk infarction is more frequent, especially with the commitment of the Retro-Olivar region of the bulb by configuring the Walolomborg syndrome. This is characterized by the installation, abruptly, of vignetting, headache and vomiting, considered a central vestibular syndrome (Nistagmus horizonto-rotating), Arquiceberellar syndrome (changes balance); from the homolateral side to the lesion: neozerebelous syndrome (dysmethrÄa, ataxia), anesthesia, hemmara thermalGÄ Y nor deviation of the bad franc, etc.). On the contralateral side of the syringing dissociation of members' sensitivity with the loss of thermalgistic sensitivity and conservation of epicytic and deep tactile, the configuration of an alternative sensory syndrome. This diagnosis is completely remote in our patient, from the clinic, evolution and imaeologic results. Primary or metastatic tumors, the symptoms are establishment by adding slow and progressive symptoms deriving from increasing intracranial pressure, such as headaches, vomiting, papilla edema or cranial paragraph involvement. A sharp failure can appear when a hydrocephalus or bleeding develops. When they are in a cerebellar hemisphere they manifest itself from homolateral neozerebeling syndrome, while Verminian tumors produce alteration balance. Primary tumors (Emangioblastoma, Papilloma del Plesso Coroid, Astrocytoma, Medulloblastoma). Metastatic tumors are more frequently associated with breasts, lungs and hokding diseases. Paraneophassic syndromes: Paraneophal neurological syndromes are rare, the configuration of 1% of tumor complications. In 75% of cases it is the first manifestation of cancer. In this case, the existence of a primary or metastasis tumor must be excluded, in the first other case, the most accredited Pathogen mechanism is the autoimmune. Various paraneophassic neurological syndromes are recognized, the most studied of everything is the Eaton-Lambert syndrome, which as a sensory neuropathy and neuromyotonia, is associated with lung cancer variety Ä é ä ~ "Oatsoell" Paraneoplastic cerebellar degeneration is characterized by astrophy of the cerebellum bark, in which Purkinje cell affectionation is the initial fact. Clinically it is characterized A subagulation beginning of bilateral progmatice ataxia, disarter and occasionally see and diplopy. The evolution of the disease is usually slowly progressive. Months or years is usually presented before the cancer. Associated mainly to breast cancer, ovary (associated anti-me and antibodies anti R), lung and Hodgkin's disease (associated with the presence of anti-HU antibodies). The brain imaging (MRI) may be normal in a large number of patients. Signal changes, particularly in the upper worm, are at the beginning of symptoms. cerebellar atrophy is often developed in the late stages. The cephaloraquide (LCR) usually liquid shows a slight pleocytosis or high levels of protein. For all the previously expressed, these etiologies are as far away as possible causes of the clinical picture of our patient. Although causes of ataxia Recently, we described various non-infectious origin, for exposure, therefore, various diseases that even the neurological table may be associated with a febrile syndrome, reason for which they must be suspected. Recalling that until the time of the clinic and the images of the images made the main focus of the fever of our patient is breathing, playing as the community acquired pneumonia, so I will not stop to realize a review of its classification since © is not the central objective of the discussion. Infectious the most frequent causes of infectious cerebellitis are antivirals, being able to be a prion, bacterial nonchÄ © parasitic and fungal infections. They are usually given in immunosuppressed patients. The infection of the cerebellum by neurotropic viruses is usually part of a box of diffuse encephalomyelitis. However, in rare cases, at least at the start, the cerebellar commitment can be predominant. Sometimes, a cerevelitis can observe happens to loccorregional infections (otitis, pharyngo-tonsilittis, varicella zoster, etc.) and whose pathogenic mechanism could be of direct virus damage, or on behalf of a immunorality Unleashed process of this, with latency periods. Consider variables via this diagnosis by evolutionary form of the framework, the clinic and not to present a compatible LCR. AAE ¼ cerebellitis acute or acute cerebely ataxia is generated in most cases due to a post-effective cerebleshoot. A small portion is produced by direct attack of the germ over the cerebellum. It usually affects preschools. In 70% of patients to identify between 5 and 21 days before the development of an infectious process it. The most occasions is a viral infection. The onset is abrupt, with a change of the progress that is understood by the basic support up disability marked for it. It can be accompanied by dysmethrÄa, intention tremor, nystagmus, and hypotonia. Symptomatology in its highest expression is completed in the early hours. And / significant that there are no signs of intracranial hypertension, affectionation mental status, seizures and other data from a systemic disease such as fever. The improvement occurs in a few days. Full recovery of the march standardization can be delayed by three weeks and six months. With regard to the case, occurs in conjunction with an infectious process in the respiratory tract of bacterial etiology likely, this is a likely diagnosis. AAE ¼ acute infectious encephalomyelitis. Acute Disseminated Encephalomyelitis (ADEM) is an acute demyelinating disease that affects the central nervous system (CNS) is characterized by widespread neurological symptoms and signs associated with multifocal demyelinating lesions neuroimags. This affectionation can be manifested spontaneously or secondary to viral infections, Or vaccination. Ä, the pathogenesis of the EAD is not completely clarified, suggests an autoimmune inflammatory mechanism. It is mainly presented in children and young adults with non-specific clinical manifestations that include alteration of the level of consciousness, ataxia, focal engine, headache, aphasia, convulsions, alteration of sphinters control, optical neuritis or affection of others Cranial couple, who translate sn multifocal affection. Ä, the study of the CSF can be normal up to a third of the cases. In other cases, moderate pleocytosis can be observed with a predominance of lymphocytes and greater protein content. Cerebral and spinal Cream RMI is the study of images of choice in the diagnosis of EAD since it detects demyelinizing lesions. Presenting RMI and a normal cephaloraquid liquid, this pathology is discarded. Once infectious causes are already mentioned, we must mention vitamilic delay etiologies and high school than Ethylism cerebellar degeneration is produced by the degeneration of Purkinje cells in the cerebellar cortex. The areas of the media line, mainly front and higher than the most affected. Usually occurs after 10 years of alcohol consumption. In relation to its pathogenesis, it is known that the nutritional deficit (Vitamil Deficiency) and other neurotoxicities intervene on the one hand. Up to 50% of chronic ethylists has been observed. 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of vitamin B12, demyelination of cervical side beams and spinal media tonous and occasionally that of cranial nerves and peripheral and cerebral white occasionally. Among the neurological manifestations we find: cerebrial: the alteration of the mental state, cognitive deficit, depression / mania / paranoia; To cranial couples: atrophy of the optic nerve, anosmia, glossite; Cordonal cordonals: myelopathy, paresthesia, loss of proprioception (ataxia, spasty and hyperreflexia, positive sign Romberg); Autonomus "miCas (orthostatic hypotension, urinary incontinence, sexual impotence) and peripheral neuropathy (Hymoneflexia, loss of skin sensitivity, paresthesia). In RMI, to medullary can present hyperintensity in sequence T2 with V-shaped design invested in the cervical region and Toland. The dosage of vitamin B12 (normal value 200-900 pg / ml) can present false positives and negatives. The measurement of methylmalonic and homocysteine in those who have not received treatment can be performed more specific. Ä °ä¼ dÄ © Fico de Tiamina (Wernicke Encephalopathy) is an acute or subacute neuropsychiatric pathology due to the DÄ © Mnao, vitamin B1. Wernicke encephalopathy in the alcoholic patient was characterized by mental confusion, ophthalmalnopop and ataxia of march. Wernicke disease (EW) It has been described in diseases such as nervous anorexia, peritoneal dialysions, widespread neoplasms, pregnant hyperemesis or patients who intervened by gastr surgery Onestestinal. In any case, the EW still divided mainly in patients with excessive alcohol consumption. The diagnosis of EW is eminently clinical, based on the classic ataxia process, eye alterations and confusion painting. Ataxia strikes mainly to the cerebellar cortex. The areas of the media line, mainly front and higher than the most affected. Usually occurs after 10 years of alcohol consumption. 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