Diagnostico de cirrhosis hepatica alcoholica pdf gratis download para

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Given the hephetic and cardiovascular morbid that generates the MAFLD, it is important to identify the early and simple ones of these subjects for proper management and treatment that manages to reduce mortality for all causes. Tymes related to non -alcoholic steatosis should be replaced by those of steatosis associated with clinical dysfunction, since it better reflects the real conceptions about the pathological process and allows a better classification and management of patients. The MAFLD is subdivided into two histological subtypes: a) Simple steatosis, which includes patients with hephetic steatosis with or without mild inflammation; and b) stoathepatitis, characterized by the presence of inflammation and hepatocyte (ballonization) with or without fibrosis [5], [6.] by anatomopatholic examination, fatty huge includes: 1) simple steatosis, 2) steatosis with lobular inflammation or portal without Ballonizing, or 3) Steatosis with degeneration of Balonization, but without inflammation. Diagnostic steatitis requires the articulation of steatosis, lobular degeneration of ballooning and inflammation. The sky inflammation of the Hama can lead to a progressive fibrosis process that eventually and after time can lead to cirrhosis. Figure 1 shows a scheme of MAFLD's natural history. Although the mechanisms that lead to MAFLD development and progress are not completely known, it is widely accepted that initial events depend on the development of obesity and insulin resistance [7]. However, not all the natives with MAFLD tannial resistance in insulin or obesity, so it is evident that environmental and gene factors contribute to the MAFLD etiopatogan [8]. In addition, it is important to note that the return of fibrosis in patients with MAFLD was described, with percentages reported between 15 and 33%, according to the particularities of the studied group [9]. NATURAL HISTARY OF HEPHIONAL DISEASE ASSOCIATED METABILICAL DISFAN. In 11% of cases, Esteato -hepatitis progresses to cirrhosis in a 15 -year period [10]. In fact, the cirrhosis presence of cirrhosis -hepatitis among patients on the hepatica transplant waiting list the main evolutionary predictor and mafldo mortality factor is the presence of hephic fibrosis, since Fibrosis determines the risk of developing cirrhosis, hepical decalio and/or or/or the development of CHC, that is, determines the risk of hephastic mortality. It has been seen that the degree of fibrosis is independently associated with mortality by all causes, including cardiovascular, especially in the case of steatohepatitis [12], [13]. For the MAFLD, the histological system of staging of the most used fibrosis is the one described by Brunt et al. [14], where F1 is defined as perisinusoidal or periodortal fibrosis, F2 as perisinusoidal fibrosis with portal or periodport extension, F3 as fibrosis in bridges and, F4 as cirrhosis. The significant fibrosis rminis refers to a F2 or higher stadium, and advanced fibrosis to a f3 or higher stadium. Hephastic biopsy is the most of the choice to diagnose Mafld in a certain way, and to distinguish between simple steatosis and stoathepatitis, allowing . But the biopsy, in addition to being an invasive world, with potential risk of complications, also presents a series of limitations that are mainly due to sampling failures and inter and intra-observer variability. For this reason, there has been intervers in the development of all non -invasive diagnostic stoathepatitis and fibrosis as first -line tools, which help identify those subjects with a significant hepical disease and greater risk of mortality. Among these non -invasive are the synchronis synchronis based on anal parameters, as we will see later. Establishing the presence and degree of fibrosis in the MAFLD is important, since this sign is associated with mortality by all causes. The majority of patients with MAFLD are asymptomatic and the suspicion of steatosis is given by a juristic casual finding, within a health exam, of an alteraãşã £ or of the hepalic profile; or by an alteraãşã £ or in the morphology en la ecogenicidad hepatices detected by an imagen studio carried out for another reason [15], [16]. Analytically, the patients with MAFLD pueden presentar un incremento en los valores de concentración de aminotransferasas, siendo la MAFLD la causa principal de una elevación persistent de these liver enzymes. In embargo, normal values of aminotransferasasases in the exclusive presence of MAFLD; of hecho, la mayoría de estos patients presentan unas aminotransferasas deemed normal [17], [18]. Cuando están inscribed, the increase in concentration of both enzymes, alanine aminotransferasa (ALT) and aspartate aminotransferasa (AST), is ligermente superior al limite superior of the reference interval. La relación AST/ALT suele ser inferior de 1 en estadios initiales de la esteatosis y, si esta relación se invierte, puede be sign of evolution to fibrosis. Sin embargo, el grado de elevation de las aminotransferasasas no se relation con el grado de fibrosis o inflamación hepatic. Similarly to las aminotransferasasas, la gamma-glutamiltransferasa (GGT) también puede finds itself high in a frecuente way in patients with EHNA, and su elevation has been related to fibrosis con riesgo [19]. For its part, la phosphatasa alkaline también puede be ligermente high, aunque rarely es la sole magnitud experimenta. Otro hallazgo anal que se observ con frecuencia es la elevation de la concentración serérica de ferritina y del indice de saturación de la transferrina, sin que se haya demostrado un increase parallel en los deposits hepatica de hierro [20]. Something similar occurs with the presence of serum titles coming from autoanticuerpos, which appears con cierta frecuencia en la MAFLD y se considern un epifenómeno [21], aunque hay studios que les atribuyen una implicación pronostica a la enfermedad hepatic [22], [23]. La bilirubin y la albumina no suelen alterse,In patients with cirrhosis, who may also have an increase in Protrombin, thrombocytopenia and availability of neutropenia, low cost and security; However, its main limitation is that it has limited sensitivity to mild steatosis detection, it does not detect steatosis if this is less than 20% or indivstments with BMI> 40 [24] Not to mention that it is not able to differentiate between simple steatosis and success. A ranked element in the diagnosis of MAFLD is the differentiating differentiation of simple steatosis and the hephonial fibrosis status and status, since patients with steat -hepatitis and fibrosis are those who are The greatest risk of developing liver complications and cardiovascular disease. As we mentioned earlier, the choice of evaluating the degree of histological lesion still is difficult to perform in most patients, restricting its use to some specific situations. Thus, it is advisable to consider the accomplishment of hephaned biion in these cases of fibrosis presence, gravity of the disease or coexistance of other liver diseases and/or In patients with higher risk of steampatitis and/or advanced fibrosis [5], [25], [26], which can be identified previously accessible. Ah, the usefulness of all the assessment of diagnosis and invasive mafldness that helps characterize patients, determine the risk of progress of hepical affection [16] and prevent hepical biion in cases £ o methods in the invasive pueden be divided into of the large groups: the officers “biological” the serums the and officers “physical” the radiological. among the latter is encuentran la elastografía de transición (FibroScan®) y la elastografía por resonancia magnetic. the biochemical serum components is taking gran auge gracias la validez, reproducibilidad y sencillez en su realization, lo que Allowía establecerlos como una herramienta de primera línea both in atención primaria e en specializ para lación identifies of patients who requieren estudios additionales. a continuation, hablaremos de los different copies sericos described for the detección de los three histologic components characteristic of la mafld (esteatosis, inflamación y fibrosis).• la inclusivesión de la ggt en el profile hepatic basic puede ayudar a clarify el diagnostic y pronostico de la MAFLD.• en el manejo de la jofld es useful evaluidad se han desarrollado varios serológicos para predecir la existencia de esteatosis liver: index of fatty liver (fli) [27], steatotest [28], NAFLD-Liver fat score [29] y hepatic steatosis index (hsi) [30.] en la tabla 2 se encuentra un curriculum de las variables que constituyen cadao. estos indices han were validated both in población general e en población con obesidad; se asocian a la resistencia a la insulina y predicen de manera futuros metabolic events, hepatica y cardiovasculares. indican de manera fiable la presencia de esteatosis pero no allown cuantifique el contenido de grasa liver [31.] biochemical indices para la predicción de esteatosis liver. variable markers and journals of oysters ref. 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A goal determined for this product a 0.82 auroc and a sensitivity and specificity of 75 and 77%, respectively, to predict the steat -hepatitis, ie a limited diagnosis precise; In addition, the published cut values are very variable, â € ught € ‹[34]. However, by including this marker in Parana © is, diagnostic effectiveness can increase to 0.92 [35], so that future studies are required to determine the scope of the utility of this marker. Other markers studied are hormones, such as Fibrobast Growth Factor 21 (FGF21) and adiponectin, but with very low diagnosis. Also studied stroke markers were studied and inflammation, such as interleukin 6 and â € hydons of necrosis tumor. All of them were evaluated in short sucks or pilot studies in heterogy groups from patients with contradictory results, and none can differentiate simple sensitivity and specificity steatosis NASH [36]. To improve the precise diagnosis of the markers, predictive models have been developed that combine any of these sylogical biomarkers with analytical and variable clinic paranters at the moment, they are not recommended in the chrostic sculptic [37]. The studies based on Metabolmon, the development allowed by a Spanish group, from a test (owl fan test) that allows the differentiation of steat -hepatitis from simple steatosis of simple sensitive steatosis and specificity (ROA greater than 0.8); This test was obtained by dwarf of samples of 465 patients [38] and was validated in blind studies in 2 independent cohorts [39]. The limits of use in other ethnic groups and in zirtam ad setnenopmoc oÉÃs euq ,soterid serodacram)2 ;e ,tla e tsa ,aniburrilib ,animo£Ãbla omoc omoc ,acit¡Ãpeh oÉÃ§Ânuf Ã sodanoicaler ,soteridni serodacraM)1 :me sodacifissalc res medop acit©Ãfeh esorbif ad socir serodacraM ©Á yS .srekraM hciR ©Á yS .aicnçÃveler ednarg meriuqda sele euq sesaf sassen etneicap o racitsongaid ,acit¡Ãmotnissa aigolotap amu ©Á ,lareg arger omoc ,omoC .odacifidom res edop etneicap mu ed atsinorp o odnauq)avitacifingis esorbif uo 2F esorbif(secocerp siam sesaf san ©Ã ,otnatne oN .)acit©Ãfeh aitapolafecne ,ogaf´Ãse ed sezirav a odived avitsegid aigarromeh ,eticsa(oditapotapeh ues ed oÉÃ§Âasnepmocsed amugla uotneserpa ¡Ãj elE odnauq acit©Ãfeh esorric ed etneicap mu racitsongaid licÃfid ©Ã oÉÃn ,acinÃlc a araP .]31[,]21[sasuac sa sadot rop ralucsavoidrac edadilatrom e ocsir od etnemetnednepedni odaicossa odis met esorbif ed uarg o ,DLFAM od osac on etnemlaicepse ,ossid m©ÃlA .esorbif ed uarg ues ocin´Ârc odagÃf moc etneicap mu ed etnaid ovitom esse rop ,]34[,]24[,]14[,]21[acit©Ãfeh a§Âneod A sadoT .etitapeh- otaetse ad samotnis sod aciss¡Ãlc acit¡Ãrp an otcapmi o radilav araP sodutse arap soir¡Ãssecen oÉÃs selE .atnemarref atsed oÉÃ§Âatnemelpmi ad edadilitu a railava arap ,sotis³Áporp sortuo ertne ,met euq ,"h¡Ãj ed oÉÃigeR" odamahc ,ociRpinU ovitacitlum odutse mu oda§Ânal iof ,)DPES(avitsegiD aigolotaP ed alohnapsE edadeicoS ad ,etnemeneceR .odacram EC o moc odazilaicremoc e odadilav ©Ã etset O .]04[roiretna ocits³Ângaid ed omtirogla on)c1AbH(adalisocilg anibolgomeh e sesanimasnart odnaroprocni sodivloser etnemetnecer marof sodalortnoc oÉÃn sonaibaid sO ed oÉÃ§Âanibmoc amu masu euq ,selpmis soledoM sotsiverp sopurg sednarg siod ed setnedecerp ,ada§Ânava esorbif ,ada§Ânava esorbif)ocin-Ãrulaih odic-anim£Ãbla ,tsaatsahs]75[1-pmut ,odic anirulaih anilubolgorcam-2 ±Â II otcepsorbif]65[,]84[osep ,sateuqalp ,anitirref ,tla ,tsa ,asoculgdlfan ortem´Ârbif]55[teletalp ,loretselohc ,tggsnrof]45[nilubolg-¾Å®Ã ,TSA]25[,]15[xes dna enirulayh odidic £Ãanilubolgorcam-2 ±Â ®Ã ,tgg ,niburilibaerocsapeh]05[tlA ,niburiliB ,TGG ,1aopA ,nibolgotpaH ,A anilubolgorcam-2 ±Â ®Ãsetitca]94[oxes ,aniburrilib ,tgg ,1a opa ,anibolgotpah ,anilubolgorcam-2 ±Â ®Äa tsetorbif]86[, 84[SALETALP ,TSA)IRPA(sateuqalp ed axat ed ecidnÃ arap TSA]66[MD ,cmitlA ,tsaerocS draB]56[,]46[1-PMÚT ,pniiiP ,ocinÃrulaiH odiC £ÃA)FLE(adaromirpa acit¡Ãpeh esorbiF]26[,]85 [arE[arE[arE" tlA ,tsA ,tsaelP)4-bif(4-esorbiF]74[,]64[CMI ,edadI-animo£ÃblA ,TLA ,TSA ,sateuqalp ,esocilG)SFN(erocS esorbiF dlfAN.feRslebairav manoicerid socimÃuqoib soteridni serodacram sO socimÃuqoib serodacraM .siev¡Ãirav sartuo e soterid ,soteridni socimÃuqoib serodacram me sadacifissalc meµÂpmoc sa euqâá siev¡Ãirav sa odnartsom ,lacipeh esorbif ad avisavni oÉÃn oÉÃ§Âailava a arap osson O .3 alebaT an sodimuser oÉÃtse euq e ,]86[,]76[,]66 [,]56[,]46[,]36[,]26[,]16[,]06[,]95[,]85[,]75[,]65[,]55[,]45[,]35[,]25[,]15[,]05[,]94[,]84[,]74[,]64[etneicap od etneilc od sacitsÃretcarac sa moc abalÃs ed serodacram sesse manibmoc euq sovitiderp soledom oÉÃs odicelebatse iof euq o ,otnatroP .lev¡Ájesed acits³Ângaid aicnªÃicife ajnita seled muhnen euq mes ,DLFAM moc setneicap me sodailava marof serodacram sessed sosoremuN .]54[,]44[negelohc ed sopitbus e zirtam ad sesanietorpolatem ,anirulaih omoc routine anals with clinical varies, and; b) Complex models, which use synchroni synchronous related to the process and degradation of extracellular matrix (direct). It is important to keep in mind that those included in these fibrosis ps. Rmacos with hephetic toxicity, Gilbert syndrome, hemóis [56] in terms of direct officiais, these tend to better reflect the extracellular matrix replacement rate than the amount of matrix deposited, so concentrations They are usually greater when the degree of inflammation is greater and, on the contrary, a high matrix deposit can be underestimated if the inflammation is minimal. On the other hand, none of the officiais is specific to Hígado and can be altered in the presence of other diseases and states (postoperative) associated with fibrosis, so the presence of concurrent extrahepatic inflammation can contribute to the increase in syntheic concentrations of the marker. Trading commentary, the release of some officiais can be affected by the dysfunction of the sinusoidal endothelial cells or the decrease in the biliary excretion of the fogged disease itself. In any case, and especially for the simple models, given their accessibility, under cost and vigor negative predictive value, they recommend their use as an initial diagnostic approximation, not forgetting to make a critical interpretation of its results. Table 4 shows the diagnostic value at the high cut -out points of advanced fibrosis fibrosis in patients with MAFLD. Valorizaã§ã £ or two high cut points ?atse roP .]16[DLFAM od aigoloite an sanepa oÉÃn e esorbif noif ajuc me ,oÉÃ§Âatpada a ©Ã odacilbup ©Ã odacilbup e rossimorP .otnemom etsen lacipeh esorbif ad oteridni rodacram rohlem o odnes etnemlevavorp ,]06[sair¡Âte saxiaf e CMI ,setebaid ed aicnªÃsua uo a§Âneserp ad etnemetnednepedni ,ada§Ânava esorbif racitsongaid/ratracsed arap sFN so e 4-BIF o euq)74,0 >(otla o e)21,0

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