Hyper beta 2 globulinémie

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Hyper beta 2 globulinémie

What is beta 1 and beta 2 globulin. Hyper beta 2 globulin n. What is beta 2 globulin. What does high beta 1 globulin mean. What is a high beta 2 globulin. Hyper beta 2 globulinémie modérée. Hyper beta 2 globulinémie isolée.

L'interprétation se fait avec la connaissance théorique que l'on a de la composition de chaque pic appliquée au tracé observé. Il faut bien évidemment tout remettre dans le contexte clinique. La majorité (proportion et quantité) des protéines contenues dans les pics alpha (1 et 2) et beta (1 et 2) est synthétisée par le foie. - On y compte surtout les protéines de phase aiguë et lipoprotéines. - Le pic béta2 comprend aussi les immunoglobulines M et A, produites par les lymphocytes B. IgA et IgM sont souvent à cheval sur le pic en gamma. Le pic en gamma contient surtout les immunoglobulines G (synthèse par les lymphocytes B et plasmocytes) Des protéines ''anormales'', produites par les lymphocytes/plasmocytes néoplasiques peuvent se retrouver de façon anecdotique partout entre alpha2 et gamma (ex : IgE, chaines légères, dimérisation des IgA ...) 2. Indications La réalisation d'une électrophorèse fait partie de plusieurs bilans de base à notre laboratoire ORBIO. Son évaluation morphologique peut donner des informations inattendues dans un contexte clinique donné, renforcer une hypothèse clinique ou encore la rendre moins probable ou l'exclure. On s'intéressera au patron global des différentes fractions ou à des pics bien particuliers. 3. Conditions de prélèvement Sang veineux Tube sec : Il est fortement recommandé d'envoyer le sérum après décantation et séparation. Prélèvement non hémolysé et sans lipémie : - La présence de ces deux changements préanalytiques vient compromettre l'interprétation. - L'hémoglobine migre en béta globulines, souvent en béta2 globuline (mais aussi béta1). En plus de migrer en béta globuline, elle peut aussi s'associer à d'autres protéines et migrer en des régions non prévisibles. - La lipémie (surtout cholestérol puis triglycérides) donne souvent lieu à un pic en albumine. Si l'albuminémie est estimée à partir de ce pic et de la valeur des protéines totales, on observe une ''hyperalbuminémie'' bien au delà des valeurs physiologiques. Peut aussi migrer en région des lipoprotéines. - Tous les autres pics doivent être évalués avec réserve, aussi bien qualitativement que quantitativement. Il est malheureusement préférable de recommencer l'analyse sur un autre prélèvement si l'information qu'elle peut apporter est pertinente. 4. Interprétation Le contexte clinique, comme toujours, permet une meilleure interprétation. Dans certains cas, le profil de l'électrophorèse peut aussi orienter vers des hypothèses non envisagées. 1er cas : observation d'une hyperglobulinémie. On parle de gammopathie lorsqu'un (au moins) des pics de globulines est élevé. Pic (s) polyclonal (aux) en béta 1 et/ou 2 globulines : - polyclonal car sa base est large si on la compare à celle de l'albumine - témoigne d'une inflammation chronique retrouvée dans le cas de maladies infectieuses chroniques (bactériennes, virales, fongiques, protozoose, parasitaire) surtout mais aussi de néoplasmes, de maladies hépatiques ou de maladies à médiation immune. On comprend donc qu'un tel pic est assez peu spécifique. Un pic polyclonal en région gamma est également souvent présent et peut signer : - Chez le chien : dirofilariose, ehrlichiose - Chez le chat : Péritionite Infectieuse Féline (PIF), FIV, FeLV, hemobartonellose - Signe une stimulation antigénique persistante Les pics béta2 et gamma peuvent sembler ''fusionnés'' ou forment une sorte d'escalier - Assez bon témoin d'une anomalie hépatique (hépatite chronique active) Exemples : cholangite, cholangiohépatite Un pic est présent en alpha2 ; chez le chien (remarquer qu'on ne parle pas de pic mono- ou polyclonal dans ce cas) - Peut suggérer la présence d'une inflammation aigue ; la mesure de la CRP est envisageable. Une inflammation chronique (voir plus haut) est aussi possible - Syndrome néphrotique (hypercholesterolemie, hypoalbuminémie +/- azotémie) Artéfact lié à l'hémolyse ou à la lipémie : en particulier en béta (1 et/ou 2) pour l'hémoglobine et à divers localisations pour la lactescence. Un pic d'apparence ''monoclonal'' est présent - Il est en région beta ou gamma, rarement alpha. Sa base est dite étroite, lorsqu'on la compare à celle de l'albumine - Le plus souvent : le pic est réellement monoclonal. Néoplasie d'origine lymphoïde Myélome multiple/tumeur des plasmocytes ; certains lymphomes ou leucémies de type B. - En béta il est le plus probablement vraiment monoclonal (IgA et IgM) - En gamma(IgG) il faut exclure un pic oligoclonal mais un néoplasme est tout de même possible Il s'agit d'un pic oligoclonal (produit par plusieurs clones de lymphocytes/plasmocytes) - Le processus sous jacent est non néoplasique - Il s'agit d'une hyperglobulinémie réactionnelle Maladies infectieuses : ehrlichiose, bartonellose, autre Autres : gastroentérocolite plasmocytaire. 2ème cas : Hypoglobulinémie (peu fréquent) observée dans le cas de : Déficiences en immunoglobulines - Congénitales (déficiences sélectives ou combinées) : RARE - Défaut de transfert colostral chez le jeune (Certaines enzymes peuvent être explorées en fonction de l'espèce) - Acquises (chimiothérapie, radiothérapie). Chez le chiot dans le cas de Maladie de Carré ou de Parvovirose, diarrhée chronique. - Baisse des gamma-globulines. Maladie hépatique : possible baisse des alpha et beta-globulines. A2MAvailable structuresPDBOrtholog search: PDBe RCSB List of PDB id codes4ACQ, 2P9R, 1BV8IdentifiersAliasesA2M, A2MD, CPAMD5, FWP007, S863-7, transcuprein, alpha-2-macroglobulinExternal IDsOMIM: 103950 MGI: 2449119 HomoloGene: 37248 GeneCards: A2M Gene location (Human)Chr.Chromosome 12 (human)[1]Band12p13.31Start9,067,664 bp[1]End9,116,229 bp[1]Gene location (Mouse)Chr.Chromosome 6 (mouse)[2]Band6 F1|6 57.49 cMStart121,635,376 bp[2]End121,679,227 bp[2]RNA expression patternBgeeTop expressed in lung upper lobe of lung right lung upper lobe of left lung liver visceral pleura kidney thoracic aorta urinary bladderMore reference expression dataBioGPSn/aGene ontologyMolecular function peptidase inhibitor activity interleukin-8 binding growth factor binding calcium-dependent protein binding protease binding GO:0001948 protein binding serine-type endopeptidase inhibitor activity enzyme binding tumor necrosis factor binding endopeptidase inhibitor activity interleukin-1 binding signaling receptor binding GO:0005097, GO:0005099, GO:0005100 GTPase activator activity Cellular component cytosol blood microparticle extracellular region extracellular exosome platelet alpha granule lumen extracellular space collagen-containing extracellular matrix Biological process blood coagulation, intrinsic pathway negative regulation of peptidase activity platelet degranulation extracellular matrix disassembly stem cell differentiation negative regulation of complement activation, lectin pathway regulation of small GTPase mediated signal transduction GO:0032320, GO:0032321, GO:0032855, GO:0043089, GO:0032854 positive regulation of GTPase activity negative regulation of endopeptidase activity Sources:Amigo / QuickGOOrthologsSpeciesHumanMouseEntrez2232345EnsemblENSG00000175899ENSMUSG00000030111UniProtP01023Q6GQT1RefSeq (mRNA)NM_000014NM_001347423NM_001347424NM_001347425NM_175628RefSeq (protein)NP_000005NP_001334352NP_001334353NP_001334354NP_783327Location (UCSC)Chr 12: 9.07 – 9.12 MbChr 6: 121.64 – 121.68 MbPubMed search[3][4]WikidataView/Edit HumanView/Edit Mouse alpha-2-Macroglobulin (α2M) is a large (720 KDa) plasma protein found in the blood. It is mainly produced by the liver, and also locally synthesized by macrophages, fibroblasts, and adrenocortical cells. In humans it is encoded by the A2M gene. Alpha 2 macroglobulin acts as an antiprotease and is able to inactivate an enormous variety of proteinases. It functions as an inhibitor of fibrinolysis by inhibiting plasmin and kallikrein. It functions as an inhibitor of coagulation by inhibiting thrombin. Alpha-2-macroglobulin may act as a carrier protein because it also binds to numerous growth factors and cytokines, such as platelet-derived growth factor, basic fibroblast growth factor, TGF-β, insulin, and IL-1β. No specific deficiency with associated disease has been recognized, and no disease state is attributed to low concentrations of alpha-2-macroglobulin. The concentration of alpha-2-macroglobulin rises 10-fold or more in the nephrotic syndrome when other lower molecular weight proteins are lost in the urine. The loss of alpha-2-macroglobulin into urine is prevented by its large size. The net result is that alpha-2-macroglobulin reaches serum levels equal to or greater than those of albumin in the nephrotic syndrome, which has the effect of maintaining oncotic pressure. Structure Human alpha-2-macroglobulin is composed of four identical subunits bound together by -S-S- bonds.[5][6] In addition to tetrameric forms of alpha-2-macroglobulin, dimeric, and more recently monomeric aM protease inhibitors have been identified.[7][8] Each monomer of human alpha-2-macroglobulin is composed of several functional domains, including macroglobulin domains, a thiol ester-containing domain and a receptor-binding domain.[9] Overall, alpha-2-Macroglobulin is the largest major nonimmunoglobulin protein in human plasma. The amino acid sequence of alpha-2-macroglobulin has been shown to be 71% the same as that of the Pregnancy zone protein.[10] Function The alpha-macroglobulin (aM) family of proteins includes protease inhibitors,[11] typified by the human tetrameric alpha-2-macroglobulin (a2M); they belong to the MEROPS proteinase inhibitor family I39, clan IL. These protease inhibitors share several defining properties, which include (i) the ability to inhibit proteases from all catalytic classes, (ii) the presence of a 'bait region' (aka. a sequence of amino acids in an α2-macroglobulin molecule, or a homologous protein, that contains scissile peptide bonds for those proteinases that it inhibits) and a thiol ester, (iii) a similar protease inhibitory mechanism and (iv) the inactivation of the inhibitory capacity by reaction of the thiol ester with small primary amines. aM protease inhibitors inhibit by steric hindrance.[12] The mechanism involves protease cleavage of the bait region, a segment of the aM that is particularly susceptible to proteolytic cleavage, which initiates a conformational change such that the aM collapses about the protease. In the resulting aM-protease complex, the active site of the protease is sterically shielded, thus substantially decreasing access to protein substrates. Two additional events occur as a consequence of bait region cleavage, namely (i) the h-cysteinyl-g-glutamyl thiol ester becomes highly reactive and (ii) a major conformational change exposes a conserved COOH-terminal receptor binding domain [13] (RBD). RBD exposure allows the aM protease complex to bind to clearance receptors and be removed from circulation.[14] Tetrameric, dimeric, and, more recently, monomeric aM protease inhibitors have been identified.[7][8] alpha-2-Macroglobulin is able to inactivate an enormous variety of proteinases (including serine-, cysteine-, aspartic- and metalloproteinases). It functions as an inhibitor of fibrinolysis by inhibiting plasmin and kallikrein. It functions as an inhibitor of coagulation by inhibiting thrombin.[15] Alpha-2-macroglobulin has in its structure a 35 amino acid "bait" region. Proteinases binding and cleaving the bait region become bound to α2M. The proteinase-α2M complex is recognised by macrophage receptors and cleared from the system. Fibrinolysis (simplified). Blue arrows denote stimulation, and red arrows inhibition. alpha-2-Macroglobulin is known to bind zinc, as well as copper in plasma, even more strongly than albumin, and such it is also known as transcuprein.[16] 10-15% of copper in human plasma is chelated by alpha-2-macroglobulin.[17] Disease alpha-2-Macroglobulin levels are increased when the serum albumin levels are low,[18] which is most commonly seen in nephrotic syndrome, a condition wherein the kidneys start to leak out some of the smaller blood proteins. Because of its size, alpha-2-macroglobulin is retained in the bloodstream. Increased production of all proteins means alpha-2-macroglobulin concentration increases. This increase has little adverse effect on the health, but is used as a diagnostic clue. Longstanding chronic kidney failure can lead to amyloid by alpha-2-macroglobulin (see main article: amyloid). A common variant (29.5%) (polymorphism) of alpha-2-macroglobulin leads to increased risk of Alzheimer's disease.[19][20] alpha-2-Macroglobulin binds to and removes the active forms of the gelatinase (MMP-2 and MMP-9) from the circulation via scavenger receptors on the phagocytes. References ^ a b c GRCh38: Ensembl release 89: ENSG00000175899 - Ensembl, May 2017 ^ a b c GRCm38: Ensembl release 89: ENSMUSG00000030111 - Ensembl, May 2017 ^ "Human PubMed Reference:". National Center for Biotechnology Information, U.S. National Library of Medicine. ^ "Mouse PubMed Reference:". National Center for Biotechnology Information, U.S. National Library of Medicine. ^ Andersen GR, Koch TJ, Dolmer K, Sottrup-Jensen L, Nyborg J (October 1995). "Low resolution X-ray structure of human methylamine-treated alpha 2-macroglobulin". J. Biol. Chem. 270 (42): 25133–41. doi:10.1074/jbc.270.42.25133. 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