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НОВЫЙ ДЕНЬ В МЕДИЦИНЕ
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MUDDATDAN OLDINGI TUG`RUQ HAVFI BŐLGAN HOMILADOR AYOLLARDA FETOPLASENTAR YETISHMOVCHILIKNI PROGNOZLASH VA OLDINI OLISH

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✓ ***Rezyume***

Fetoplasentar tizimning yetishmasligi bugungi kungacha zamonaviy akusherlikning dolzarb muammolaridan biri bo'lib qolmoqda. Fetoplasentar yetishmovchilik-bu homila va platsentadan turli xil ekstragenital va ginekologik patologiyalar, shuningdek, yuqumli virusli, bakterial kasalliklar, endokrin, ekzogen omillar ta'sirida platsenta shakllanishi va organogenez bosqichlarida yuzaga keladigan homiladorlikning asoratlari tufayli rivojlanadigan platsentaning morfofunktsional kasalliklari majmuasi. Fetoplasentar yetishmovchilikdan kelib chiqadigan asoratlarga homilaning intrauterin rivojlanishining kechikish sindromi, homilaning o'tkir va surunkali gipoksiyasi, platsentaning erta qarishi, rivojlanmagan homiladorlik kiradi. Plasentatsiya jarayonining buzilishi va platsentaning rivojlanmaganligiga olib keladigan erta FPY erta tug'ilishning sabablaridan biridir. Barcha tug'ilishlar orasida FPY darajasi 20-30% ga, erta tug'ilish holatlarida esa 23% ga yetadi. Shu sababli, hozirgi vaqtda zamonaviy akusherlikda klinikadan oldingi bosqichda fetoplasentar kompleksdagi patologik o'zgarishlarni aniqlashga imkon beradigan zamonaviy diagnostika usullarini qo'llash va klinik amaliyotga joriy etish ustuvor yo'nalish bo'lib qolmoqda.

Kalit so`zlar: Muddatdan oldin tug`ruq,fetoplasentar yetishmovchilik, plasentaning morfofunktsional kasalliklari,ekstragenital kasalliklar, ferroptoz, glutation peroksidaza 4, arginin.

ПРОГНОЗ И ПРОФИЛАКТИКА ФЕТОПЛАЦЕНТАРНОЙ НЕДОСТАТОЧНОСТИ У БЕРЕМЕННЫХ С ПОВЫШЕННЫМ РИСКОМ ПРЕЖДЕВРЕМЕННЫХ РОДОВ

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✓ ***Резюме***

Недостаточность фетоплацентарной системы и по сей день остается одной из актуальных проблем современного акушерства. Фетоплацентарная недостаточность-комплекс морфофункциональных заболеваний плаценты, развивающихся вследствие различных экстрагенитальных и гинекологических патологий со стороны плода и плаценты, а также осложнений беременности, возникающих на этапах формирования плаценты и органогенеза под влиянием инфекционных вирусных, бактериальных заболеваний, эндокринных, экзогенных факторов. Осложнения фетоплацентарной недостаточности включают синдром задержки внутриутробного развития плода, острую и хроническую гипоксию плода, преждевременное старение плаценты, неразвитую беременность. Нарушение процесса плацентации и преждевременная FPY, приводящая к недоразвитию плаценты, являются одними из причин преждевременных родов. Среди всех родов FPY достигает 20-30%, а в случае преждевременных родов-23%. Поэтому в настоящее время в современном акушерстве приоритетным направлением остается применение и внедрение в клиническую практику современных методов диагностики, позволяющих выявить патологические изменения в фетоплацентарном комплексе на доклинической стадии.

Ключевые слова: преждевременные роды,фетоплацентарная недостаточность, морфофункциональные заболевания плаценты, экстрагенитальные нарушения, ферроптоз, глутатионпероксидаза 4, аргинин.

FORECASTING AND PREVENTION OF FETOPLASENTAR DEFICIENCY IN PREGNANT WOMEN WITH PRETERM OBSTETRIC CARE

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✓ ***Resume***

The lack of a Fetoplasentary system remains one of the pressing problems of modern obstetrics to this day. Fetoplasentar insufficiency is a complex of morphofunctional diseases of the placenta, which develop from the fetus and placenta due to various extragenital and gynecological pathologies, as well as complications of pregnancy caused by infectious viral, bacterial diseases, endocrine, exogenous factors at the stage of placenta formation and Organogenesis. Complications from Fetoplasentar insufficiency include delayed fetal intrauterine development syndrome, acute and chronic fetal hypoxia, premature placental aging, underdeveloped pregnancy. Premature FPY, which leads to impaired placentation and underdevelopment of the placenta, is one of the causes of premature birth. Among all births, the FPY rate reaches 20-30%, and in cases of premature birth it reaches 23%. Therefore, at present, in modern obstetrics, the application and introduction into clinical practice of modern diagnostic methods, which allow to identify pathological changes in the fetoplasentar complex at the pre-clinical stage, remains a priority.

Keywords: premature labor,fetoplasentar insufficiency, morphofunctional diseases of the placenta,extragenital diseases, ferroptosis, glutathione peroxidase 4, arginine.

Dolzarbligini

Muddatdan oldingi tug`ruq homiladorlikning 22-haftasidan 37-haftasiga qadar bo`lgan gestatsion davrda homiladorlikning muddatidan oldin to`xtashiga aytiladi. Homilaning gestatsiya muddatini inobatga olib erta tug`ruqni 3ta davrga bo`lamiz 1) 22-27-haftasiga qadar, 2) 28-33-haftasiga qadar, 3) 34-37-haftasiga qadar sodir bo`lgan tug`ruq jarayonlarini inobatga olib, quyidagi sabablar bu jarayonning xatar omili xisoblanadi: ijtimoiy-biologik tashqi ekstrakorporal omillar (alimentar ovqatlanishning qoloqligi, kofeinga boy ichimliklarni haddan ziyod istemol qilish, narkotik xususiyatga ega bo`lgan vositalarni ichish va chekish, ruhiy stress jarayonlar, travmalar, ish faoliyati bilan bog`liq radioaktiv nurlanishlar). Akusherlik-ginekologik anamnez ma`lumotlari (onaning juda yosh ekanligi bunda onalikka jismonan tayyor emasligi yoki yoshi katta tug`uvchi onalar,anamnezidan erta tug`ruqlar kuzatilganligi,ko`p bor tabiy abort kechganligi, ko`p xomilalik,og`irlashgan akusherlik anamnezlari ), ekstragenital kasalliklar (qandli diabet, pielonefrit, miopiya, periferik qon tomirlar varikozi, semizlik, bronxial astma, pnevmaniya, urogenital kasalliklar, virusli kasalliklar) ,homiladorlik asoratlari (homiladorlar gipertenziyasi,eklampsiya preeklampsiya, yo`ldoshni oldinda joylashuvi,yo`ldoshning erta ko`chishi, fetoplasentar yetishmovchilik,qog`onoq suvining ko`pligi yoki haddan ziyod kamligi, homilani chanoq bilan kelishi,bachadon bo'yni yetishmovchiligi, bachadon malformatsiyasi) .Fetoplasentar yetishmovchilik-bu homila va platsentadan turli xil ekstragenital va ginekologik patologiyalar, shuningdek homiladorlikning asoratlari tufayli rivojlanadigan plasentaning morfofunktsional kasalliklar majmuasi bo`lib, birlamchi yetishmovchilik homiladorlikning dastlabki bosqichlarida (16-18 xafta), platsenta shakllanishi va yuqumli virusli,bakterial kasalliklar, endokrin, yatrogen omillar ta'sirida organogenez bosqichlarida paydo bo'ladi. Ikkilamchi fetoplasentar yetishmovchilik dastlab normal shakllangan platsenta bilan, onalik omillari yoki homiladorlikning asoratlari ta'siri ostida rivojlanadi. [1]. Perinatal kasallanish va homilaning perinatal o'limi fetoplasentar yetishmovchilik tufayli yuzaga keladigan asoratlarga homilaning intrauterin rivojlanishini kechikish sindromi, homilaning o'tkir va surunkali gipoksiyasi, platsentaning erta qarishi, rivojlanmayqolgan homiladorlik kiradi. Platsentatsiya jarayonining buzilishi bilan yuzaga keladigan va platsentaning rivojlanmay qolishiga olib keladigan erta FPY, erta tug'ilishning sabablaridan biridir. Shu sababli, hozirgi vaqtda kasallikning dastlabki, klinikadan oldingi bosqichida fetoplasentar kompleksdagi patologik o'zgarishlarni aniqlashga imkon beradigan zamonaviy diagnostika usullarini ishlab chiqish va klinik amaliyotga joriy etish dolzarbdir. Ona-platsentaplod tizimida gomeostazni ta'minlaydigan

himoya-adaptiv mexanizmlarning neyrogen regulyatsiyasining asosiy kontseptsiyasi shubhasiz qiziqish uyg'otadi. Gormonal holatning o'zgarishi bilan bog'liq homilador ayollar tanasidagi fiziologik jarayonlar yallig'lanish va allergik reaktsiyalarni rivojlanishida ishtirok etadigan ba'zi vositachilarning yani gistamminning proliferatsiyasi, il-3, il-4 interleykinlarni semiz hujayralaridan sitokinlar oqimi kuchayishi sodir bo'ladi. Sitokinlar, o'z navbatida, eozinofillar, t-limfotsitlar va bazofillarning burun shilliq qavatiga infiltratsiyasini osonlashtiradi. Infiltratsiya qiluvchi yallig'lanish hujayralaridan, ayniqsa eozinofillardan ajralib chiqadigan leykotrienlar asosiy rol o'ynaydi. Lekin bu hammasi emas. Allergiya reaktsiyasi, shuningdek, immunoglobulin E - IgE (antitanachalar) va agronulositlar ko'payishiga olib keladi, bu esa o'z navbatida turli organlar va tizimlarda (o'pka, teri, ko'z va burun) alomatlari bilan yallig'lanishni keltirib chiqarishi mumkin. Gistamin vazoaktiv, differentsiatsiya va o'sishni rag'batlantiruvchi xususiyatlari tufayli embrion–bachadon o'zaro ta'siriga hissa qo'shadi deb taxmin qilingan va homiladorlikdagi qonda gistamin darajasining ko'tarilishi turli xil salbiy natijalarga olib kelishi ma'lum, masalan, homila tushish havfi, erta tug'ilish xavfi va preeklampsiya. Ma'lumki, homiladorlik davrida platsenta ko'p miqdorda diamin oksidaza (gistaminaza) ishlab chiqarishga qodir, buning natijasida bu davrda sarum gistamin miqdori kamayadi, bu esa homiladorlik davrida allergik holatlarning kamroq namoyon bo'lishiga yordam beradi. Ammo Gistamin kindik arteriyalarida vazokonstriksiyada vositachilik qiladi va inson homiladorligidagi tomirlar va preeklampsiyaning og'irligi gistaminning qondagi darajasini oshirish orqali ortadi. Disfunktsional platsentadan yallig'lanishga qarshi moddalar kontsentratsiyasining oshishi tufayli endotelial disfunktsiyaga olib keladigan tizimli yallig'lanishning kuchayishi yana bir muhim yo'ldir. Shuningdek, platsentadan sFlt-1 (soluble Fms-Like Terosine Kinase-1,eriydigan FMS-tirozin kinaz-1) komplement tizimining faollashishi bilan bog'liq, (SFlt-1 funktsiyasi uning patofiziologiyasida asosiy o'rinni antiangiogen oqsil, eriydigan FMSga o'xshash tirozin kinaz-1 (sFlt-1) egallaydi. sFlt - 1 bir qator to'qimalardan qon oqimiga chiqariladi, u erda qon tomir endotelial o'sish omili va platsenta o'sish omili faolligiga qarshi turadi, bu esa endotelial disfunktsiyaga olib keladi) bu semiz hujayralarning gistaminni chiqarishiga olib kelishi mumkin.Platsentada gistamin ishlab chiqaruvchi gistidin dekarboksilaza fermentining yuqori ifodasi, homila-ona chegarasidagi gistamin retseptorlari va EHRF mavjudligi homiladorlik paytida gistaminning fiziologik rolini ko'rsatadi. [13-17]. Gistamin va Diamin oksidaza o'rtasidagi muvozanat asoratlanmagan homiladorlik uchun juda muhim ko'rinadi. Diamin oksidaza faolligining pasayishi homiladorlikning bir nechta geterogen asoratlari, masalan, diabet, xomila tushish xavfi va rivojlanmagan homiladorlik va trofoblastikaning buzilishga olip keladi. Ba'zi ma'lumotlarga ko'ra, platsenta yetishmovchiligining kompensatsiyalangan shakli bilan homila rivojlanishining kechikish sindromi homilador ayollarning taxminan 70 foizida aniqlanadi. Agar platsenta yetishmovchiligiga olib keladigan asoratlarni davolash mumkin bo'lmasa yoki umuman amalga oshirilmasa, platsentaning unda yuzaga keladigan buzilishlarni qoplash qobiliyati susayishni boshlaydi va subkomnensatsiyalangan platsenta yetishmovchiligi rivojlanadi. Himoya mexanizmlarining charchoq bosqichi boshlanadi, bu har xil og'irlikdagi homila rivojlanishining kechikishiga olib keladi. So`ngi yangiliklardan ferroptozning ham fetoplasentar yetishmovchilikga aloqadorligi aniqlangan. Ferroptoz-bu ko'p to'yinmagan yog'li kislotalarni o'z ichiga olgan fosfolipidlarning oksidlanish-faol temirga bog'liq peroksidlanishi va lipid peroksidatsiyasidan keyin tiklanish qobiliyatining yo'qolishi bilan aniq belgilanadigan dasturlashtirilgan hujayra o'limining yangi aniqlangan shakli. Ferroptoz atamasi 2012 yilda fanga kiritilgan bo`lib, sistin importini inhibe qiluvchi past molekulyar og'irlikdagi erastin tufayli hujayra o'limining shaklini tavsiflash uchun glutationning kamayishi va fosfolipid peroksidaza glutation peroksidaza 4 (GPX4) ning inaktivatsiyasiga olib keladi. GPX4 potentsial toksik lipid gidroperoksidlarini (LOOH) toksik bo'lmaganlarga aylantiradi [24].

Ko'pgina tadqiqotchilar ferroptozga uchragan hujayralarda nekrozga o'xshash morfologik o'zgarishlarni topgan, degan fikrga qo'shiladilar [35]. Ushbu xususiyatlarga plazma membranasining yaxlitligini buzish, sitoplazmaning shishishi (onkoz) va sitoplazmatik organellalar, xromatinning o'rtacha kondensatsiyasi kiradi. Ultrastrukturaviy darajada, mitoxondriyal anomaliyalar odatda ferroptoz hujayralarida uchraydi, masalan, kondensatsiya yoki shishish, membrana zichligining oshishi, kristallarning kamayishi yoki yo'qligi va tashqi membrananing yorilishi Temir va lipid peroksidlari ferroptozning ikkita asosiy ishtirokchisidir [24, 29, 31]. Lipid peroksidlarining to'planishi, asosan fosfatidiletanolamin-OOH (Fe-OOH) oxir-oqibat ferroptozga olib keladi [30], temir esa ferroptozning asosiy regulyatorining katalizatori yoki komponenti bo'lib xizmat qiladi [38]. Shunday qilib, temir xelatatorlari (masalan, deferoksamin) va ba'zi lipofil antioksidantlar (masalan, a-tokoferol) ferroptozning oldini oladi [39]. Bundan tashqari, fentonning temir bilan katalizlangan reaktsiyasi natijasida hosil bo'lgan ros ferroptozning boshlanishiga yordam beradi. GPX4 kofaktori ishtirokida toksik yog ' kislotasi peroksidlarini toksik bo'lmagan yuqori spirtlarga qaytaradi [48, 40]. GPX4 hujayra ichidagi yog ' peroksidlarini yo'q qilish orqali hujayralarni ferroptozdan himoya qiladi. GPX4 qon zardobida kamayishi ferroptozni qo'zg'atadi [25, 31, 41]. 8 ta nukleofil aminokislotalarni o'z ichiga olgan GPX4

hujayradagi elektrofillar bilan reaksiyaga kirishishi mumkin [31]. Selen GPX4 ning ferroptozga chidamli faolligini saqlab turishi uchun kerak va selenotsisteinni sistein bilan almashtirish hujayralarni ferroptozga sezgir qiladi [31, 43]. GPX4 ning inaktivatsiyasi yoki yo'qligi lipid peroksidlarining to'planishiga olib keladi, bu ferroptotik hujayra o'limining signali sifatida qaraladi. Shunday qilib, GPX4 miqdorining kamayishi ferroptozning muhim bosqichidir. Lipidlar hujayra ichidagi membrana tizimining energiya ta'minoti va tarkibida hal qiluvchi rol o'ynaydi. Fosfolipidlarning (FL) kislorodlanishi (masalan, fosfatidilxolin, kardiolipin) hujayralardagi ferroptozga yordam beradi [30, 31, 43]. Ma'lumki, lipid peroksidlari hujayralarda uchta asosiy yo'l orqali hosil bo'ladi, ularning har birini amalga oshirish uchun temir kerak:

1) fentonning fermentativ bo'lmagan reaktsiyasi natijasida hosil bo'lgan lipid ros. Temir (II) ionlari vodorod peroksid bilan temir (III)ionlariga oksidlanadi: $Fe2{+}{+}H2O2 \rightarrow Fe3{+}{+}OH{+}OH{-}$;

2) ko'p to'yinmagan yog'li kislotalarning (pufa) kislorodlanishi va esterifikatsiyasi natijasida hosil bo'lgan lipid peroksidlari [30, 31].

3) lipidlarning avtooksidatsiyasi paytida temir bilan katalizlangan usulda hosil bo'lgan lipid peroksidlari [45].

Fenton reaktsiyasi noorganik kimyoviy reaktsiya bo'lib, tabiatda keng tarqalgan. Yaqinda lipotoksik hujayra o'limining ushbu o'ziga xos shakli insonning ko'plab kasalliklari, jumladan, yurakning ishemik-reperfuzion shikastlanishi, miya shikastlanishi, buyrakning o'tkir shikastlanishi, saraton va astma bilan bog'liqligi aniqlandi. Qizig'i shundaki, ferroptoz bilan bog'liq bo'lgan parametrlar platsenta fiziologiyasi va trofoblastning shikastlanishi bilan bog'liq. Bunday holatlarga platsentaning rivojlanishi paytida gipoksiya-reperfuziya, bachadonning fiziologik qisqarishi yoki platsenta to'shagining patologik o'zgarishi, trofoblastdagi temirning ko'pligi, preeklampsi, homila o'sishining kechikishi va erta tug'ilish kabi asosiy platsenta kasalliklarining patofiziologiyasida lipotoksiklik belgilari, shuningdek glutationning peroksidlanish qobiliyatining pasayishi va lipid peroksidatsiyasining pasayishi kiradi. Platsenta trofoblastlarini ferroptoz shikastlanishidan himoya qilishda glutation peroksidaza-4 rol o`ynaydi. Bizning matnimiz platsenta trofoblastlarini lipotoksik shikastlanishdan himoya qilish vositasi sifatida ferroptozni tartibga solishning yangi imkoniyatlari ustida izlanish olib borilmoqda . Tartibga solinadigan hujayra o'limi ko'plab fiziologik va patologik jarayonlarning asosiy tarkibiy qismidir. Nekrotik hujayra o'limi hujayra membranasi va organellalariga zarar yetkazadigan stressga javoban sodir bo'lishi mumkin.Shuning uchun ferroptozni o'rganish kasallikning patogenezini aniqroq aniqlash va yangi terapevtik vositalar va kasalliklarning oldini olish choralarini ishlab chiqish uchun tadqiqotning asosiy yo'nalishiga aylandi [18]. Bunday holatni aniqlash qon zardobidagi GPX4 miqdorini aniqlash orqali amalga oshirilib, Ferroptozni oldini olish mumkun. Homiladorlikning turli davrlarida FPY uchun turli xil davolash sxemalari mavjudligiga qaramay, ushbu patologiyani davolash va oldini olishning yanada samarali usullarini izlash davom etmoqda. Arterial gipotenziya homiladorlikning birinchi uch oyligida ko`p kuzatiladi, anemiya holati bu holatni asoratlanishiga sabab bo`ladi: Erta toksikoz (6,1% dan 38,4% gacha), gestoz (ichida) 18,9% - 34,7%, muddatidan oldin tug'ish (6,2% - 20,1%). homiladorlikning keng tarqalgan asoratlaridan biri bu fetoplasental yetishmovchilikning erta boshlanishiga ham olip keladi [4]. Homilador ayollarda qonning kislorod bilan to'yinganligi kamayadi. Ma'lumki, arterial gipotenziya intrauterin gipoksiya paydo bo'lishida muhim omil hisoblanadi. Uteroplasentar qon oqimining pasayishi tufayli homila rivojlanishi (35% gacha) kechikishiga sabab bo`ladi. Miyometriy tonusining vaqti-vaqti bilan yoki uzoq vaqt davomida oshishi venoz oqimning pasayishi tufayli fetoplasentar bo'shliqda qon aylanishining buzilishiga olib kelishi ma'lum. Homiladorlikning erta muddatlarida qo`llash mumkun bo`lgan dorilardan Tivortin preparati ham zararsiz hisoblanadi. Tivortin dori vositasi sitoprotektorlik, antioksidantlik, antigipoksik, membranani stabillashtiradigan, detoksifikatsion xususiyatiga ega bo`lib, Arginin (a-amino-δ-guanidinovalerian kislotasi) aminokislotalar sinfiga kiruvchi va organizmning ko'plab hayotiy funktsiyalarining faol va ko'p qirrali hujayra regulyatori ta'siriga ega bo`lib, tanadagi gormonal muvozanatni saqlashda ma'lum rol o'ynaydi. Arginin qondagi, prolin, poliamin, agmatin sintezida ishtirok etadi, fibrinogenoliz jarayonlariga ta`sir qiladi va membranani depolarizatsiya qiluvchi ta'sirga ega. Arginin NO-sintaza fermenti uchun substrat bo'lib, endoteliotsitlarda azot oksidi sintezini katalizlovchi fermentdir. Preparat guanilat siklazani faollashtiradi va qon tomir endoteliyasida SGMF darajasini oshiradi, qon tomir endoteliyasiga leykotsitlar va trombotsitlarning adgeziya va agregatsiyasini kamaytiradi .VCAM-1 va MCP-1 yopishqoq oqsil sintezini inhibe qiladi, shu bilan aterosklerotik blyashka hosil bo'lishi va rivojlanishining oldini oladi, qon tomir devorining silliq mushak hujayralarining vazokonstriktorlik va stimulyatori bo'lgan endotelin-1 sintezini inhibe qiladi. Bu esa bachadon, qon tomir gipertonusini kamayishiga yordam beradi . Fetoplasentar qon aylanishni yaxshilaydi aterosklerotik belyashkalar hosil bo`lishini oldini oladi. Homiladorlarga ineksiya qilgan vaqtdan 30-40 minut o`tgach homilaning yurak urishlari va onaning yurak urishlar sonining normallashganini ko`rishimiz mumkun. Davo kursi kamida 5 kunni tashkil qiladi. Oral 200 ml lik flakon,

15ml dan 3 mahal 10-15 kun ichishga, yoki tezroq ta`sir etishi uchun vena ichiga 100ml kuniga 1mahal 5 -7 kun ineksiya qilish orqali amalga oshiriladi.

Xulosa

Zamonaviy adabiyotlarni tahlil qilish shuni ko'rsatadiki, ko'pincha fetoplasental yetishmovchilik uning sababidan alohida ko'rib chiqiladi. Gipovolemiya, tromboz va qon tomir qarshiligining oshishi tufayli fetoplasentar kompleksning tomirlarida qon aylanishining buzilishi qayd etiladi va yetkazib berishning yetarli emasligi to'g'risida xulosa chiqariladi.

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