

Aplastik anemiyasi bilan homiladorda kasallikning turli bosqichlarida boshqarish tajribasi

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Annottatsiya: Aplastik anemiya - gematopoetik ildiz hujayralarining buzilishi bo'lib, uning natijasida gematopoetik o'zak xujayralarining patologiyasi bo'lib, unda suyak iligi gipoplaziyasi yoki aplaziyasi, 2 yoki undan ortiq hujayra avlodlari (eritrotsitlar, leykotsitlar va trombositlar) sitopeniyalari rivojlanadi. Klinik simptomlar anemiya, trombositopeniya (petexiya, qon ketish) yoki leykopeniya (infeksiya) rivojlanishidan kelib chiqadi. Tashxis suyak iligi biopsiyasi paytida periferik qonda pansitopeniya va suyak iligining gipotsellyulyarini aniqlashga asoslangan. Davolash odatda antitimotsitar globulini va siklosporin yoki suyak iligi transplantatsiyasi bilan immunosupressiyani o'z ichiga oladi.

Kalit so'zlar: aplastik anemiya, homiladorlik, genetik mutatsiyalar, gemotransfuziya, ildiz hujayralari

Experience in managing a pregnant woman with aplastic anemia at different stages of the disease

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Abstract: Aplastic anemia is a hematopoietic stem cell disorder, resulting in a pathology of hematopoietic stem cells, in which bone marrow hypoplasia or aplasia, cytopenias of 2 or more cell lineages (erythrocytes, leukocytes and platelets) develop. Clinical symptoms are due to the development of anemia, thrombocytopenia (petechiae, bleeding) or leukopenia (infection). Diagnosis is based on the detection of pancytopenia and bone marrow hypocellularity in peripheral blood during bone marrow biopsy. Treatment usually includes immunosuppression with antithymocyte globulin and cyclosporine or bone marrow transplantation.

Keywords: aplastic anemia, pregnancy, genetic mutations, blood transfusion, stem cells

Kirish. Aplastik anemiya (AA) qon tizimining kam uchraydigan kasalligi bo'lib, u gematopoetik xujayralarining kamayishi bilan og'ir suyak iligi yetishmovchiligi ildiz hujayralari, suyak iligi aplaziyasining rivojlanishi va pansitopeniya paydo bo'lishi bilan tavsiflanadi. AA bilan homiladorlik onaga ham, homilaga ham tahdid soladigan og'ir asoratlarning rivojlanishi bilan birga kelishi mumkin. AA bilan homilador ayollarni davolash gematologlar va tegishli shifokorlar uchun qiyin vazifadir.

Aplastik anemiya etiologiyasi

Kimyoviy moddalar (masalan, benzol, noorganik mishyak)

Dorilar (masalan, o'smaga qarshi dorilar, antibiotiklar, steroid bo'lmagan yallig'lanishga qarshi dorilar, antikonvulsanlar, atsetazolamid, oltin tuzlari, penitsilamin, xinakrin) yoki toksinlar

Gepatit (gepatit viruslari uchun seronegativ)

Homiladorlik

Radiatsiya ta'siri

Viruslar (Epshteyn-Barr virusi va sitomegalovirus)

Genetik mutatsiyalar natijasida kelib chiqqan suyak iligi yetishmovchiligining irsiy kasalliklari (masalan, Fankoni anemiyasi, Shvaxman-Daymonda sindromi, diskeratoz)

Aniq mexanizm noaniq bo'lib qolmoqda, ammo orttirilgan holatlarning aksariyati gematopoetik ildiz hujayralariga immunitet hujumini o'z ichiga oladi. Klonal gematopoez tez-tez kuzatiladi va miyeloid malignizatsiyaga o'tish xavfi mavjud.

Aplastik anemiyaning belgilari

Aplastik anemiya odatda sekin bilan boshlanadi, ko'pincha virus bilan kasallanganidan, dori vositalarining ta'siridan yoki zaharli moddalar (masalan, insektitsidlar, benzol) ta'siridan bir necha hafta yoki oy o'tgach, ba'zida o'tkir bo'lishi mumkin.

Aplastik anemiyada kamqonlik zaiflik va charchoqqa olib kelishi mumkin, og'ir trombotsitopeniya esa petexiyalar, ekimozlar, tish milkidan, konyunktiva va boshqa to'qimalardan qon ketishiga olib kelishi mumkin. Agranulotsitoz odatda hayot uchun xavfli infeksiyalarga olib keladi.

Aplastik anemiya diagnostikasi

To'liq qon tahlili va retikulotsitlar soni

Sitogenetik va molekulyar tahlil yordamida suyak iligi tekshiruvi

Paroksizmal tungi gemoglobinuriyada (PTG) klonni (hajmini) aniqlash uchun oqim sitometriyasi

PTG klonini aniqlash irsiy sindromni istisno qilishga imkon beradi (1).

Aplastik anemiya pansitopeniya bilan ogʻrigan bemorlarda, ayniqsa yoshlarda koʻproq rivojlanishi mumkinligi aniqlanadi. Ogʻir aplastik anemiya suyak iligi hujayraliligi $< 25\%$ va quyidagilardan ≥ 2 tasining mavjudligi bilan tavsiflanadi:

Mutlaq neytrofillar soni $< 500/\mu\text{L}$ ($< 0,5 \times 10^9/\text{L}$)

Mutlaq retikulotsitlar soni $< 60\,000/\mu\text{L}$ ($< 60 \times 10^9/\text{L}$)

Trombotsitlar soni $< 20\,000/\mu\text{L}$ ($< 20 \times 10^9/\text{L}$)

Juda ogʻir aplastik anemiya mutlaq neytrofillar soni $< 200/\mu\text{L}$ ($< 0,2 \times 10^9/\text{L}$) sifatida aniqlanadi.di.

Ishning maqsadi. Homiladorlik davrida AA bilan kasallangan bemorlarni kasallikning turli bosqichlarida (boshlanish, remissiya, refrakterlik) davolash xususiyatlarini tahlil qilish.

Materiallar va usullar. 2012 yildan 2023 yilgacha boʻlgan davrda Samarqand tibbiyot markazining gematologiya va ginekologiya boʻlimlarida AA bilan 16 homilador ayol kuzatildi. AA tashxisining oʻrtacha yoshi 25 yil (18-33 yosh). Ogʻir boʻlmagan AA (NAA) boʻlgan 11 bemor va ogʻir AA bilan 11 bemor bor edi.

Natijalar va muhokama. 6/16 bemorlarda AA tashxisi birinchi marta homiladorlik davrida qoʻyildi. Oʻrtacha homiladorlik muddati 21 hafta (8-35 hafta). Ulardan ikkita bemorga 21-22-haftadan boshlab tugʻilgunga qadar kuniga 5 mg / kg dozada CSA terapiyasi buyurildi, buning fonida transfuziyaga bogʻliqlikning pasayishi qayd etildi. Uchta (32 haftadan ortiq) zudlik bilan favqulodda yetkazib berishdan oʻtdi va keyin ATG kursini oldi (1-3-jadvallarda). 7/16 hollarda homiladorlik ATG va CSA terapiyasidan soʻng toʻliq remissiya fonida sodir boʻldi (oʻrtacha remissiya 7 yil). Ulardan 2 nafar bemorda 9 oylik remissiya davri kuzatilgan. relaps rivojlandi (CSA terapiyasi fonida) va remissiya davri 13 yil boʻlgan 1 bemorda tugʻruqdan keyin qon miqdorini tiklash bilan oʻrtacha sitopeniya rivojlandi. 2/16 hollarda homiladorlik refrakter AA holatida sodir boʻldi va homiladorlikni boshqarish qon tarkibiy qismlarini transfuziyasi yordamida amalga oshirildi. U1/16 - AA-PTG sindromi rivojlanishida (ekulizumab terapiyasi oʻtkazildi). Natijada, 15/16 sogʻlom bolalarni tugʻdi. Bir abort erta bosqichda (12 hafta) CSA terapiyasi fonida AA ning qaytalanishi rivojlanishi tufayli amalga oshirildi. Bir bemor ISTga chidamli boʻlgan TAA kursi fonida ogʻir yuqumli asoratlarni rivojlanishi natijasida vafot etdi. Tugʻish asosan kesar kesish bilan amalga oshirildi. Tabiiy yoʻl bilan tugʻish terapiyadan chiqarilgan toʻliq remissiyadagi ayollarga ruxsat berildi.

Xulosa. AA aniqlangan tashxisi bilan homiladorlikni rejalashtirish uzoq muddatli toʻliq remissiya va ISTni toʻxtatish holatida zarur. Bunday holda, retsidiv rivojlanishining oldini olish mumkin.

Bizning bemorlar guruhida, AAning toʻliq uzoq muddatli remissiyasidagi ayollar orasida homiladorlik asoratsiz davom etdi va qisqa muddatli remissiya davri

bo'lgan bemorlarda remissiyaga erishildi. 21-22 haftadan so'ng CSA terapiyasi tug'ilishgacha qon quyish ehtiyojini kamaytirishi mumkin. Har biri xomilador uchun individual yondashuv talab etiladi

AA bo'lgan homilador bemor, gemotransfziyaning optimal ta'minotini ta'minlash, shuningdek, har bir holatda klinik xususiyatlarni (shu jumladan akusherlik, ginekologik) hisobga olish uchun shifokorlar guruhining muvofiqlashtirilgan ishini talab qiladi.

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