

<p>PROFEX SZAKNYELVI VIZSGA</p> <p>Középfok – angol nyelv Szóbeli Beszédértés</p>		<p>vizsgáló sorszáma:</p> <p>◀ ◀ ◀ ◻</p> <p>dátum: 2007. április 14.</p>
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Oldja meg a két feladatlapot a hallott szöveg alapján! Elérhető pontszám: 20 pont
 Figyelem! A vizsga akkor lehet sikeres, ha a vizsgáló részegységenként legalább 40%-ot teljesít.
 Végző megoldásként csak a tintával írt változatot fogadjuk el.
 Kérjük, hogy jól gondolja meg a választát, mivel bármilyen válaszmódosítás esetén válasza érvénytelen.

MEGOLDÓKULCS

1. Szöveg – Feladatlap

I. Töltse ki az üresen hagyott helyeket, mindegyiknél csak 1 angol szót használjon. Az elsőt példaként megoldottuk. (4 pont)

Primary biliary cirrhosis (PBC) is a liver disease characterised by **inflammation**, formation of scar tissue and **obstruction** of the bile ducts. The cause is still not **clear**, however, it is probable that the body's own tissues are **attacked** by the person's immune system. Bile flow from the liver is blocked by the inflammation in the bile ducts, consequently bile **remains** in the cells of the liver, or passes into the bloodstream.

II. Töltse ki az alábbi táblázat üresen hagyott helyeit. Az egyes helyekre 1 angol szót írjon. (4 pont)

first symptoms of PBC	(1) <u>itchiness</u> fatigue
other features of PBC	enlargement of finger tips bone/ nerve/ kidney abnormalities
signs and symptoms detected on physical examination	enlarged firm liver /spleen (2) small yellow <u>deposits</u> in skin or eyelids (3) increased skin <u>pigmentation</u> (4) <u>jaundice</u>

III. Karikázza be azon egyetlen állítás betűjelét, amely az Ön által hallottaknak leginkább megfelel. (2 pont)

In c. half of the cases, when the patient is asymptomatic, PBC is discovered during

- A routine blood testing.**
- B a liver biopsy.
- C liver function tests.
- D an ultra sound together with an MRI.

In the treatment of PBC

- A cholestyramine is usually combined with ursodeoxylic acid.
- B Ca and vitamin supplements are needed in a fat-soluble form.
- C drug-intolerance usually develops against ursodeoxylic acid.
- D transplantation is needed if the liver is severely damaged.**

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1. Szöveg

Primary biliary cirrhosis (PBC)

Primary biliary cirrhosis is inflammation and eventual scarring and obstruction of the bile ducts inside the liver.

Primary biliary cirrhosis is most common among women aged 35 to 60, though it can occur in men and women of any age. The cause, though not clear, is probably autoimmune - a case in which the person's immune system attacks the body's own tissues. *Primary biliary cirrhosis* occurs in association with rheumatoid arthritis, scleroderma, or autoimmune thyroiditis.

Primary biliary cirrhosis begins with inflammation of the bile ducts inside the liver. The inflammation blocks the flow of bile out of the liver, thus, bile remains in the liver cells or spills over into the bloodstream. As inflammation spreads from the bile ducts to the rest of the liver, scar tissues develop in the liver.

Usually, *primary biliary cirrhosis* starts gradually. Itchiness and sometimes fatigue are often the first symptoms. Other features, which may not occur until months or years later, include enlargement of the tips of the fingers and abnormalities of the bone, nerves, and kidneys. Later, any of the symptoms and complications of cirrhosis can form. Metabolic bone disease occurs in most people.

On physical examination, a doctor feels an enlarged, firm liver in about half of people and an enlarged spleen in about 25%. In the late stages, the scarred liver shrinks in size. Almost 15% of people have small yellow deposits in the skin or eyelids. About 10% have an increased skin pigmentation. Fewer than 10% have only jaundice early on; jaundice tends to develop in others later.

In nearly 50% of people with *biliary cirrhosis*, the disorder is discovered before symptoms even appear because of abnormalities detected during routine blood testing. Antibodies against mitochondria are found in the blood of more than 90% of people with the disorder.

When jaundice is evident or results of liver function tests indicate abnormalities, an ultrasound scan, or occasionally an MRI of the bile duct can be used to show any abnormalities or obstruction of the bile ducts outside the liver. The diagnosis of *primary biliary cirrhosis* is supported by finding that the bile ducts outside the liver are unobstructed, thus identifying the liver as the site of the problem. A liver biopsy confirms the diagnosis and stages the disease.

The progression of *primary biliary cirrhosis* varies greatly. People who initially have no symptoms often develop symptoms after 2 to 7 years. Others have no symptoms for 10 to 15 years. Still others become very ill in 3 to 5 years. The disorder culminates in severe cirrhosis.

At this time, no cure is known. The drug cholestyramine may control itchiness. Supplements of calcium and vitamins A, D, and K in a water-soluble form may be needed. The drug ursodeoxycholic acid appears to slow the disease's progression and is well-tolerated. Liver transplantation is the best treatment for people entering the final stages of the disorder.

Source: www.merck.com

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MEGOLDÓKULCS
2. Szöveg - Feladatlap

I. Töltse ki az üresen hagyott helyeket, mindegyiknél csak 1 angol szót használjon. Az elsőt példaként megoldottuk. (3 pont)

Dr Neil MacIntosh is a **paediatrician** investigating rickets in prematurity. Rickets is a defect in the bone **mineralisation** of premature infants. Dr MacIntosh is dealing with babies of less than a kilo in weight and less than 28 weeks' **gestation** . The period of investigation lasts for 5 to 16 weeks of **postnatal** age.

II. Töltse ki az alábbi táblázat üresen hagyott helyeit. Az egyes helyekre legfeljebb 2 angol szót írjon. (7 pont)

population of study	(1) <u>premature babies</u>
effects of rickets:	(2) <u>demineralisation</u> of bones (3) <u>(spontaneous) fractures</u> in bones (4) rachitic respiratory <u>distress</u>
past treatment:	(5) extra <u>vitamin D</u>
dosage during past treatment:	(6) <u>2000</u> units of vitamin D a day
laboratory experiment carried out:	(7) measurement of <u>metabolites</u> in blood
possible cause of disease:	(8) defect in <u>mineral</u> supply

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2. Szöveg

Rickets is usually caused by lack of vitamin D. So it was with considerable surprise that paediatrician Dr Neil MacIntosh of St George's Hospital in London found that premature babies were suffering from rickets of prematurity despite being dosed with adequate vitamin D. We asked him about this form of rickets.

Dr MacIntosh: It's a defect in bone mineralisation that we see in these very premature infants that we're dealing with these days.

Reporter: Now you're talking about these very premature babies, but how old are they and how heavy would they be?

Dr MacIntosh: These are the babies that are born less than a kilo in birthweight and usually less than 28 weeks' gestation, and we begin to see it from about 5 weeks of postnatal age going up to probably 16 weeks of postnatal age.

Reporter: And what sort of effects does the condition have on babies?

Dr MacIntosh: Well there's a fairly spectacular demineralisation of the bones and when this is severe you find spontaneous fractures in the long bones, sometimes in the ribs, and there's also a condition known as rachitic respiratory distress where it's felt that the ribs are so demineralised that they are rather rubbery so that rather than the thorax expanding during breathing each inspiration leads to the chest caving in on itself.

Reporter: Now how would this condition have been treated in the past?

Dr MacIntosh: Rickets in the past has always been viewed as a defect of vitamin D metabolism or supply, so it would have been treated with extra vitamin D.

Reporter: And in fact this is the way you've been treating it until quite recently.

Dr MacIntosh: Yes. About 4 years ago Dr Oliver Brook and myself started treating babies in our unit with 2000 units of vitamin D a day, which is more than, I think, any other unit in the country but we've still been seeing the rickets, and we've become aware during this time that there's no problem therefore of supply of vitamin D. But we've also measured the vitamin D metabolites in the blood, both 25-hydroxyvitamin D in our own laboratory and other people have measured 1.25-dihydroxyvitamin D, and even when there is rickets these metabolites are high. So we can't really say that this is due to either a defect in supply or a defect in metabolism.

Reporter: So what do you think is causing the disease?

Dr MacIntosh: Well we feel that it's very likely indeed that this is a defect in mineral supply. If a baby is being fed after birth on expressed breast milk, for example, he will be receiving about an eighth of the amount of calcium and the amount of phosphorus that he would be getting at a similar period across the placenta as if the premature baby he was still inside the mother.

Source: Audiotranscript ATO32

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