

AGE DISTRIBUTION OF CERVICAL CANCER IN SAINT PETERSBURG

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Population based cancer registry, St. Petersburg, Russia

Objective: From 1980 to 1996 years incidence of cervical cancer (CC) decreased from $13,1^0/_{0000}$ to $8,2^0/_{0000}$, increased indices ($9,5^0/_{0000}$) were registered at 1997 year in St. Petersburg. The main aim of our study was to identify the age distribution of cervical cancer (CC) in St. Petersburg at 1997.

Methods: Information about 367 cases of CC at 1997 was presented from the Population based cancer registry of St. Petersburg.

Results: There were no incidence of CC at the age before 15 years old, from 15 to 30 years - $4,7^0/_{0000}$, from 30 to 40 years - $14,9^0/_{0000}$, from 40 to 50 years - $16,9^0/_{0000}$, from 50 to 60 years - $20,1^0/_{0000}$, from 60 to 70 years - $18,0^0/_{0000}$, from 70 to 80 years - $36,1^0/_{0000}$ and older 80 years old - $21,1^0/_{0000}$. Squamous carcinoma was observed in 92,3% cases of CC, adenocarcinoma - in 7,7% cases among women to 50 years old and ones in 9,8% cases among women older than 50 years. Distribution according to FIGO clinical stages of patients with CC to 50 years was: I stage - 32,6% cases, II - 33,1%, III - 23,4% and IV - 10,9%. Distribution according to FIGO clinical stages of patients with CC older 50 years was: I stage - 17,6% cases, II - 19,6%, III - 44,9% and IV - 17,9%. Advanced CC increased from patients older 50 years. 73,6% patients with CC at the age before 50 years were treated by surgery, 30,2% - by surgery and X-ray therapy and 24,5% - only by X-ray therapy. 21,2% patients with CC older 50 years were treated by surgery and X-ray therapy and 63,6% of ones were treated by X-ray therapy only.

Conclusions: Incidence of CC was increased in all ages in St. Petersburg at 1997. The I and II clinical stages were revealed in 66,7% patients to 50 years and till in 37,2% patients older 50 years. Patients to 50 years with CC were treated more frequently by surgery (73,6%) comparing with patients with CC older 50 years who were treated by X-ray therapy (63,6%).

PATHOPHYSIOLOGY OF INCREASED NUCHAL TRANSLUCENCY IN CHROMOSOMALLY ABNORMAL FETUSES

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Objective: In about 80% of fetuses with trisomies 21, 18 or 13 and Turner syndrome there is increased collection of fluid in the neck region that can be visualized sonographically at 10-14 weeks of gestation as increased nuchal translucency thickness. The pathophysiology of this common phenotypic expression of different chromosomal abnormalities is uncertain but there is some evidence that the underlying mechanism may be cardiac failure, possibly due to abnormalities of the heart and great arteries, and altered composition of the skin. The latter may be due to a gene dosage effect of the three, rather than the normal two copies of genes, found in trisomies causing an alteration of the extracellular matrix in the skin or abnormal development of the heart and great arteries.

Methods: We performed a number of studies investigating nuchal skin tissue for extracellular matrix components and lymphatic hypoplasia. We also performed studies investigating cardiac heart failure using molecular techniques and doppler studies by measuring the ductus venosus at 12-14 weeks of gestation. Big vessels were analyzed using light microscopy.

Results: Studies investigating the heart found increased mRNA gene expression of ANP and BNP and reduced mRNA gene expression of Calcium ATPase. Doppler studies of the ductus venosus in chromosomally abnormal fetuses found signs of heart failure like a negative A-wave in chromosomally abnormal fetuses. Studies of great arteries in chromosomally abnormal fetuses found narrowing of the aortic isthmus. Studies investigating components of the extracellular matrix in nuchal skin of trisomic fetuses found overexpression of ECM genes in trisomies or an altered ratio of genes and lymphatic hypoplasia in Turner.

Conclusions: The present data provide some evidence, that chromosomally abnormal fetuses with increased nuchal translucency at 12-14 wks may suffer from transient cardiac heart failure due to narrowing of the aortic isthmus or an altered extracellular matrix of the heart and skin. This could be transient because an increase in the radius of the big vessels leads to a decrease in the vascular resistance by ten to the minus four (equation of Hagen-Poiseuille).