Thin Cord Syndrome: case report

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Summary: Thin Cord Syndrome: case report.

The Thin Cord Syndrome (TCS) seems to be liable of the 10% of foetal deaths during the second trimester. The TCS has been specifically used to designate the cases of stillbirths presenting a marked thinning of a short length and a marked reduction of Wharton jelly at the emergence of funiculus from the ombelicus in presence of normally developed foetuses for gestational age. Some or many cases of TCS seem though to be undetected. The causes of TCS are unknown as well as why the presumable caliper reduction of funicular vessels not determine flow abnormalities and growth retardation and arrest before the sudden death. The existence of a foetal factor able to offer a “guaranteed optimal body development” is supposed.

Key words: Thin cord - Intrinsic cord abnormalities. Cordone ombelicale - anomalie del funicolo.

Introduction

Foetal second trimester mortality is a quite rare event which causes an important psychic impact in mothers and several etio-pathogenetic dilemmas in the perinatologists.

Most of times the etiology has no immediate reasonable explanations and a late answer comes from autoptical and laboratory studies of the born. Sometimes however is possible to identify some abnormalities in the funiculus which may disclose the cause of death (1).

The acronym TCS (Thin Cord Syndrome) was first used in 1961 (2) to identify stillbirths generically imputable to stenosis present in any tract of the funiculus. Since years ’90 (3, 4) the acronym has been specifically used to designate the cases of stillbirths presenting: 1) a marked thinning or a marked reduction of Wharton jelly of short length at the emergence of funiculus from the ombelicus in presence 2) of appropriated gestational age (AGA) and sane fetuses (1).

We report a case of TCS.
Case report

Miss Z.A. (n. 2015/02/15995), 34 y.o., para 0000, enters the Maternity Unit of Asiago Hospital (VI, Italy) on October 7 2015 reporting the absence of fetal movements since two days. The past and recent history is negative; at the beginning of the pregnancy a doubt of CMV infection is asserted by elevated IGM in absence of IgG. Being the avidity test negative, the CMV is rejected and a serologic cross reaction is supposed. The echography confirms the gestational age at 11th week and the presence of a normally conformed and developed fetus at 22th week. An office ecography at the hospital admission confirms the presence of a deceased fetus at 24th week.

Three endovaginal administrations of dinoprostone 10 mg (Cervidil®) are necessary to enable the birth of an embryo presenting incipient maceration. A marked stenosis and hyper-coiling with absence of Wharton jelly is present in a tract of three centimetres at the emergence of funiculus from the umbilicus (Figure 1).

The autopsy confirms the presence of a normally developed for gestational age female; three vessels funiculi; marked thinning of 2 cm length at the emergence from umbilicus; marked Wharton jelly reduction; umbilical vein thrombosis and hypercoiling of the rest of funiculus.

Discussion

In the English-language literature, the intrinsic abnormalities of the umbilical cord have several appellations: coiling abnormalities, hypercoiling, overcoiling, constriction, spiralling, stenosis, stricture, thinning, torsion, twists and others, reaching 43 different funicular abnormalities (1). It has also been supposed that a funicular stenosis may be a post mortem artefact rather than a cause of death (5).

Although in case of TCS the death is imputed to the blockage of the blood flow due to stenosis of the funicular vessels secondary to the deplection and/or absence of the Wharton jelly, it is unknown the time between the beginning of the stenosis and of its progression till the exitus; it is unknown the mechanism triggering the stenosis and it is unknown why the exitus comes in adequately developed fetuses for gestational age (6). Histology shows a reduction in the involved funicular vessel caliper (1) which should determine a progressive reduction in blood flow. Actually no guide lines dictate flowmetry during the second trimester in presence of AGA.

At this, we hypothesize the existence of a foetal factor able to offer a “guaranteed optimal body development” analogously to the “maximum allowed weight” in some mammals. Such hypothesis would further theorize the minimal adequate weight of extrauterine survival (today reputed critic or incompatible with a good quality of life) once an adequate ability in extremely immature neonatal assistance is reached (1).

Conclusions

Because probably some or many cases of TCS are undetected either in prenatal echographic screenings or at the post natal inspection and because TCS is responsible of 10% of second trimester stillbirths (4), we foretell a special prenatal and postnatal attention in detecting any possible funicular abnormalities, TCS included, causes of second trimester stillbirths.
References


