

LETTER TO THE EDITOR

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Unexpected sudden death related to medullary brain lesionsReceived: 10 February 2005 / Revised: 10 February 2005 / Accepted: 11 February 2005 / Published online: 18 May 2005
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Lesions of the tractus solitarius nucleus, generally of a congenital nature, may be the pathophysiological substrate of the sudden infant death syndrome (SIDS). The neuropathological studies we have conducted on a large population of victims of SIDS, of unexpected neonatal deaths and fetal deaths at term, revealed analogous congenital lesions of the structures of the brainstem that modulate respiratory activity, as well as cardiovascular functions, arousal and upper digestive activities [1, 2, 3].

The common denominator of all these deaths was the absence of neurological symptoms, generally associated with the presence of congenital lesions and in some cases of acquired lesions. In our studies, we have observed a wide variability of degree and localization of congenital lesions, such as hypoplasia/agenesis of the arcuate nucleus (present in about 50% of the cases we examined), associated with hypoplasia of the reticular formation in fetuses, hypoplasia of the parabrachial/Kölliker-Fuse complex, lesions of the nucleus of the solitary tract, gliosis (an expression of acute lesions or chronic injury), as well as functional alterations of the neurotransmitters, such as catecholamines in the locus coeruleus and somatostatin in the hypoglossus nucleus [3, 4, 5, 7, 8, 9].

We are in agreement with Jaster et al. [10] that subtle lesions involving the solitary tract nucleus, and other nuclei (particularly the arcuate nucleus and the Kölliker-Fuse nucleus), are responsible for disruption of neural pathways, and consequently for sudden perinatal death (of both the term fetus and the newborn) and early infant death without any premonitory neurological dysfunctions.

Our findings of a wide range of acquired lesions, also involving the solitary tract nucleus, determining unexpected infant death, raise very interesting issues. In particular, we have observed a T lymphocytic leptomeningitis affecting the ventral medullary surface [7], two cases with encephalitic features (probably of viral etiology) [9], and a hemangioendothelioma identified in the area postrema, which widely infiltrated the postero-middle brainstem bilaterally, sparing only the dorsal vagus nucleus [8]. Three cases of sudden adult death due to prions were also described [11].

As the volume of data on new morphological and functional alterations of the cardiorespiratory centers of the brainstem accumulates, it becomes ever more clear that it is essential that sudden death victims be submitted to an in-depth necropsy examination, focusing particularly on the brainstem in serial sections [1, 3, 8].

At the 7th International Conference on SIDS in 2002, we proposed [6] that the current definition of SIDS as “the sudden unexpected death of an infant under 1 year of age which remains unexplained after a thorough case investigation, including the performance of a complete autopsy, examination of the death scene, and review of the clinical history” can be effective only if the complete autopsy includes an in-depth histological examination of the cardiorespiratory innervation and specialized myocardium, performed by an experienced pathologist, as described on our web site: http://users.unimi.it/~pathol/sids_e.html.

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