The alteration is characterized by the presence of a non-cardiogenic pulmonary edema of toxic mechanism, with diffuse endothelial damage which, in a small number of cases, developed into ARDS.

The authors say that corticoids do not cause evident modifications in the evolution, but this does not agree with the other known studies.

In addition to satisfactorily resolving edema of the face and extremities which appeared in the acute stage, steroids were very useful in treating the pulmonary affection. They improved the pulmonary lesion clinically, radiologically and functionally, and sometimes saved the life of patients in compromised situations, especially in cases where they were applied early and in high doses.

However, there is no evidence that they have or have not been beneficial in avoiding development of the chronic stage of the disease.

It is said that in the origin of pulmonary hypertension the poison produces pulmonary vasoconstriction. However, the catheterizations carried out with pharmacologic tests to induce vasodilatation did not demonstrate the existence of vasospasm, and it is considered that the fundamental cause of hypertension is the vascular lesion with endothelial damage, vasculitis, thromboembolic complications, and fibrotic sequela in the intima. Up to the present time, pulmonary fibrosis has not been found.

Despite having carried out several studies on different animal species, it has not been possible to reproduce the fundamental lesions of toxic oil syndrome (TOS). There is no definitive model that has been accepted and can be recommended as yet.

Finally, in regard to the toxicologic aspect, we would point out that it was the aniline added to denature the rapeseed oil for industrial uses, and not the acetanilide, which, when reacting with the fatty acids, formed anilides which is considered the index of toxicity of the oil.

Some authors have referred to alteration of the lipid metabolism in the lung of rats fed with oleylanilides. However, this experimental event has not been shown to play an important pathogenic role in ARDS of TOS.


REFERENCES


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Classification of Mycobacteria vs Classification of Mycobacterial Diseases

To the Editor:

It was with a sinking sense of deja vu, subspecies depressant, that I read the comments of McDonald and Reichman (Chest 1983; 84:511-12) on Bailey’s proposed classification of disease caused by different mycobacteria (Chest 1983; 84:625-28). It was as if in the last 50 years of my professional life and those of a number of my colleagues had disappeared into a black hole. McDonald and Reichman bemoan the “complexity” and “nonclinical” nature of the Runyon classification, and then imply that nothing more happened in the field until the development of the “newer” serologic and lipid analyses. They then propose that “a sorely needed next obvious step is to look at the entire family of mycobacteria.” Where have these authors been since 1954, the year of Timpe and Runyon’s landmark paper? Hundreds of microbiologists have labored diligently and effectively to bring order to the genus Mycobacterium. Yes, the classification and the tests needed to achieve that classification are complex. The bacilli are not so obliging as to wear bumper stickers announcing their pathogenic proclivities. We classify them on the basis of their phenotypic properties and phylogenetic relationships, put a name on them in accord with an internationally accepted code of bacterial nomenclature, and then try to establish the relationship of members of each named group to various clinical conditions. A specific pattern of clinical association, difficulty in treatment, etc., may be the only features shared by organisms bearing different species names.

For these reasons, it is not the responsibility of the scientists to get together with a name for the “major groups;” these major groups do not exist as natural phenomena, although individual species do exist, at least as defined by phenotypic and evolutionary discontinuities that can be measured by current methodology. Clinicians can use...