Alcoholic Ketoacidosis as a Cause of Death, Who Came First?

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I read with interest the article ‘The Postmortem Diagnosis of Alcoholic Ketoacidosis’ by Palmiere and Augsburger (2014). However for the sake of truth I must protest against the statement on page 272: ‘The first report in the forensic field suggesting that ketoacidosis could be partially responsible for unexplained deaths in alcoholics dates back to 1993 and concerns a study performed by L.N. Denmark on 49 autopsy cases that included chronic alcohol-abuse related deaths’. Together with my co-authors I submitted an article that was acknowledged by Forensic Science International on 19 January 1993 and published in vol. 60 (Thomsen et al., 1993). In that article we described our results as ‘strongly indicative of ketoacidosis as the sole or contributing cause of death…’. We were convinced that we solved the riddle of ‘Fatty liver deaths’. L.N. Denmark’s excellent work on beta-hydroxybutyrate was received at Forensic Science International, 4 April 1993 and published in vol. 62.

It had been known for many years that alcoholics, who stop drinking after a binge, may be found dead shortly after. The only abnormality to be found is a fatty liver. There are no drugs to be detected and only insignificant levels of alcohol or none at all. There have been numerous speculations as to the cause and mechanism of death. Severe metabolic disturbances including high levels of free fatty acids do probably play a major role due to the effect on the Krebs Cycle. It has since our publications been confirmed that alcoholic ketoacidosis is the cause of death in a substantial number of alcohol abusers.

A quantitative measurement of ketone bodies is now a routine analysis at the institutes of forensic medicine in Denmark. Denmark (1993) measured the levels of beta-hydroxybutyrate in vitreous and urine in 49 forensic cases and found high concentrations in six chronic alcohol abusers with no specific immediate cause of death. He rightly mentioned diabetes, hypothermia and starvation as possible differential diagnoses. In my opinion alcoholic ketoacidosis may well be signed out as the cause of death, but we must be aware of the fact that the mechanism of death is not yet fully understood. The pH levels are usually not very low, and as described by Palmiere and Augsburger (2014), a multitude of biochemical indicators are affected in these cases.

REFERENCES

Thomsen et al. (1993) published an extremely interesting and innovative preliminary communication in Forensic Science International, vol. 60 (1993), in which they presented the results of a forensic study performed on 27 alcoholics with known causes of death, 16 alcoholics with unknown causes of death and 79 control subjects. The determination of blood acetone + acetoacetate revealed significantly increased levels in alcoholics with unknown causes of death compared with the other studied groups. Based on these findings, the authors concluded that their results strongly indicated ketoacidosis as the sole or contributing cause of death in chronic alcoholics with negative postmortem investigation results.

In Forensic Science International, vol. 62 (1993), Denmark (1993) reported the results of a study focusing on beta-hydroxybutyrate as a marker for sudden death in chronic alcoholics. Denmark measured beta-hydroxybutyrate concentrations in vitreous humor and urine. In 6 out of 49 studied subjects, the author found vitreous beta-hydroxybutyrate levels ranging from 19 to 26.9 mg/dl and urine beta-hydroxybutyrate values ranging from 26.7 to 493 mg/dl. In all these six cases, death was initially thought to be related to chronic alcohol abuse and no specific causes of death were obtained based on postmortem investigation findings. Denmark concluded that increased beta-hydroxybutyrate concentration might suggest alcoholic ketosis in subjects with a history of chronic alcoholism, even in those in which previous withdrawal seizures were documented, and therefore be useful in explaining the death. Nevertheless, Denmark emphasized that other situations such as diabetic ketoacidosis and starvation could be responsible for metabolic changes potentially leading to marked ketosis.

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