DIVING DOCTOR'S DIARY

A DIABETIC DAMSEL IN DISTRESS

Carl Edmonds

This story is that of a qualified scuba diver who developed diabetes and required insulin.

Key Words
Diabetes, drugs, safety.

Case report

A 24-year-old was qualified as an open water diver in June 1994. She logged 40 dives after her course and was an enthusiastic and a capable diver.

She had no problems when diving until early in January 1996 when she felt nauseated and vomited soon after reaching depth. She vomited through the regulator and purged it afterwards. She then felt better and continued the dive.

Later that month, after a period in which she had not eaten very much, she was aware of a nervousness during a dive. This was very atypical for her. She was also aware of a dry throat, but the main problems were tremor and distress during a dive which she had completed uneventfully on previous occasions, and in conditions which were otherwise pleasant.

The depth was 12-15 m and there was no environmental or equipment cause for concern. She was aware of hyperventilation, but felt that this would settle after the dive was underway. It did not.

The descent had been feet-first and there was no history of air swallowing or middle ear problems. At depth she felt nauseated and vomited into her regulator. She took it out of her mouth and purged it. She refrained from any further exertion and informed her buddy that she needed to ascend. They did this and she vomited again while being assisted on board.

She was very distressed by the dive and the associated emotional sensations, but this did not prevent her from diving again that evening, uneventfully.

Following this diving incident, and because of it, she sought assistance a few days later, when the history of polyuria and polydipsia, associated with glycosuria, resulted in positive investigations for diabetes. Subsequently she was adequately controlled on isophane insulin (Protaphane) 6 units b.d. and neutral insulin (Actrapid) b.d., 2 units a.m. and 4 units p.m.

During stabilisation she was aware of occasional hypoglycaemic symptoms and signs, in the form of irritability, aggressive behaviour, shaking, paraesthesia and numbness, and a sensation of derealisation. She was not aware that hypoglycaemia could lead to unconsciousness and convulsions.

Now the only complications that she has from her diabetes are mild hypoglycaemic episodes, which are easily controlled.

She knew that there was disagreement in the diving medical fraternity about the safety of diving with diabetes.

Discussion

I explained to her that, as she had a perfectly valid open water certificate (C Card) I had no legal right to stop her from scuba diving. Nevertheless I strongly advised her against such an activity. I also stated that I would not consider her suitable for passing the Australian Standards 4005.1, which is normally required for scuba diver training.

I agreed with her about the importance of maintaining a good state of physical fitness, nutrition and the associated diabetic control.

HYPOGLYCAEMIA

The control of diabetes, in the scuba diving environment, is particularly difficult. This is partly due to the variable exertion that can be required, and often is required, to regain the safety of land or boat. When exertion is maximal this puts a great deal of strain on the insulin-glycogen-carbohydrate metabolism. Under these conditions there is an increased likelihood of hypoglycaemia.

The possibility of mild hypoglycaemic attacks producing atypical or fewer symptoms in the aquatic environment was also explained, together with the progression of this condition to unconsciousness and epileptic convulsions (and the probable fatal consequences of this, if it occurs underwater).

It would certainly be wise, if she were to insist upon scuba diving, to restrict it to extremely gentle environmental conditions that are not likely to cause any significant physical demands on her, i.e. diving in waters

without current and with facilities to ensure no significant exertion.

Statistics on unselected insulin dependent diabetic divers are not available. A retrospective survey of survivors who continue diving, (always the best population to demonstrate "favourable" results) suggested that 15% had experienced hypoglycaemic episodes underwater.[1]

The diabetic diver is between the traditional rock and a hard place. The hypoglycaemic episodes, which are less likely to be recognised under water, will be induced by a situation in which the person is already at risk, swimming against a current in an attempt to return to safety. The energy requirements and glucose utilisation will be close to maximum. Hypoglycaemia will be far more likely, and the accident will be particularly difficult to cope with, both during the convulsive phase and the pre- and post-ictal confusional states.

KETO-ACIDOTIC REACTIONS

Most of these cases have been in experienced divers. Dr Peter Chapman-Smith presented a case of a diabetic physician/diver at the 1982 SPUMS Annual Scientific Meeting in Madang,[2] over a decade ago, although I am not sure if Peter (or anyone else) realised the significance of the case at the time.

The association of a diabetic syndrome (with acidotic state), dyspnoea and hyperventilation (with excessive air consumption), a confusional state and atypical (panic) behaviour, impressed both Peter and his buddy, as did the result.

The problem has also been referred to briefly in diving medical texts,[3] based on similar cases.

Some diabetics have continued diving but have found it necessary to suspend the pre-diving dose of insulin, to reduce the hypoglycaemic episodes. Unfortunately this is likely to predispose to the development of the keto-acidotic reactions, which this diver describes so very accurately in her "pre-diagnostic" dives (all my other cases were in established diabetics, so this lass posed a temporary diagnostic dilemma).

If insulin dosage is reduced to a significant degree, to ensure that adequate blood glucose levels are maintained despite the excessive metabolic demands, then the combination of insulin deficiency and glucagon excess is likely to increase the fatty acids and other ketones in the blood, cause a reduction of pH and the associated changes in potassium, sodium, magnesium and bicarbonate metabolism. Respiration is stimulated.

The resultant increased respirations are magnified in their psychological effect by the restrictions induced by excessive breathing through a demand valve. This will be especially so with increased depth, as the resistance to breathing increases.

The keto-acidotic episode, which is nowhere near that seen in the diabetic ketotic coma, can and does produce the episodes described above. It is unfair to dismiss these manifestations as merely a "near panic" or anxiety episode. That belittles the probable organic (biochemical) basis of the disorder.

In this case, the additional and possibly precipitating symptoms of anorexia, nausea and vomiting, preceded the more typical dyspnoeic complex and the emotional reaction/confusional states. The latter can occur in the most well-balanced, non-neurotic diabetics.

In this case, the relative effects of physical stress (a common precipitant of keto-acidosis), insufficient food intake and the vicious cycle of vomiting and dehydration, could well have had a less favourable outcome with less capable divers.

DECOMPRESSION SICKNESS

A warning was given regarding the increased risk of decompression sickness (DCS) with diabetes, and the need to reduce both the allowable bottom time and the maximum depths, with dive exposures. A suggestion (not based on any factual information) was made that reduction of the allowable bottom time by at least 50%, and diving to a maximum of 15 m, with a minimum surface interval of 6 hours, might reduce the risk of significant DCS.

In considering the explanations for the increased likelihood of DCS, the causes could be multiple. Possibly the dehydration associated with the diabetic state (especially after withheld insulin), hyperosmolarity, increase in blood viscosity, and increased thrombotic tendency, are all likely to increase the DCS syndrome.

Alternately, the already damaged walls of diabetic vessels might be a factor in the intravascular bubbles causing further pathology. This is all theoretical, but the results are not. It does seem as if diabetics, once they get DCS, get it with gusto.

OTHER PROBLEMS

I briefly mentioned autonomic neuropathy, cardiac sequelae and other problems associated with sea water exposure, including infections, and hoped that she would not pursue her intention to continue scuba diving.

The seminar, at the 1996 Undersea and Hyperbaric Medical Society Annual Scientific meeting, on diabetes and diving is, according to my information, likely to be no more informative than the one on asthma. So one is going to have to rely on common sense, a knowledge of diabetes and one's experience in diving medicine to advise patients. Extremely "soft" statistics, enthusiasm from protagonists and a desire to be avant-garde will also influence some medical advisers.

Others will use medical approval for motor vehicle driving as a corollary for diving, despite the vastly different demands of the two environments and the occasional case report showing that even driving for "controlled" insulin dependent drivers is sometimes lethal for them and their passengers.

References

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THE WORLD AS IT IS

CHAMBER FAILURE

Harry Oxer

Key Words
Fire, hyperbaric facilities, hyperbaric oxygen, treatment.

The incident

On February 25th 1996 at 1505 on the first floor of a hospital at Yamanishi, Japan, there was an event that rocked the hyperbaric units of the world. There had been an apparent explosion associated with a monoplace chamber, and the violent disruption killed two people, and seriously injured a third.

Professor Hideo Takahashi, president of the Japan Hyperbaric Society and Head of Hyperbaric Medicine at Ngoyo University, gave a special presentation on this tragic accident at the Undersea and Hyperbaric Medicine Society Scientific meeting, in Anchorage, Alaska, in June 1996. This is a report of that presentation.

A 74 year old man was undergoing hyperbaric treatment for the chronic results of a brain infarct. He was recovering slowly, but had expressed a keen desire to have hyperbaric oxygen with a view to accelerating his recovery, and had been accepted for hyperbaric oxygen therapy.

All the treatments in this unit are run by two clinical engineers", as they call the technician operators, under the supervision of one hyperbarically trained neurosurgeon. There were three monoplace chambers in the unit, a 1989 Kawasaki, a 1990 Seechrist, and a 1992 Seechrist.

At the time of the occurrence there were patients in two of the chambers There were two technician engineers running the chambers, and observing the patients. At the time of the problem nobody was looking at this particular patient as the clinical engineer caring for this patient was speaking to a visiting doctor at the door of the room. The other was caring for a second patient in another monoplace chamber in the same room. There was an explosion and the 74 year old male in the chamber was severely burned. A hatch blew off one end and killed his 70 year old wife instantly. One of the engineers received a fractured skull from a flying end plate and two other people were slightly injured.

There was no fire and the external fire extinguishers were not activated. There was evidence of an intense fire within the shell which was smoke blackened. The chamber failed in the way in which it was designed to fail. Both the safety relief valves had operated, and there was evidence of soot passing through them, but of course they could not accommodate an explosive force.

The oxygen supply ceased immediately with the explosion and there was no subsequent fire within the unit. The windows of the room were blown out, as were light partition walls, and the ceiling was disrupted.

The patient was 45 minutes into a treatment at 2.7 ATA on 100% oxygen.

Initially a statement was released that all recommended safety procedures had been fully carried out.