RED HERRINGS

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Red Herrings do not actually occur in New Zealand's subtropical waters but the handsome red snapper is an attractive alternative. It is hardly surprising that New Zealand has a larger population of divers per capita than any other nation. This reflects a readily available underwater environment, lengthy coastlines, and a fascinating marine world. Most sports diving occurs on the East Cape and the Three Kings Islands in the far North, beckoned particularly by the off-shore island chain. Perhaps SPUMS may have an opportunity to explore this region in years to come.

Inevitably diving related incidents or accidents present to interested medical practitioners, and I would like to present two short cases for discussion. I have chosen my title because it could be that neither problem was related to diving.

Case 1

An experienced New Zealand Underwater Association (NZUA) trained 27 year old male scuba diver presented twice in 6 months with recurrent facial swelling, apparent at shallow depths on ascent. He initially felt "numb" in his right cheek ("like going to the dentist"), the swelling spreading across his upper lip to the midline. This increased over 24 hours and subsided spontaneously over 2 to 4 days.

He dived on most days, predominantly for crayfish. They were frequently hard working dives with reputed non-decompression bottom times of between 5 and 20 minutes. This was to depths of between 100 and 140 feet. (Several other divers confided that he dived below 200 feet on occasions so there is doubt whether his histories are reliable). His ascent rate was allegedly normal, and he always had a snack of potato chips and coke just before diving. He was a non-smoker with no history of previous diving accidents. He was a mild asthmatic on no regular medications but with multiple allergies (to pollen, grass, dust, etc.), and occasional sinusitis, presumably on the basis of allergic rhinitis.

This swelling occurred on 4 occasions, but not on successive dives, and while using different regulators. On one of these occasions he noted reverse ear squeeze (barotrauma of ascent) on ascent, and on another, developed a post-dive bi-temporal headache for approximately 10 minutes.

He presented acutely, as invited, 6 months after his initial presentation. On examination several hours post-dive, he was neurologically normal with an oedematous right upper lip, which was 2 to 3 times its usual size. There were no other relevant signs or symptoms. (On his initial presentation 3 days post-dive he had a similar facial wheal appearance with several small red maculopapules in the same distribution).

X-rays were reported to demonstrate "moderate soft tissue swelling in the inferior aspect of the left antrum". There was apparent dental caries in the right posterior upper molar (which appeared to be non-vital) and also in a right upper premolar. No subcutaneous emphysema was noted.

The viability of a root pulp cannot be determined on radiological appearances alone, but the loss of bone density around the apices was indicative of non-vitality.

He was referred for a dental opinion, but was last seen bound for Hawaii on a yacht.

The differential diagnoses

1. Lymphatic or capillary obstruction causing lymphoedema as in minor decompression sickness.

2. Dental caries with apical gas tracking, but against this is the fact that no subcutaneous emphysema was noted, either clinically or on X-ray.

3. Local allergy to the rubber or metal of the regulator, but against this is that he used a number of different regulators.

4. Any other suggestions.

Case 2

This case is reported with the subject's blessing.

A trained and experienced 54 year old male scuba diver presented with a sudden visual deficit on a charter trip to the Three King Islands 50 miles West of North Cape right off the top of New Zealand. The vessel "Elingamite" attempted to bulldoze the West King Island in a storm during a night in 1902. The island is perhaps 100 yards wide! Some divers have made their fortune and some have since met their maker in subsequent salvage attempts on this moderately accessible wreck. The cold blue water and abundant fish life inevitably command attention, but strong, unpredictable currents and remoteness make it still a relatively untouched and potentially hostile area for divers. This diver had dived in the area previously.

After 5 days of regular diving, mostly in search of that elusive superb underwater photograph, this man had a leisurely first non-decompression dive at 100 feet. After approximately a three hour surface interval he was loading film in his camera aboard the charter boat when he stooped forward head down. He then coughed feeling as though he was perhaps developing a cold. On sitting upright he felt that maybe something was unusual in his vision. Coming out from the dark cabin to the daylight it was apparent that a red curtain was descending in his right superior visual field. This continued to descend equatorially over the next few hours. He was aware of some light above the redness peripherally from the onset of symptoms. After specialist consultation by radio, he aborted his trip. He had sustained an inferior pre-retinal haemorrhage in his right eye.

This man was physically unfit, being mildly obese with uncontrolled maturity onset diabetes melitus, diagnosed 9 years before. He was usually normotensive. His non-insulin dependant diabetes was "controlled" with diet and a small dose of an oral hypoglycaemic agent which had not been changed for some years. A medical practitioner by occupation he had not had any laboratory investigations in the preceding 18 months, and did not attend a GP.

In the 2 days before this incident he had been caught in a powerful surface current involving heavy finning. Wisely he had decided not to dive after contending with the current for several minutes. He could not swim the 20 feet to the anchor chain. The following day he had made a non-decompression dive to 120 feet and had rapidly ascended from 20 feet after running out of air after an abnormally increased air consumption! His buddy had 1500 psi remaining at the end of the dive. It had been a very gentle dive.

Eventually it turned out that he had significant diabetic proliferative retinopathy, severe in one eye, and strangely enough to a mild degree in his remaining left good eye. Fundoscopy and fluoroscein angiography confirmed that he had sustained a vitreous haemorrhage in his right eye, presumably from neo-vascularisation capillary loops. His fasting blood glucose was in excess of 17 mmol/1.

The retina receives its blood supply from two sources. The inner two-thirds is supplied from the central retinal artery, while the outer one-third photo-receptors and pigment epithelium receive their arterial supply from the choriocapillaris, single layer of capillaries attached to the outer layer of Bruch's membrane.

Both juvenile and maturity onset diabetes predispose a diabetic retinopathy which is related to the duration of the disease and its degree of control. In proliferative retinopathy fragile new vessels grow from the retina or the optic nerve head adhering to the posterior surface of the vitreous. Vitreous contraction can pull on these new vessels causing bleeding into the retrohyaloid space or into the vitreous body itself. Associated retinal detachment may occur. Many conditions may cause such bleeding, eg. trauma, systemic diseases (diabetes, hypertension, leukaemia), increased arterial pressure, increased venous pressure, inflamed vessels, arterio-venous malformation, retinal tears, others (tumours etc).

Blood in the vitreous body clots rapidly, forming a red mass with sharp borders. This is related to the collagen fibres and clotting substances present. In the retrovitreal space however blood remains fluid.

It is worth noting that vitreous haemorrhages can occur at rest in bed even with stable diabetic control. This is more dependent on the presence of proliferative retinopathy.

The intraocular pressure is related to the mean arterial pressure, but is generally a stabilising influence on the arterial and venous pressures. Intraocular pressure (IOP) is increased by hypoxaemia, hypercapnia, coughing, sneezing, straining, venous obstruction in the head and neck, and increased CSF pressure. IOP is decreased by hyperoxia, hypocapnia, hyperventilation, and systolic BP less than 85 mmHg.

This case raises the following points.

1. What was the cause of the haemorrhage? Was it merely concurrent uncontrolled systemic disease, or transient hypertension with exertion in previous days, or a cough causing raised venous pressure and IOP, or a bubble phenomena due to decompression sickness, silent bubbles or air embolism.

2. The medical practitioner as a diver. How significant was the denial of the importance of systemic disease, the likely absence of proper medical screening, and the absence of a general practitioner.

3. Fitness to dive. Surely this story raises the problem of medical problems arising in our latter years and perhaps causing unfitness to dive. I would suggest that there is a need for medicals every 5 years over the age of 40, to retain certification.

<u>*COMMENTS*</u>

These are two fascinating cases from our New Zealand colleague . The first case seems to be consistent with a number of other similar ones who have had recurrent episodes of purely lymphoedema with decompression sickness. I feel that had either a trial of pressure, or the inhalation of 100% oxygen for a few hours been performed, then the diagnosis would have been verified.

Like the first case, the second is a very informative one and serves to remind us of similar situations in the past. The final accident that the diabetic diver experienced is well described and explained. I think Peter skipped over a very good demonstration of one of the problems of the diabetic diver, when he glossed over the incident the day before. Peter and his diabetic buddy performed the same dive but the diabetic panicked and was in an "out of air" situation while Peter had over half his air pressure remaining.

Diabetics often have this trouble. In an attempt to avoid the very likely episode of hypoglycaemia associated with extreme or unexpected exertion, he has no option other than to reduce his insulin or anti-diabetic medication. In doing this, he must increase his blood glucose level, together with the associated acidotic products. Thus he has no option other than to dive in a more or less acidotic state. He is then likely to over-breath his regulator and result in either excessive usage of gas and therefore an out of air situation, or panic because of the resistance from over-breathing the regulator. In this case, apparently both things happened.

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