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HAEMOCONCENTRATION IN NEUROLOGICAL DECOMPRESSION ILLNESS

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Running Head :

Haemoconcentration in Neurological DCI

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Abstract

Decompression illness (DCI) is attributed to the formation of bubbles, resulting from the reduction of the ambiant pressure. Circulating bubbles lead to capillary leak syndrome, extravasation of plasma and haemoconcentration. Experimental model of animals have shown that an haemoconcentration carried a poor prognosis. We have measured the haematocrit level in fifty-eight consecutive sport divers victims of neurological DCI admitted to our hyperbaric center, and in sixteen control divers.

No significant difference was found in the haematocrit values between the divers with neurological DCI (median 42.5 per cent) and the controls (median 41.75 per cent) ($p= 0.1$).

The median haematocrit level was significantly higher for divers with neurological sequelae when compared with control ($p=0.01$) or with divers without sequelae ($p< 0.05$). An haematocrit level $\geq$ 48 per cent was correlated with persistent neurological sequelae one month after the accident ($p=0.01$). However, an haematocrit $<$ 48 per cent had no prognostic value.

Key words : Decompression Illness, Haematocrit, Prognosis

Introduction

Decompression illness is a serious disease because neurological decompression accidents among amateur divers can be permanently incapaciting. Numerous biological disorders, and particularly haemoconcentration, have been reported in decompression illness (19) and previous experimental study have reported that a high haematocrit level carried a poor prognosis (3). Although little is known about haematocrit in sport divers.

The aim of this prospective study was to evaluate the haematocrit levels in divers victims of neurological decompression illness (DCI) and to assess whether there is a relationship between the haematocrit level and the prognosis, defined as the persistence of sequelae.

Patients and Methods

PATIENTS :

From May 1991 to September 1994, all the divers who presented troubles suggesting a decompression accident following a SCUBA (Self Contained Underwater Breathing Apparatus) dive and admitted to the Salvator hospital hyperbaric center were included in the study. Patients who were suspected of a pulmonary barotrauma with cerebral air embolism were excluded.

The therapeutic course after the admission in our hyperbaric center associated hyperbaric oxygen (HBO) therapy and a medical treatment.

HBO therapy consisted of recompression to 2 Atm. abs. with 100% oxygen during at least two hours. Hyperbaric treatment was stopped when two successive examinations one hour apart showed no clinical progression, and after no more than six hours in every case. This HBO procedure has been performed in our hyperbaric center

for the patients with neurological DCI, since 1985 (17). It prevents nitrogen saturation secondary to compression with air and we observed no neurological aggravation during the decompression. The therapeutic results, with this HBO procedure, are comparable with those of traditional HBO procedures (2).

A medical treatment consisting of aspirin (1 gr) and fluid loading according to the haematocrit value, was always associated.

In cases of residual signs or symptoms after the first hyperbaric course, additional courses of one hours at 2.5 bar (FiO2=1) were undertaken daily until total recovery or stabilization of symptoms. All the patients with motor disorders received physiotherapy.

METHODS :

Neurological evaluations

A complete neurological examination was performed close in admission at the hyperbaric center. Data were reported in a standardized file, which permitted reproductible examinations. Neurological examinations were also performed every hour during the HBO procedure, after the decompression, and every day until the patients were discharged from the hospital.

A neurological examination was also performed one month after the initial SCUBA dive, the medical examiner was blind to patients' pretreatment haematocrit. The results of treatment were assessed one month after the accident and classified as : total recovery or sequelae. Sequelae were defined as persistent neurological troubles one month after the accident. Neurological troubles were defined as neurological signs or symptoms (subjective or objective sensory, motor, urinary disorders). Longer-term outcome could not be determined because a large proportion of the divers were lost to follow-up.

Controls

During the same period, sixteen control divers were also studied, as the control group. They were healthy volunteers, experimented divers, who had performed a recreational dive with the respect of the MN 90 decompression tables (recommandations of the "Marine nationale, 1990"). Informed consent was obtained for the sixteen control divers

Laboratory investigations.

Haematocrit levels were measured for the divers and the controls, at the hyperbaric center.

Blood samples (5 ml) were taken in citrate tubes on admission to the hyperbaric center. After centrifugation (3000 rpm, 2 minutes), the haematocrit levels were measured by the ratio of the globular volume to the volume of the total blood. A second measure was systematically performed to confirm the first result.

Haematocrit levels were also measured in the group of controls using the same technic as those applied to the group of patients. Samples were taken three hours after emersion. This delay was predetermined because in our hyperbaric center the median delay between emersion and admission is three hours (1).

Statistical analysis.

The Mann-Whitney's U test was used to compare median haematocrit level.

We have evaluated the sensibility, specificity, positive predictive value and negative predictive value of several consecutive haematocrit values to determined the best prognostic value of the haematocrit level

The chi-square test was used to determined correlation between qualitatives parameters. Yate's correction was used in cases of small numbers.

Differences between groups were considered significant at $p < 0.05$.

Results

Patients.

From May 1991 to September 1994, sixty-one consecutive divers suspected of having neurological DCI were treated in our hyperbaric Center. Among the sixty-one divers, pulmonary barotrauma with cerebral air embolism was diagnosed in two patients, unconsciousness resulting from a near-drowning episode in one patient. These three divers were excluded from this study.

The remaining fifty-eight divers consisted of fourty-eight men and ten women, with a mean age of 33 +/- 9 years. They were experimented divers who had performed a recreational dive. The mean diving depth was 40 +/- 15 msw. Rapid ascent or evident disrespect of decompression tables were noted in twenty five cases (43 percent), repetitive dives in eight cases (14 percent). All of them had evidence of neurological signs and/or symptoms resulting from neurological decompression illness. Eighty eight percent of the divers developed symptoms within 10 minutes of emersion. Initial manifestations consisted of sensory (95% of cases), motor (47%) and urinary (24%) disorders. Twelve patients (21 percent) had paresthesia and pain evocative of a spinal cord injury without motor disorders. The sensory disorders were of a subjective (paresthesia or pain) or objective nature (superficial, deep and thermo-algesic disorders). Motor disturbances ranged from monoparesis to paraplegia. TABLE 1 shows the initial manifestations in the 58 divers.

Fourty eight divers (83 percent) received medical treatment, consisting of normobaric oxygenotherapy and/or aspirin, before the admission at hyperbaric center. Twenty-eight divers (48 per cent) received 500 ml to 1000 ml fluid loading (drink or through an intravascular catheter). During transport to hospital, fourty two patients improved (72 per cent), twelve remained stable (21 per cent), and four deteriorated (7

per cent). The median delay between emersion and HBO therapy was 192 minutes (3 H 12 min).

After the first hyperbaric oxygen session, we observed a total recovery in 29 divers (50 per cent), a partial recovery in 25 (43 per cent), and no clinical progression in 4 (7 per cent). Secondary relapse many hours after the first HBO therapy was noted in six cases (10 per cent). Thirty-one patients (54 per cent) had additional HBO courses (mean six procedures, ranging from 2 to 30).

One month after the accident, 67 per cent of the divers were asymptomatic (n=39), while 33 per cent had sequelae (n=19).

controls

There were appropriate matching of patients and controls (TABLE 2). None of them had any neurological trouble following the dive.

Haematocrit values.

No significant difference was found in the haematocrit values between the divers with neurological DCI (median 42.5 per cent) and the controls (median 41.75 per cent) (p= 0.1).

The median haematocrit level was significantly higher in divers with sequelae when compared with control (p=0.01) or with divers without sequelae ($p<0.05$). (TABLE 3).

There was no significant difference in the delay between emersion and HBO therapy in the divers with sequelae (median = 3 H15), and the divers without sequelae (median = 3 H); (p=0.9).

There was no significant difference in the prescription of fluid loading between the divers with sequelae (12/19), and the divers without sequelae (16/39); (p=0.1).

Divers with sequelae performed a deeper dive (median = 45 msw) when compared with the divers without sequelae (median = 36 msw); (p= 0.04) No correlation was found between the haematocrit level and the depth of dive. Spearman test; (p = 0.3).

The best prognostic value was determined with a cut off of a haematocrit of 48%.

Sensitivity : 47.37 %

Specificity : 87.18 %

Positive predictive value : 64.29 %

Negative predictive value : 77.27 %

As shown in TABLE 4 an haematocrit ≥ 48 per cent was correlated with the presence of sequelae one month after the accident (p=0.01).

No significant difference was found in the depth of dive between patients with an haematocrit level ≥ 48 percent (36.5 msw) compared with patients with an haematocrit level < 48 percent (39 msw); (p=0.8)

No significant difference was found in the delay of admission between patients with an haematocrit level ≥ 48 percent (3 H 05 min) compared with patients with an haematocrit level < 48 percent (3 H 40 min); (p=0.24).

Discussion

With the growing popularity of diving, accidents are increasingly frequent. Neurological decompression illness is a serious accident, as they can lead to permanent neurological disorders. It is attributed to the formation of gas bubbles resulting from a reduction in ambiant pressure during the ascent. The pathogenesis of the neurological disorders is complex, including formation of bubbles in blood or in tissues, rheological changes and reduced tissue perfusion (8). Furthermore, circulating bubbles disrupt endothelial cells, activate leukocytes and platelets and induce an activation of the complement system and a release of kinines (6, 10, 16, 18, 21, 22). This global phenomenom results in a capillary leak syndrome with extravasation of plasma (14) and heamoconcentration.

Experimental study have reported that the haematocrit increases during the first hour after a decompression and then decreases slowly to reach normal value after a 24 hours delay (15). Bove, in a 1974 experimental animal model observed that the existence of an haemoconcentration is correlated with a poor prognosis (5).

In DCI following diving accidents, an haemoconcentration have been reported with a frequency ranging from 40 to 80 percent of cases (8, 12). Hypovolaemia can involve a circulatory inefficiency (7). This haemoconcentration may increase the blood viscosity, which can reduce the tissue perfusion and increase the neurological lesions (23). However, no study had determined whether haemoconcentration is associated with a poor outcome in divers victims of neurological DCI.

In the present study, no difference is found in the haematocrit level between divers with decompression illness and controls. However, blood samples were collected after a fluid loading in 48 per cent of cases. Treatment with fluid loading and HBO therapy give a better prognosis in animals victims of severe decompressions (24), so that, a treatment including fluid administration, normobaric oxygen therapy and aspirin, recommended by the "Fédération Française d'étude des sports sous marins" (French

Federation for Underwater Sports and Study) in neurological DCI (25), is often performed before the admission at our hyperbaric center.

We have reported a significantly higher haematocrit in divers with sequelae when compared with controls or with divers without sequelae. Furthemore we found a correlation between an haematocrit ≥ 48 per cent and the persistence of sequelae one month after the accident. Then the haematocrit seems to be correlated with the severity of the neurological DCI.

However, patients with sequelae have performed a deeper dive compared with patients without sequelae. An high haematocrit level can be secondary to a deeper dive with a longer dive, a more dehydrating exercise and an hyperbaric diuresis (9,20). In the present study, no correlation was found between depth and haematocrit level, and these two parameters could be two independant factors. Furthemore, no difference was found in the depth of dive in patients with an haematocrit ≥ 48 per cent when compared with patients with an haematocrit < 48 per cent.

So, the haematocrit level on admission may be an important prognostic factor and a high haematocrit level may be evocative of a serious DCI even if the neurological examination suggest mild disease. Then, among the six divers with secondary relapse, four had a haematocrit level ≥ 48 per cent. A previous study shown that secondary relapse is correlated with persistent neurological disorders (3).

An haematocrit level < 48 per cent can be observed in divers with a poor neurological outcome. Among the 44 divers with an haematocrit < 48 per cent, 10 had neurological disorders one month after the accident. Then, the haematocrit level is a poor discriminator in individual cases and must only warn the pratician when the level is high.

The persistance of a high haematocrit values, despite initial intravascular fluid infusion shows that important volume of fluid can be necessary. There might be a need for repetitive measurement of haematocrit because haemoconcentration is deleterious in cerebral or medullar ishemia. (13)

Finally, the haematocrit level could be used in a gravity index to assess the gravity of a population of divers victims of decompression illness (4).

Conclusion

In this study, no difference is found in the haematocrit level between sport divers victims of neurological decompression illness and controls.

An haematocrit ≥ 48 per cent is correlated with persistent neurological sequelae one month after the accident and must warn the physician even if the initial neurological examination suggest mild disease. However, an haematocrit < 48 per cent has no prognostic value.

The measure of the haematocrit on admission to the hyperbaric center could be used in a gravity index to assess the gravity of a population of divers victims of decompression illness .

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TABLE 1 : Initial symptoms in the 58 patients

	numbers of cases	percentage
hypoesthesia	13	22
paresthesias	42	72
paraplegia	8	14
monoplegia	3	5
tetraparesis	3	5
paraparesis	10	17
monoparesis	3	5
dysuria	14	24

TABLE 2 : Characteristics of the 58 patients and the 16 controls.

	patients	controls	p
age (median)	31.5 years old	31.5 years old	0.3
sexe	48 men, 10 women	13 men, 3 women	0.8
mean depth of dive (median)	39 msw	32 msw	0.1
intervals between emersion and blood sampling (median)	3 H 12	3 H	0.5
lengths of dive (median)	28.5 min	30 min	0.9

TABLE 3 : Haematocrit level in patients and controls

		Haematocrit	(per cent)	
	n	Médian	min	max
Controls	16	42.5	39	48
Divers without neurological sequelae	39	42	35	57
Divers with neurological sequelae	19	47.5	32	69.5

TABLE 4 : Haematocrit ≥ 48 per cent and prognosis

	Ht < 48 per cent n= 44	Ht ≥ 48 per cent n= 14
TOTAL RECOVERY n= 39	34 (77 per cent)	5 (36 per cent)
SEQUELAE n= 19	10 (23 per cent)	9 (64 per cent)

Haematocrit ≥48 per cent significantly correlated with the existence of neurological sequelae one month after the accident (p= 0.01).