for a fulfilling life and career. Any deficit in it is as frightening for the person as a loss of vision. It's a handicap potentially severe enough to deny a person a normal life and livelihood.

Sensorineural hearing loss (SNHL) is often encountered by the ENT surgeon. It is established globally as a significant morbidity. Even more traumatizing is the development of a sudden sensorineural hearing loss by a patient, a disease afflicting 1 in every 5000 patients in the general population [1]. Various etiologies are proposed to explain the sudden SNHL in general population and include inflammatory (bacterial, viral and autoimmune), vascular (hemorrhage, microemboli, thrombosis), metabolic (hormonal, chemical, drugs), traumatic (direct, noise, surgical), membrane rupture, functional, or idiopathic. In many of the unidentifiable cases there is still debate between a viral versus vascular etiology.

Sensorineural hearing loss can also be explained as a surgical complication of otologic surgeries. However, what has raised the great interest of medical professionals around the world in recent decades is the occasional development of sudden sensorineural hearing loss with non-otologic surgeries carried out under general anesthesia. A considerable majority of these surgeries include cardiopulmonary bypass procedures. The first report of this kind was made by Arenberg (1972) [2] who reported sudden unilateral deafness immediately following cardiopulmonary bypass. Earlier Brownson et al (1971) [3] had tried unsuccessfully to achieve the same in a prospective study with a limited sample. This was followed by a larger retrospective study by Plasse et al [4,5] in which he evaluated 7000 patients and found the incidence of SNHL with aortocoronary bypass surgery to be 1 in 1000 (0.1%). However, these results are highly questionable considering the presence of false-negatives and false-positives associated with retrospective studies. It has been established since that SNHL can be objectively determined only in routine and prospective evaluation.

There was controversy in the past in the demarking of criteria for SNHL. This was evident in the study by Shapiro et al [6] who applied overly inclusive criteria and found the hearing loss to be 13.2% in a prospective study involving 68 patients. Doubts were always raised though, regarding the inclusion in his study of the patients who reported a hearing deficit of up to 10 dB, which can be owing to the potential variations and audiometric testing errors due to patient concentration and co-operation during the testing.

This was followed by two case reports from Millen et al [7] and a prospective study by Ness et al [8].

A review of the literature indicates several distinct variants. First, it is found that the SNHL in patients undergoing coronary artery bypass grafting (CABG) occurs in an older age group with known preexisting cardiovascular disease [1]. Second, there is a clear male to female preponderance in the incidence of these cases that cannot merely be explained by the greater number of male patients undergoing the procedure [4].

It has also been found that there is little or no recovery from the insult with a partial recovery of hearing occurring in less than 50% of patients [1]. Another point is the absence of vertigo in most of these cases [4,5]. Tinnitus may also be found [6].

The magnitude of the problem has long been recognized considering the incidence rates that are reported up to 10–15% [6], and the fact that even in 1980 over 100,000 bypass operations were carried out in the United States [9]. Various etiologies had thus been proposed to look into the pathophysiology of this phenomenon and thus offer a possible preventive or curative measure. Microembolic phenomena (fat, air or particulate thrombi)[2], perioperative hypotension or perfusion failure [6], hypercoagulable states [10], and ototoxic drug usage [6] are some of the better recognized ones.

However, many of the mechanisms involving CABG (e.g. fat emboli, anti-foaming agents) do not apply to those other non-otologic surgeries, which have reported sudden idiopathic SNHL. Since all of the reported cases have occurred under general or spinal anesthesia, the hemodynamic fluctuations during induction and maintenance of anesthesia common to both of these could be a factor [11]. Cochlear membrane leaks and perilymphatic fistulae have been invoked as possible mechanisms for loss of hearing. Both implosive and explosive mechanisms have been implicated [12]. Nitrous oxide is often blamed as it can generate very high pressures in the middle ear [13,14]. However, there have been described cases in which nitrous oxide was not used, and its presence in many cases may only reflect its very widespread anesthetic use. It is also important to exclude a brainstem event causing bilateral hearing loss [15,16], though in these reports other neurological signs like ataxia, dysarthria, gaze palsies and multiple cranial nerve and neurological deficits have been mentioned alongside.

Methods

Study design

This is a prospective matched cohort study.

Setting

Aga Khan University Hospital, Karachi, Pakistan.