

Intralabyrinthine Hemorrhage After General Anesthesia: Case Report

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ABSTRACT

Several cases of intralabyrinthine hemorrhage are described in the literature. The causes are mainly anticoagulants and blood disorders. The diagnosis is suspected on clinical examination (sudden cochleo-vestibular syndrome) and confirmed by inner ear magnetic resonance imaging (MRI). We report the case of a 58-year-old woman with a history of neuro-syphilis with motor sequelae, ischemic heart disease under antiplatelet therapy, rheumatoid arthritis and colonic adenocarcinoma treated by surgery and adjuvant chemotherapy. This patient presented a unilateral cochleovestibular syndrome immediately after a short general anesthesia. MRI showed a spontaneous T1 signal hyperintensity confirming a left labyrinthine hemorrhage. The clinical course was marked by regression of balance disorders without auditory recovery. Intralabyrinthine hemorrhages are rare and often secondary to anticoagulants or blood diseases. Through this article, we report a case of intralabyrinthine hemorrhage secondary to a brief general anesthesia.

Keywords: *Intralabyrinthine Hemorrhage, General Anesthesia, Deep Deafness, Vertigo, MRI*

Introduction

Intralabyrinthine haemorrhage is a rare cause of acute idiopathic unilateral cochleovestibular deficit (Shinohara *et al.*, 2000). Several etiologies have been identified such as anticoagulant treatments and blood diseases (Meunier *et al.*, 2020). To our knowledge no cases of intralabyrinthine hemorrhage secondary to general anesthesia were reported in the literature. Advances in magnetic resonance imaging (MRI) make it possible to confirm the diagnosis suspected by clinical examination by showing spontaneous hyperintensity in T1-weighted sequences not enhanced with gadolinium.

Case Presentation

A 58-year-old woman was referred by gastroenterology department for sudden deafness associated with vertigo in the immediate aftermath of a colonoscopy under general anesthesia. She has a twenty-year history of neuro-syphilis with motor sequelae such as gait disorder, right hemibody clonus, cognitive disorders of short-term memory, epilepsy since childhood, ischemic heart disease treated with antiplatelet therapy (Clopidogrel@75mg), rheumatoid arthritis and colonic adenocarcinoma treated with surgery and adjuvant chemotherapy in 2004. The patient also presents a decrease in visual acuity for 10 years without any clear pathology for cause.

The anamnesis confirmed a significant decrease of the left auditory acuity associated with rotatory vertigo in the immediate aftermath of a colonoscopy performed under general anesthesia. A feeling of blocked ear and homolateral hemi-cranial headache have also been reported.

Physical examination noted normal tympanum, unstable Romberg test with no clear latero-deviation and non-significant deviation index. There was no cerebellar syndrome. Videonystagmoscopy does not reveal any spontaneous or induced nystagmus.

Tone audiometry performed the same day (Fig. 1) revealed a left cophosis and a right sensorineural hearing loss of 60 dB. Auditory Steady-State Responses (ASSR) performed on the tenth day confirmed the left cophosis (Fig. 2).

A 60% persistent left vestibular deficit was demonstrated on videonystagmography performed two months after the onset of symptoms (Fig. 3).

Magnetic resonance imaging performed 3 days after the onset of symptoms showed normal encephalic floor with constitutional hypoplasia of the basilar trunk and vertebral arteries. The sections centered on the inner ear showed a spontaneous hypersignal in T1 and Flair of the basal turn of the cochlea.

The clinical course was marked by progressive regression of vertigo in 15 days. The cophosis was persistent three months after the first ENT consultation.

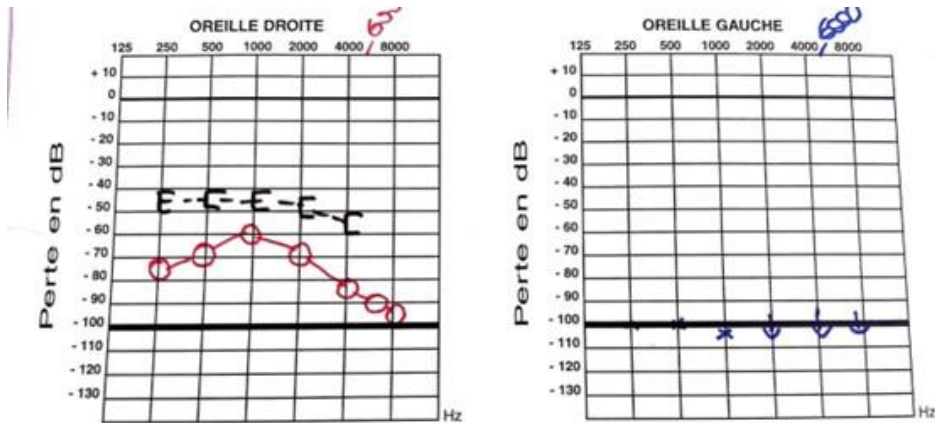


Figure 1: pure tone audiometry.

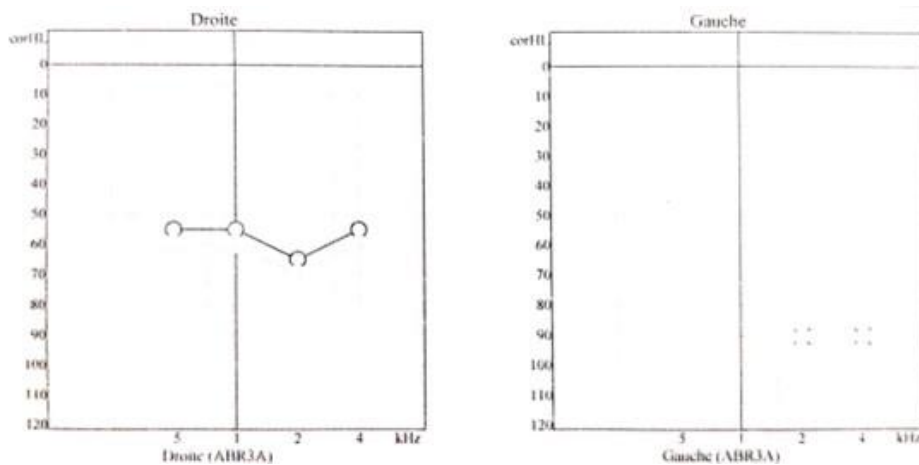


Figure 2: auditory evoked potentials (ASSR) : left cochlosis.

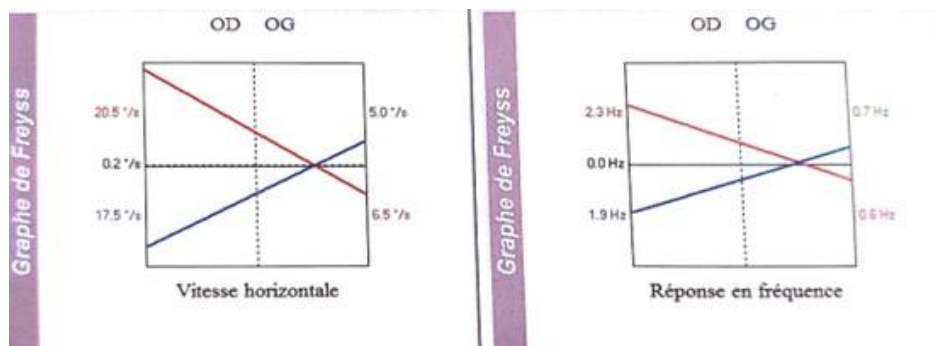


Figure 3: Videonystagmography caloric test: left sided vestibular deficit.

Discussion

Intralabyrinthine hemorrhage is a rare cause of acute cochleovestibular deficit. About twenty cases have been described in the Anglo-Saxon literature (Shinohara *et al.*, 2000). These symptoms may occur simultaneously (Vellin *et al.*, 2005) or can be delayed (Shibata and Johnston, 2015).

Several causes of intralabyrinthine hemorrhage have been identified. Anticoagulant treatment, which is not the only etiology, is often found during anamnesis. Hematological diseases such as leukemia, myeloma, Waldenstrom's disease or sickle cell disease are also described in the literature (Meunier *et al.*, 2020).

Autoimmune diseases such as systemic lupus erythematosus (Sugiura *et al.*, 2006) or iatrogenic causes such as radiotherapy and some chemotherapies have been incriminated (Poh and Tan *et al.*, 2007).

The use of toxic products such as cocaine with its vascular effects can also induce intra labyrinthine hemorrhage (Nicoucar *et al.*, 2005).

In this case we describe a patient with a history of a former chemotherapy not explaining the recent symptomatology.

In the literature, we have not found any intralabyrinthine hemorrhage following general anesthesia. In the case described here by the authors, the general anesthesia was of short duration without any complication.

One may wonder if a hypertensive crisis could have triggered this haemorrhage. The intervention sheet does not mention any blood pressure lability (blood pressure oscillating between 90/60 mmHg and 120/75 mmHg).

Following the operation, after the onset of vertigo and deafness, our patient underwent a haematological check-up including a CBC with a study of the blood count, which came back without any particularity.

Let us recall that the body mass index of our patient was 19.5 kg/m² and that she did not report any headache, nor any headache associated with the vertigo and the decrease in her hearing, which did not allow us to exclude with certainty the absence of intra-cranial hypertension.

No bacteriological or virological research was carried out, as the clinical context was not in favour (no fever, normal biological check-up, no clinical signs of call).

Our patient had undergone colonoscopy under general anesthesia with Propofol as part of the surveillance of his colonic adenocarcinoma. Propofol is known as a drug that causes controlled arterial hypotension and vasodilatation of the surgical site, thus allowing a surgical procedure, especially an endoscopic one, to be performed under better conditions.

However, despite this vasodilatory and hypotensive effect, Propofol can cause capillary bleeding despite a low systolic pressure (Ko, 2012).

The diagnosis of intralabyrinthine hemorrhage, strongly suspected in anamnesis and physical examination is confirmed by MRI in the form of a spontaneous T1 hypersignal unenhanced after injection of Gadolinium. The T2 signal is variable depending on the age of the hemorrhagic stroke (hypo signal initially progressing to iso, then hyper signal) (Fig. 4).

The T2 FLAIR sequences of the encephalic floor allow to erase the signals of the cerebrospinal fluid (CSF) and of the inner ear fluids, and to reveal the presence of blood in the acute and subacute phase in the form of a frank hyper-signal located at the level of the temporal bone (Vellin *et al.*, 2005).

The evolution of intralabyrinthine hemorrhage is unfortunately not very favourable with persistent deafness in most cases (Tan *et al.*, 2010).

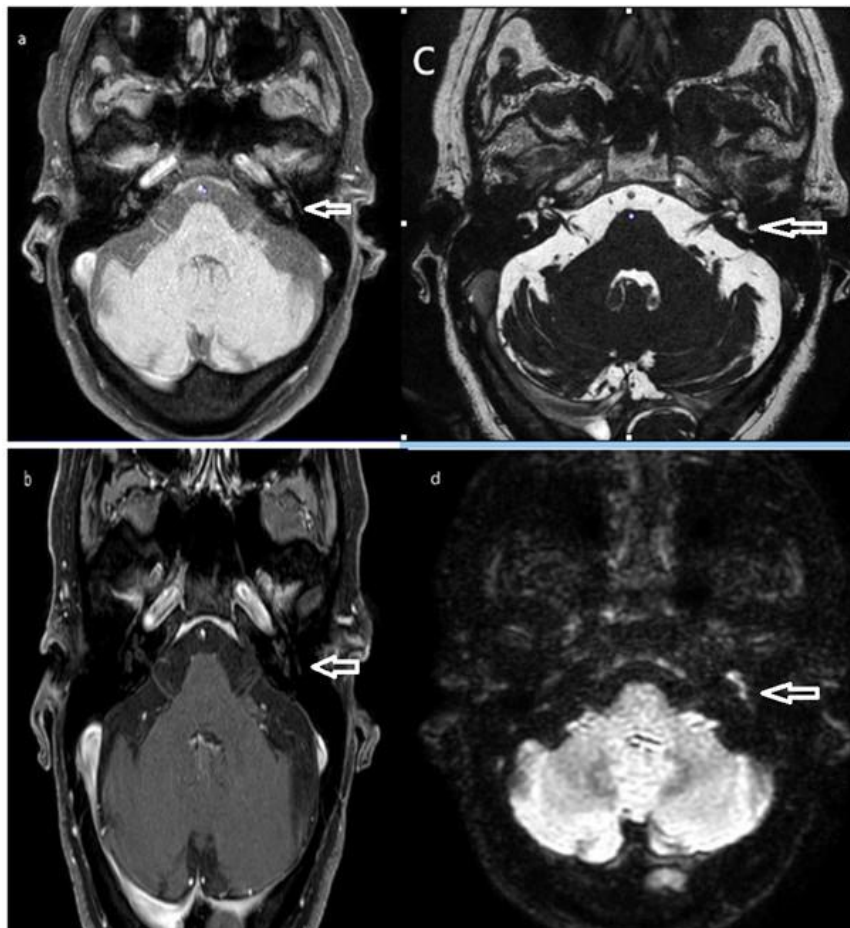


Figure 4: MRI of the inner ear: axial slices on the 10th day of the accident. A spontaneous hypersignal in the cochlea (Arrow points to the hypersignal) and vestibule of the left ear in a T1 sequence (a), No enhancement after gadolinium injection (b) symmetric signal of the labyrinth is observed in T1 sequence (c) and a clear hypersignal of the left cochlea and vestibule in T2 FLAIR sequence (d).

Conclusion

Intralabyrinthine hemorrhage should be evoked in front of any acute cochleo-vestibular syndrome in a vascular terrain or coagulation disorders. No previous case of intra labyrinthine hemorrhage secondary to general anesthesia have been reported in the literature. MRI allows confirmation of the diagnosis.

Patient Consent: The consent to publish this information was obtained from study participant. We confirm that written proof of consent to publish study participants are available when requested and at any time.

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