Guillain-Barre syndrome after lung lobectomy: is there any relationship?

Plojoux, Jérôme Pierre Olivier; Buffard-Morel, Marine; Ducamp, Diane; Laroumagne, Sophie; Dutau, Hervé; Grapperon, Aude-Marie; Thomas, Pascal; Astoul, Philippe J

How to cite

PLOJOUX, Jérôme Pierre Olivier et al. Guillain-Barre syndrome after lung lobectomy: is there any relationship? In: The annals of thoracic surgery, 2014, vol. 97, n° 5, p. e133–134. doi: 10.1016/j.athoracsur.2013.12.067

This publication URL: https://archive-ouverte.unige.ch/unige:79231
Publication DOI: 10.1016/j.athoracsur.2013.12.067

Guillain-Barre Syndrome After Lung Lobectomy: Is There Any Relationship?

Jérôme Plojoux, MD, Marine Buffard-Morel, MD, Diane Ducamp, MD, Sophie Laroumagne, MD, Hervé Dutau, MD, Aude-Marie Grapperon, MD, Pascal Thomas, MD, PhD, and Philippe J. Astoul, MD, PhD

Department of Thoracic Oncology, Pleural Diseases, and Interventional Pulmonology, Hôpital Nord; Department of Neurology and Neuromuscular Diseases, La Timone; Thoracic Surgery Department , Marseille Lung Transplant Group, Hôpital Nord; and School of Medicine, Aix-Marseille University, Marseille, France

Guillain-Barré syndrome (GBS) is an acute inflammatory polyradiculopathy frequently triggered by infection. It has also been reported in some cases after surgical procedures. We describe the first case of GBS occurring 9 days after lung lobectomy for localized lung cancer and efficiently treated with intravenous immunoglobulins. The exact physiopathology of GBS after surgical procedures is unknown. An immune-mediated process and perioperative infection are the most accepted etiologic hypotheses.

(Ann Thorac Surg 2014;97:e133–4)

Among postsurgical inflammatory neuropathies, Guillain-Barre syndrome (GBS) has been infrequently reported. This acute inflammatory polyradiculopathy, characterized by autoimmune peripheral nerve demyelination, is frequently associated with digestive or respiratory infections [1]. Other triggers are probably related. The role of vaccinations is subject to debate, as are all the events involving cell-mediated and humoral immunity directed against host tissue [2]. GBS has been described after epidural anesthetic procedures and in the setting of cardiac operations, operations on the spine, gastrectomy/esophagectomy, obstetrical maneuvers, and renal transplantation [3–5]. We herein describe the case of a patient in whom GBS developed after upper right lung lobectomy for lung cancer.

A 67-year-old man, a former smoker (20 pack-years) with ischemic heart disease and former laryngeal cancer (T1aN0M0) cured by radiotherapy 3 years previously was discovered to have a 10-mm nodule in the upper right lung during regular follow-up care. Right hilar adenopathy was seen on a computed tomographic scan, and because of a high suspicion of primary pulmonary malignancy (T1aN1M0), the patient underwent upper right video-assisted thoracoscopic lobectomy and complete mediastinal lymph node dissection. Pathologic analysis confirmed complete resection of an 18-mm poorly differentiated epidermoid carcinoma without lymph node involvement (Stage Ia, pT1aN0M0R0). There was no use of locoregional anesthesia and no requirement for blood transfusion. Postoperative follow-up was marked by fever without recognized infection and right chylothorax, successfully cured with a low-fat diet. The chest tube was removed after 8 days of drainage, and the patient was discharged from the hospital.

The next day (ninth postoperative day), the patient was admitted because of a high fever (40°C). Surgical infectious complication was ruled out, but the patient experienced progressive ascending four-limb paresis on the 11th postoperative day, which suggested GBS. Muscle strength was preserved, but reflexes were abolished. The results of serologic testing for infectious conditions (*Campylobacter jejuni,* mycoplasma, cytomegalovirus, human immunodeficiency virus, hepatitis B virus, hepatitis C virus, syphilis, and salmonellosis) and autoimmune conditions (protein electrophoresis and immune electrophoresis, antineutrophil cytoplasmic antibodies, antinuclear antibodies, antinucleoprotein antibodies, complement, anti-myelin associated glycoprotein antibodies, serum and cerebrospinal fluid antineuronal antibodies) were negative. The CSF biochemistry was normal (protein 0.43 g/L, glucose 3.8 mmol/L), and cellular elements were low (red blood cells 3/mm^3, white blood cells 0/mm^3). There was no oligoclonal repartition of CSF proteins. A nerve conduction study (NCS) on the fourth day after the onset of symptoms yielded normal results. Marked neurologic worsening was observed on the eighth day, with lower limb muscle weakness impeding upright stance, anesthesia of the hands and feet, ataxia, and dysautonomy. A second NCS on the tenth day revealed motor and sensory abnormalities consistent with segmental demyelination, and a diagnosis of postoperative GBS was made. The patient was transferred to the intensive care unit and received a 5-day course of intravenous immunoglobulin therapy (Clairyg, LFB Biomédicaments, Courtaboeuf, France) 0.4 g/kg/day, total dose 140 g. A steady-state phase was noted on the 11th day after the first GBS symptom, and the recovery phase started on the 19th day. The patient was discharged home after 1 month with the help of a walker and had fully recovered after 1 year as seen during a follow-up visit.

Comment

Guillain-Barré syndrome is the most common cause of neuromuscular paralysis, with an annual world incidence of 1.3/100,000 [6]. Even though the underlying cause is not fully understood, digestive or respiratory infections in the 6 weeks preceding GBS are well-known triggers. Frequently associated microorganisms are *Campylobacter jejuni,* human immunodeficiency virus, Epstein-Barr virus, and cytomegalovirus [1]. The incidence of GBS after surgical procedures is not precisely known. Gensicke

Accepted for publication Dec 18, 2013.

Address correspondence to Dr Astoul, Thoracic Oncology, Pleural Diseases, and Interventional Pulmonology Department, Hôpital Nord, Chemin des Bourrely, 13915 Marseille Cedex 20, France; e-mail: philippejean.astoul@ap-hm.fr.

0003-4975/$36.00
http://dx.doi.org/10.1016/j.athoracsur.2013.12.067

and colleagues [5] conducted a retrospective study of 63 patients with diagnoses of GBS, of whom those, 6 patients (9.5%) had undergone a surgical procedure in the 6 weeks preceding GBS symptoms. The relative risk of GBS in patients undergoing operations was 13.1-fold higher than in the rest of the population studied, and authors concluded that an operation should be considered a risk factor for the onset of GBS. The risk of postoperative GBS was 4.1 per 100,000 surgical operations [5]. Other forms of inflammatory postsurgical neuropathies are less frequently reported [7]. They can present in either a focal, a multifocal, or a diffuse fashion. The incidence of postsurgical inflammatory neuropathy is unclear. The total incidence of postsurgical neuropathies constituting mechanical nerve injuries is highly variable (0.03% to 0.14%) [7].

Many cases of postoperative GBS have been reported, mostly after cardiac operations [3], brain procedures, operations on the spine [4], or locoregional anesthetic procedures [8]. It has been less frequently reported after gastric, esophageal, and colon operations or orthopedic procedures. To our knowledge, we describe the first case of GBS after surgical lung lobectomy.

The underlying mechanism of GBS after surgical procedures is unknown. The most reported hypothesis is based on a surgically induced transient immunosuppression that could favor subclinical exogenous infections as a trigger of GBS on one side and exposition of antigen by a surgical procedure inducing an immune cross-reaction against myelin proteins on the other side. For instance, it has been noticed that bacteriologic colonization of the airway is found in more than 48% of patients at the time of lung cancer diagnosis [9]. This high prevalence of airway colonization could be an infectious trigger component, particularly in lung operations. A vigorous systemic inflammatory response characterized by early and late phases involving both the humoral and cellular pathways is well described in cardiac operations with cardiopulmonary bypass [10] and could be a favorable trigger for the more frequent description of GBS after cardiac operations. Corticosteroid prophylaxis has been attempted to reduce complications related to inflammatory burst after cardiopulmonary bypass, with variable results on the incidence of atrial fibrillation and the length of stay in the intensive care unit [10]. In cases related to epidural anesthesia, anesthetic agents could, by a local effect, interact with myelin function or properties, and the needle itself could damage myelin proteins, exposing antigens as a trigger of immune reaction [8]. In most reported cases, the delay between the operation and the first symptom of GBS was less than 10 days. NCS is highly sensitive and specific for establishing the diagnosis of GBS. Most patients receive immunoglobulin or plasmapheresis therapy. The rationale for these therapies is the high titer of antibodies in the early course of GBS.

Plasmapharesis replaces the plasma with albumin and is the most direct method of reducing the concentration of antibodies. Its clinical use has been proved to be effective [11]. The intravenous administration of high-dose immunoglobulin was found to be as effective as plasmapheresis [12]. The potential therapeutic effects of intravenous immunoglobulin include modulation of complement-activation products, neutralization of pathogenic antibodies, saturation of macrophage receptors, and suppression of cytokines and chemokines [6]. The prognosis for recovery in weeks to months is good for the great majority of patients with postsurgical GBS.

Postoperative GBS is a severe but rare neurologic complication. It has been observed after different types of surgical procedures, including a lung operation in this case. This diagnosis should be considered by the physician who faces unexpected neurologic complications after operation and confirmed by NCS. Establishing the diagnosis allows for the provision of appropriate therapy and monitoring with recovery in the great majority of cases.

References

1. Jacobs BC, Rothbarth PH, van der Meché FG, et al. The spectrum of antecedent infections in Guillain-Barré syndrome: a case-control study. Neurology 1998;51:1110–5.
2. Lasky T, Terracciano GJ, Magder L, et al. The Guillain-Barré syndrome and the 1992-1993 and 1993-1994 influenza vaccines. N Engl J Med 1998;339:1797–802.
3. Campbell AJ, McKinlay K, Scott NB. Guillain-Barré syndrome after cardiopulmonary bypass. J Cardiothorac Vasc Anesth 2009;23:82–3.
4. Battaglia F, Sevy A, Moyse E, Roche P-H. Guillain-Barré syndrome following severe head trauma and spine surgery. Rev Neurol (Paris) 2013;169:166–8.
5. Gensicke H, Datta AN, Dill P, Schindler C, Fischer D. Increased incidence of Guillain-Barré syndrome after surgery. Eur J Neurol 2012;19:1239–44.
6. Kuwabara S. Guillain-Barré syndrome: epidemiology, pathophysiology and management. Drugs 2004;64:597–610.
7. Staff NP, Engelstad J, Klein CJ, et al. Post-surgical inflammatory neuropathy. Brain J Neurol 2010;133:2866–80.
8. Steiner I, Argov Z, Cahan C, Abramsky O. Guillain-Barré syndrome after epidural anesthesia: direct nerve root damage may trigger disease. Neurology 1985;35:1473–5.
9. Laroumagne S, Lepage B, Hermant C, et al. Bronchial colonisation in patients with lung cancer: a prospective study. Eur Respir J 2013;42:220–9.
10. Augoustides JGT. The inflammatory response to cardiac surgery with cardiopulmonary bypass: should steroid prophylaxis be routine? J Cardiothorac Vasc Anesth 2012;26:952–8.
11. Plasmapheresis and acute Guillain-Barré syndrome. The Guillain-Barré syndrome Study Group. Neurology 1985;35:1096–104.
12. Van der Meché FG, Schmitz PI. A randomized trial comparing intravenous immune globulin and plasma exchange in Guillain-Barré syndrome. Dutch Guillain-Barré Study Group. N Engl J Med 1992;326:1123–9.