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Diffuse Large B-cell Lymphoma Associated With Exposure to Agent Orange

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Abstract

Agent Orange (AO) is a toxic herbicide carcinogen mixture of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) that was used extensively during the Vietnam conflict of the 1960s and 1970s. We present a case of an elderly veteran who presented with a progressively enlarging right neck mass. During active combat in Vietnam, he experienced extensive exposure to AO. He was eventually diagnosed with stage I diffuse large B-cell lymphoma (DLBCL) with locally advanced/bulky disease. The patient was deemed higher risk for systemic disease given the skin breakthrough at presentation. Additional labs revealed prior infection with hepatitis B with immunity. There is an association between hepatitis B virus infection and non-Hodgkin lymphoma (NHL). Large epidemiologic studies demonstrate a two times higher risk of developing DLBCL, and it is linked to worse outcomes. He commenced therapy on a rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone (R-CHOP) regimen for six cycles. He was also treated with oral entecavir prophylaxis for 18 months and continued for at least one year after his last rituximab dose.

Categories: Pathology, Internal Medicine, Oncology
Keywords: 2 3 7 8-tetrachlorodibenzo-p-dioxin (tcdd), agent orange, bcl-2, diffuse large b-cell lymphoma, sub-mandibular mass

Introduction

Agent Orange (AO) was used extensively as an herbicide during the Vietnam War and classified as a human carcinogen by the International Agency for Research on Cancer in 1997 [1]. Although hostilities ended over 50 years ago, the long-term consequences still affect the health of our veterans in the present day. The most significant medical consequences of AO exposure include multiple oncologic conditions; of these, lymphoid malignancies, like B-cell lymphomas, are the most common hematologic cancers seen in clinical practice [2]. The harmful effects suffered by exposed veterans are encountered in clinical practice up to the present day. Over the past five decades, research into AO and its long-term effects has identified multiple comorbidities that increase the likelihood of not only developing a malignancy but also developing a more aggressive form [3].

Our case of aggressive diffuse large B-cell lymphoma (DLBCL), germinal center B cell (GCB)-like, is a timely reminder of the hazards associated with AO, especially for patients with multiple systemic co-morbidities. These comorbidities include type 2 diabetes mellitus, ischemic heart disease, hypertension, hyperlipidemia, obesity, pulmonary disease (tobacco-induced chronic obstructive lung disease), liver disease (hepatitis and cirrhosis), and dementia [4]. Our patient had a 40-pack-year smoking history and prior hepatitis B infection with an elevated hepatitis B surface antibody. We elucidate the effects of prior hepatitis B and the prognostic implications in the setting of DLBCL.

Among all lymphoma subtypes, DLBCL is consistently identified and supported as the most common subtype and also demonstrates an elevated risk after AO exposure (OR, 1.26) [5]. 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), or the chemical compound of AO, inhibits cell apoptosis and allows malignant B-cells to survive, thus impairing immune response [5]. Our case highlights these common comorbidities and sheds light on their related and cumulative effects. Clinicians who care for veterans from this era should use a multifactorial risk assessment when managing this subset of patients.

Case Presentation

A 78-year-old male veteran who served in the Vietnam War from 1965 to 1967 was admitted with an enlarging right-sided neck mass that started growing eight months ago. Four months after its onset, the patient presented to his primary care physician for the submandibular mass and was given a month-long trial of oral clindamycin and referred to an oncologist for a computed tomography (CT) scan of his head and neck. The mass expanded rapidly over the next four months and eventually began ulcerating with episodes of burning pain over his right mandible and maxilla. In the current presentation, he denied fever, night sweats, recent significant weight loss, and fatigue. His previous medical history included late-onset ulcerative colitis (not treated with immunosuppressants), squamous cell carcinoma (SCC) of his left ear

status post-surgical excision without chemotherapy, two years prior. He had a 40-pack-year smoking history but no alcohol or other substance use. During his active combat in Vietnam, he experienced extensive exposure to AO. He recounted that he would often see aircraft overhead dispersing AO across the surrounding terrain. He described many instances where his platoon would traverse through regions that had just been exposed or were undergoing AO dissemination. He often referred to these experiences as "getting slimed." Throughout his service in the Vietnam War, the chemical was dispersed in his immediate vicinity, and he was directly exposed multiple times.

On physical exam, the patient had a right submandibular mass measuring approximately 10 × 8 cm with a large area of overlying ulceration and necrosis that was firm and fixed but without tenderness to palpation (Figure *1*). The area had spontaneous drainage of serosanguinous fluid. He also had a mild right-sided facial droop and an inability to puff out his right cheek due to difficulty closing his mouth. Sensation and facial nerve movements were otherwise intact. The rest of his physical exam revealed poor dentition, no other lymphadenopathy, thyromegaly, or hepatosplenomegaly.



FIGURE 1: Right submandibular mass, approximately 10 cm × 8 cm with a large area of central ulceration and necrosis.

Initial laboratory results were positive for low serum protein and albumin and mildly elevated serum transaminases (Table *1*). Peripheral blood smear demonstrated leukocytosis with neutrophilia. Additionally, there was mild normocytic anemia and thrombocytopenia. Some neutrophils showed cytoplasmic vacuoles. No abnormal cells were seen.

Laboratory test	Result at presentation	Normal range
White cell count (WCC)	***7.4 × 10^9/L***	4.5-11.0 × 10^9/L
Hemoglobin	***13.1 g/dl***	14-18 g/dl
Platelets	***144,000 per µL***	150,000-450,000 per µL
MCV	90 fL	80-100 fL
Blood urea nitrogen (BUN)	19 mg/dL	6-20 mg/dL
Creatinine (Cr)	1.0 mg/dL	0.6-1.2 mg/dL
Alkaline phosphatase	78 U/L	44-147 U/L
ALT SGPT	***73 U/L***	7-56 U/L
AST SGOT	***53 U/L***	8-33 U/L
Total bilirubin	0.4 mg/dL	0.1-1.2 mg/dL
Total protein	5.8 g/dL	6.0-8.3 g/dL
Serum albumin	***2.8 g/dL***	3.5-5.0 g/dL
Lactate dehydrogenase (LDH)	174 U/L	87-271 U/L
Hepatitis Panel	-	-
Hepatitis A antibody IgM	Non-Reactive	Non-reactive
Hepatitis B surface antibody (Q)	***42.56***	<10 IU/L
Hepatitis B core antibody (HBcAb)	***Reactive***	Non-reactive
Hepatitis B surface antigen (HBsAg)	Non-reactive	Non-reactive
Hepatitis B core antibody IgM	Non-reactive	Non-reactive
Hepatitis C antibody total	Non-reactive	Non-reactive
HIV	Negative	Negative

TABLE 1: Laboratory findings (abnormal values boldened and italicized).

A CT scan of the patient's head and neck (Figure *2*) demonstrated a 7.2 cm × 7.2 cm × 7.3 cm heterogenous soft tissue density mass in the right submandibular region with a large area of overlying irregular ulceration, necrosis, and vascularity alongside an approximately 1.1 cm × 1.4 cm × 2.5 cm mass likely representing an enlarged metastatic lymph node. The patient was admitted for expedited workup and further management. The initial differential diagnosis included adenocarcinoma, recurrent SCC, and lymphoma.

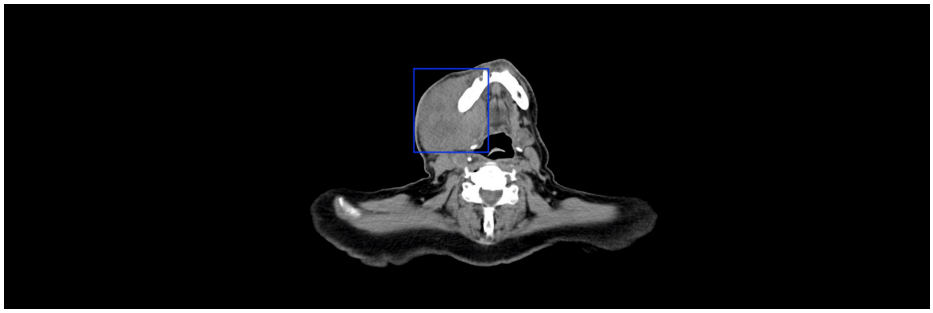


FIGURE 2: CT scan of jaw with a 7.2 cm × 7.2 cm × 7.3 cm heterogenous soft tissue mass in the right submandibular region (blue box).

A positron emission tomogram (PET) scan (Figure 3) demonstrated an intensely hypermetabolic right submandibular mass with a maximum standardized uptake value (SUVmax) of 24.6 and hypometabolic regions, likely from necrosis, and two hypermetabolic subcutaneous nodules in the posterior neck: CT 119 (SUVmax 8.4) and CT 132 (SUVmax 3.6).

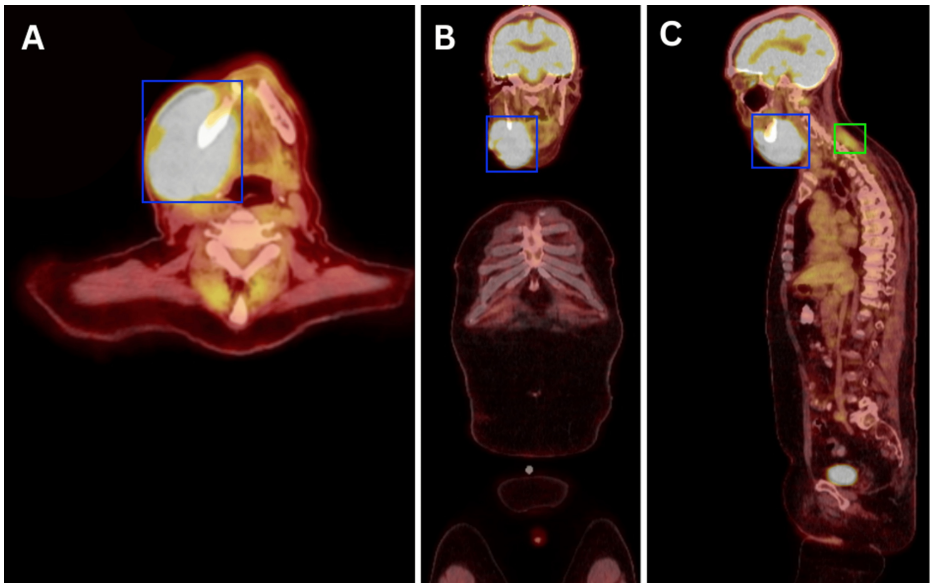


FIGURE 3: Image A: Positron emission tomography (PET) scan demonstrating an intensely hypermetabolic right submandibular mass (blue box, SUVmax 24.6) with hypometabolic regions, likely from necrosis, and two hypermetabolic subcutaneous nodules in the posterior neck. Images B and C: Frontal and lateral PET scan images demonstrating right jaw (blue box) and posterior neck adenopathy (green box).

Our surgical pathologist performed a core biopsy (Figure 4) of the lesion. Initial bedside microscopy demonstrated small blue round cells, most concerning for malignancy. Final biopsy results were notable for a high-grade malignant DLBCL, GCB-like.

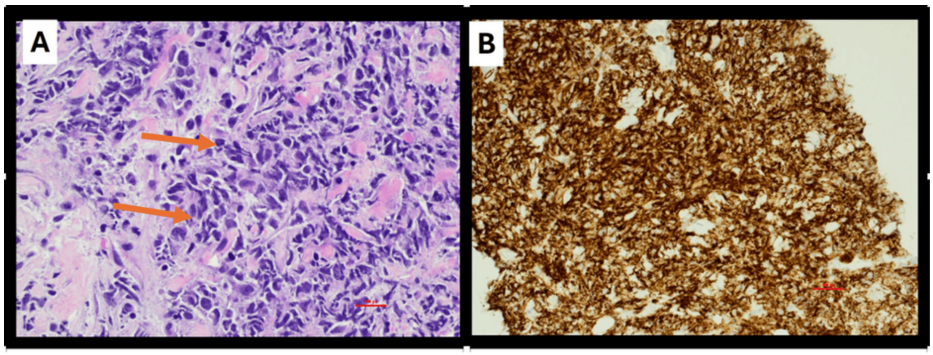


FIGURE 4: Immunohistopathology of the core biopsy of the right neck mass. 2A: H&E stain demonstrating lymphoid cell sheets with irregular nuclear contours, vesicular chromatin, prominent nucleoli, and reduced cell cytoplasm (orange arrows). 40× magnification. 2B: Demonstrates diffuse staining for CD20. 20× magnification.

Immunohistochemical stains (Table 2) demonstrated atypical cells positive for CD10, CD20, CD45, BCL2, BCL6, and PAX5. All immunohistochemical stains were performed with appropriate positive and negative controls. An excisional specimen was submitted for flow cytometry and Fluorescence In Situ Hybridization

(FISH) Double/Triple Hit panel (MYC, BCL-2, and BCL-6). The FISH was positive for only the BCL-2 rearrangement and negative for Epstein-Barr virus-encoded RNA.

Stain	Result	Clinical significance in DLBCL
CD45	Positive	Establishes a tumor of lymphoid/hematopoietic origin
CD10	Positive	Germinal center B-cell (GCB) subtype and better prognosis
CD20	Positive	Therapeutic target for rituximab, better prognosis
BCL2	Positive	Associated with inferior overall survival and progression-free survival
BCL6	Positive	Associated with the GCB subtype and a more favorable prognosis
PAX5	Positive	Establishes B-cell lineage

TABLE 2: Immunohistochemistry.

Based on Hans' algorithm, immunohistochemistry is used to classify DLBCL into GCB versus non-GCB subtypes. This is important for prognostication, as GCB subtypes have a significantly better prognosis than non-GCB subtypes when treated with immunotherapy (77% versus 34% overall survival) [6].

The final assessment was stage I DLBCL (if not, stage II) but with locally advanced/bulky disease. The patient was deemed higher risk for systemic disease given the skin breakthrough at presentation. His Eastern Cooperative Oncology Group (ECOG) status [7] was assessed as 1. His International Prognostic Index (IPI) score was calculated at 1 due to his age over 60. This confers an 80% chance of progression-free survival. He commenced therapy on a rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone (R-CHOP) regimen for six cycles. Oncology recommended six cycles of R-CHOP given the patient's rapidly enlarging jaw mass, skin ulceration, and remote rural location with a complicated follow-up plan, despite his IPI score and ECOG status being favorable. Bone marrow biopsy (BMB) was also declined. This was unfortunate as PET CT can replace BMB in most DLBCL patients when PET CT is positive for bone marrow disease; however, when PET CT is negative, BMB is indicated to uncover occult disease that could potentially change prognosis and management [8].

During the patient's admission, he was mostly asymptomatic. He reported occasional pain that was managed well with Tylenol, gabapentin, and oxycodone. Facial droop seen on physical exam was concerning for nerve involvement. Given his motor and sensory function was intact, facial droop was likely secondary to the mass effect of the tumor. Imaging was not correlated with any facial nerve impingement. Our wound consult team also assisted with bandaging the lesion, which frequently had bloody discharge. Additional labs revealed prior infection with hepatitis B with immunity but were negative for hepatitis A, C, and HIV. As he was to receive rituximab, he was seen by the hepatology service, who recommended continuing oral entecavir prophylaxis throughout the treatment period and up to one year after the last dose of rituximab.

After the third and fourth cycles of R-CHOP, there was significant shrinkage in his right mandibular lesion. He was able to tolerate chemotherapy well with some fatigue and right-sided facial discomfort. Following completion of chemotherapy, the patient returned about a month later. All that remained of the original jaw mass were two 2-3 cm exophytic nodules of unclear onset along his right mandible with associated right V3 numbness and pain as evaluated by the otorhinolaryngologist. The follow-up PET scan demonstrated persistent intense fluorodeoxyglucose uptake near the right mandibular/submandibular region (SUVmax 18.5) with several nodular densities and a significant interval decrease in prior right neck mass burden. He was then referred to interventional radiology for core biopsy. His biopsy yielded results pertinent for a DLBCL, most likely a GCB-like lymphoma since the patient's immunostains were positive for CD10, CD20, CD45, BCL2, BCL6, and PAX5 and likely part of the original lesion. His excisional specimen is still undergoing flow cytometry and FISH analysis. At his last follow-up visit, given the persistence of DLBCL and challenges of further cytotoxic chemotherapy, he was referred to radiation oncology for consideration of a course of consolidative radiation therapy. The consensus for this approach was based on discussions with the patient and his family. He complained of significant fatigue after the course of chemotherapy and new neuropathy in his hands and feet.

Discussion

Reports have shown that B-cell lymphoma is associated with exposure to the toxic herbicide carcinogen mixture TCDD or AO, which was widely used during the Vietnam conflict of 1961-1974 [1]. Exposure to AO has an increased risk of DLBCL with an OR of 1.26 [4]. TCDD increases cyclooxygenase-2 and deregulates endogenous B-cell leukemia/lymphoma 2 protein (BCL-2) genes [9]. This cascade eventually inhibits cell

apoptosis and allows the malignant B-cells to survive. Other factors driving oncogenesis after AO exposure include immunosuppression, diminished cytotoxic B and T-cell function, altered cytokine levels, dysregulation of hematopoietic stem cells, increased prevalence of monoclonal gammopathy of uncertain significance (MGUS), and apoptotic enhancement of risk gene loci such as BCL-2 and human leucocyte antigens (HLA) [5]. The subtypes of lymphoma associated with AO exposure other than DLBCL include follicular lymphoma (FL), chronic lymphocytic leukemia (CLL), and multiple myeloma (MM). Exposure to AO has increased the risk of FL (OR, 1.71), CLL (OR, 1.61), and MM (OR, 1.58) [5]. Our patient had a 40-pack-year smoking history. There is no data that directly supports the combination of cigarette smoking and AO exposure to a higher incidence of DLBCL, but there is data from preclinical models to suggest TCDD in AO and tobacco carcinogens act in concert to increase malignancy risk, especially in lung cancer models [10].

Although not present in our patient, white veterans exposed to high-TCDD AO levels have an 80% increased risk of MGUS progression to MM [3]. The clinical presentation of DLBCL in patients who have had significant exposure to AO resembles that of other patients with non-Hodgkin lymphoma [6]. Painless lymphadenopathy is the most common presentation. Fever, unintentional weight loss, and fatigue are not infrequent constitutional associations. Drenching night sweats and significant weight loss are usually associated with more aggressive disease. Areas of the lymphoreticular system affected are usually cervical, axillary, and inguinal nodes, and in more advanced cases, the gastrointestinal tract, skin, and bone marrow may be involved. Based on the World Health Organization classification of large B-cell lymphomas, in over 80% of cases, the large B-cell lymphoma not otherwise specified (NOS) is the most common type [11]. There is no data that links AO exposure with a different clinical presentation of B-cell lymphoma compared to those due to other causes, but younger patients and the presence of monoclonal gammopathies have been reported at a higher frequency [7,12].

Our patient was also found to have a prior history of a hepatitis B virus infection, likely acquired after exposure to AO, which probably contributed to his development of DLBCL, as epidemiological studies demonstrate a two times higher risk of developing DLBCL, and it is linked to worse outcomes [13]. However, his IPI score is 2, demonstrating an 80% chance of progression-free survival, likely because treatment started at an early stage. Prior HBV infection, defined as Hepatitis B Surface Antigen (HBsAg) negative or Hepatitis B Core Antibody (HBcAb) positive, also increases DLBCL risk at an odds ratio of 2.4 compared to other B-cell lymphomas [14]. HBV-associated DLBCL usually occurs in younger individuals, unlike our patient. Unfortunately, like our patient, they tend to have more advanced disease, extra-nodal involvement, abnormal liver function, and hypoalbuminemia more commonly than HBV-negative patients [15]. These patients are less likely to respond to chemotherapy and tend to have shorter survival [16]. Our patient underwent six cycles of R-CHOP given his rapidly enlarging jaw mass, skin breakthrough, and complicated follow-up in a remote rural location.

All patients with DLBCL should undergo HBV screening prior to treatment. The American Society of Clinical Oncology (ASCO) recommends testing for HBsAg and HBcAb in all patients getting anti-CD20 monoclonal antibody therapy [17,18]. If either screening test is positive, quantitative HBV DNA by polymerase chain reaction and hepatitis B surface antibody should also be obtained; however, guidelines state that screening should not delay cancer therapy initiation. Our patient had a positive HBcAb and elevated HBsAb. Entecavir is the preferred antiviral agent for prophylaxis in HBsAg-positive patients receiving anti-lymphoma therapy. Serial monitoring of HBV viral load (monthly during treatment and every three months after) is a reasonable alternative prophylaxis but is associated with a 25% reactivation rate in patients on rituximab-based regimens [18]. Tenofovir is an alternative, but lamivudine should not be used due to high resistance rates. Prophylaxis should start one week before chemotherapy and continue throughout the duration of treatment [19]. Antiviral prophylaxis should continue at least 12 months after completion of therapy for standard regimens and 12-18 months for rituximab-based regimens. This is due to the prolonged risk of reactivation, which has been documented to occur well after a year in patients receiving anti-CD20 therapy, necessitating extended duration prophylaxis [20].

Conclusions

When AO exposure is combined with comorbidities, lifestyle, other environmental exposures, and genetic predisposition, the risk of malignancy increases significantly. Evidence demonstrates both independent and combined effects across multiple cancer types. We postulate that our patient's aggressive malignancy was the cumulative aggregation of potential risk factors like cigarette smoking for many years, prior hepatitis B infection, and significant and prolonged exposure to AO.

Risk stratification should incorporate genetic predisposition (where available), metabolic comorbidities (hypertension, type II diabetes mellitus, and obesity), viral hepatitis status, alcohol and tobacco use, and documentation of other exposures known to increase malignancy risk. This clinical case should serve as a reminder for clinicians who routinely care for veterans of this era to utilize a comprehensive, multifactorial approach to risk assessment when evaluating this patient population. Furthermore, despite our patient's good performance status and tolerance of six cycles of R-CHOP, he had residual disease requiring consolidative radiation therapy.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Christopher Tom, Michael E. Lazarus, Michelle Chen, Jacob Klickstein

Acquisition, analysis, or interpretation of data: Christopher Tom, Michael E. Lazarus, Michelle Chen, Jacob Klickstein, Michael Lewis

Drafting of the manuscript: Christopher Tom, Michael E. Lazarus, Michelle Chen, Jacob Klickstein, Michael Lewis

Critical review of the manuscript for important intellectual content: Christopher Tom, Michael E. Lazarus, Michelle Chen, Jacob Klickstein, Michael Lewis

Supervision: Michael E. Lazarus, Michael Lewis

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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