(Fig. 2a,b), strongly positive for plasmocyte markers CD38 (Fig. 2c) and CD138 (Fig. 2d), and negative for CD20, CD3, CD30 and Epstein–Barr virus *in situ* hybridization. ALK expression was positive, with a granular cytoplasmic pattern (Fig. 2e). Ki67 was expressed in 90% to 95% of cells. FISH analysis with a dual-colour break-apart rearrangement probe (CymoGen Dx) showed the typical split signal pattern consistent with a rearrangement involving the ALK gene (Fig. 2f).

The patient received six cycles of a cyclophosphamide, doxorubicin, vincristine and prednisone (CHOP) protocol, associated with prophylactic intrathecal chemotherapy including methotrexate, cytarabine and dexamethasone (MADIT), in the first day of each CHOP cycle. Because of the aggressive nature of the tumour, treatment was complemented with radiation therapy and autologous stem-cell transplantation. The response was complete, with no recurrence during a 2-year follow-up period.

Large B-cell lymphomas are a clinically and biologically heterogeneous group of lymphomas formed by immature cells similar to immunoblasts, encompassing lesions that express mature B-cell markers, such as CD 20, and neoplasms with plasmocytic differentiation designated as plasmablastic lymphomas.[4] LBCL-ALK+ lymphoma is a rare example of the latter type, usually diagnosed in young immunocompetent patients, with a negative profile for Epstein–Barr virus and Human Herpes virus 8, and primarily involving lymph nodes. Since the first description of LBCL-ALK+, around 70 cases with nodal or extranodal involvement have been reported. This number however may be underestimated, as several reports of presumed plasmablastic lymphomas lack a reference to ALK staining. If this specific marker is not performed, the tumour may be easily misdiagnosed, as Colomo and co-workers have already highlighted.[4] This has important prognostic and treatment implications because ALK-positive LBCL tumours are more aggressive than ALK-negative lesions[5] and new therapeutic options acting as ALK inhibitors are being developed with promising results. In the present case, the main challenge was an atypical presentation as a well-circumscribed lesion causing globe indentation and displacement with marked inflammatory-like signs, not commonly seen in lymphoproliferative lesions. Incisional biopsy was an essential step for a correct diagnosis and management. Even without detectable systemic involvement, the diagnosis warranted an aggressive therapy involving chemotherapy, radiotherapy and autologous stem-cell transplantation, with an excellent outcome.

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Efficacy of selective laser trabeculoplasty after canaloplasty

Glaucoma is a family of diseases of multi-factorial aetiology, which typically lead to a slow and progressive degeneration of the optic nerve resulting in blindness. Canaloplasty (CP) is a relatively new technique that attempts to restore the natural trabeculocanalicular flow through dilation of Schlemm's canal and the collector channels.[1] Selective laser trabeculoplasty (SLT) is a non-invasive procedure used to lower the intraocular pressure (IOP) by improving the outflow through the trabecular meshwork, which is thought to act by stimulating the inflammatory cytokines of the trabecular meshwork with laser energy resulting in improved

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outflow.[2] A wide range in the overall decrease of IOP after SLT has been reported in the literature ranging from 3–7 mm Hg (11–40% decrease from baseline).[2–4]

To this date, no studies have looked at the utilization of SLT in patients who have had a CP. Because of the dilation of Schlemm's canal after CP, the distal aqueous outflow from the trabecular meshwork would be more patent and may be more responsive to SLT.

A retrospective chart review of patients who had undergone SLT after CP by two surgeons from January 2010 to August 2014 was performed. Patients were excluded if they had undergone a trabeculectomy or a drainage device prior to SLT. This study received institutional review board approval from the University of Oklahoma with accordance to the Declaration of Helsinki.

The decision for performing SLT was left to the individual surgeon based on the patients IOP and medications. Success of treatment was defined as a greater than 20% drop in IOP at 2–4 weeks or a decrease in the number of medications 3 months after SLT. The patients were then separated into 'responders' and 'non-responders' and compared based on age, ethnicity, sex, medical diagnosis, ocular medical history, ocular surgical history, laterality of eye involved, surgeon, pre-operative IOP, pre-operative visual acuity, pre-operative IOP medications, post-operative pressure, post-operative visual acuity and post-operative IOP medications at (2–4 weeks, 4–6 months, 8–12 months and 18–24 months). Statistical significance was measured using a 2-sample *T*-Test and Fisher's Exact Test.

A total of 19 eyes of 16 patients met the inclusion criteria. Of the 3 patients with both eyes meeting inclusion criteria, all eyes were non-responders. The average time between CP and SLT was 17 months for the non-responders group and 18 months for the responders ($P < 0.81$). The earliest SLT was performed 204 days after CP. Three patients were found to have a greater than 20% response rate to SLT at 2–4 weeks, and two patients were able to stop one of their medications. The follow up rate was 68% at 8–12 months.

Of the 19 eyes, 15 were on a prostaglandin analog prior to SLT (4/5 responders and 11/14 non-responders), which was not statistically significant.[5] Older age was the only statistically significant factor between the two groups ($P < 0.044$), which showed older patients (age 75 +/− 10) to have a more favourable outcome.

Of the greater than 20% drop in IOP responders, two patients required a second medication 2 years after the procedure, and one patient actually reduced his medications by one at 3 years. Of the two patients who were able to decrease their medications, one patient underwent a YAG Goniopuncture 5 months after SLT. No statistically significant difference was found between the two surgeons ($P < 0.63$) or completeness of CP ($P < 0.77$). Notably, none of the responders had previous SLT as compared with 5/14 non-responders ($P < 0.26$).

The effect of SLT was not synergistic with CP and appeared to be independent and less efficacious compared with previously published results.[2–4] The majority of our patient population was on a prostaglandin inhibitor at the time of SLT and had only moderately elevated IOP, both of which had been reported to decrease SLT efficacy.[5] In our patient population, only the patients naïve to SLT appeared to receive a benefit; unfortunately, this was not statistically significant, possibly because of a small patient population. Interestingly, the outcome did not appear to be affected by the type of glaucoma or completeness of CP, also possibly because of our small patient population. The use of SLT in our study was, on average, a year and half after CP, which may be too long a time to utilize the full effect of the procedure.

However, a greater than 20% reduction of IOP 2–4 weeks after SLT in CP patients appears to create a sustainable and beneficial effect, particularly in older individuals with no prior SLT treatments. Future prospective studies with a larger, randomized and a less refractory treatment group would be beneficial to further address this question.

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