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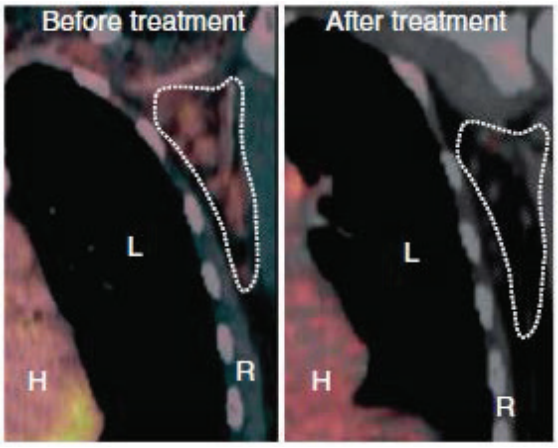
New use of PDGFRbeta inhibitors for treatment of T-cell lymphomas

BACKGROUND

The major clinical challenge in treating **NPM-ALK+ anaplastic large cell lymphomas (ALCL)** is its high rate of relapse. Researchers of the Medical University of Vienna identified high expression of PDGFRß in the NPM-ALK induced ALCL. Pharmaceutical targeting of PDGFRB using a tyrosine kinase inhibitor (TKI) strongly reduced tumor size in SCID-mice xenografted with NPM-ALK mouse tumor cells, and increased the survival of NPM-ALK+ transgenic mice by more than 90%. These data provide evidence for the **use of FDA approved tyrosine kinase inhibitors for the effective treatment of lymphomas and encourage the development of specific clinical trials.**



PET-CT scans before and 10d after initiation of imatinib treatment.
Axillary lymph nodes (delineated by white dotted line) were PET-CT negative after treatment.
L, lung; H, heart; R, rib cage

RESULTS

- Human ALCL patients express high levels of PDGFRB
- PDGFRB is a novel target of AP-1
- Clinical relevance: Complete remission in 2 terminally ill patients who were resistant to other established therapies

ADVANTAGE

Imatinib treatment: less toxic, less side effects, first line therapy

NEXT STEPS

Phase II trial: evaluation of the response rate / efficacy to various doses of TKI administered orally in relapsed/refractory patients with ALCL and other PDGFRß expressing peripheral T-cell lymphomas (PTCLs), to demonstrate clinical proof of concept.

- Mouse xenograft models
- Mouse NPM-ALK transgenic models

FURTHER READING

Nat Med. 2012 Nov;18(11):1699-704;
Front Biosci (Schol Ed). 2015 Jun 1;7:226-35

REFERENCE:
245.09

AVAILABLE FOR:

- development partnership
- license agreement
- patent sale or other

APPLICATIONS:

- Treatment of lymphomas
- Solid tumors

IPR:
US2012252736 granted

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