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RÉSEAU NATIONAL

ANCERS RARES POST-TRANSPLANTATION

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Spectrum of Cancer Risk Among US Solid Organ Transplant Recipients

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Context Solid organ transplant recipients have elevated cancer risk due to immunosuppression and oncogenic viral infections. Because most prior research has concerned kidney recipients, large studies that include recipients of differing organs can inform cancer etiology.

Objective To describe the overall pattern of cancer following solid organ transplantation.

Design, Setting, and Participants Cohort study using linked data on solid organ transplant recipients from the US Scientific Registry of Transplant Recipients (1987-2008) and 13 state and regional cancer registries.

Main Outcome Measures Standardized incidence ratios (SIRs) and excess absolute risks (EARs) assessing relative and absolute cancer risk in transplant recipients compared with the general population.

Results The registry linkages yielded data on 175 732 solid organ transplants (58.4% for kidney, 21.6% for liver, 10.0% for heart, and 4.0% for lung). The overall cancer risk was elevated with 10 656 cases and an incidence of 1375 per 100 000 person-years (SIR, 2.10 [95% CI, 2.06-2.14]; EAR, 719.3 [95% CI, 693.3-745.6] per 100 000 person-years). Risk was increased for 32 different malignancies, some related to known infections (eg, anal cancer, Kaposi sarcoma) and others unrelated (eg, melanoma, thyroid and lip cancers). The most common malignancies with elevated risk were non-Hodgkin lymphoma (n=1504; incidence: 194.0 per 100 000 person-years; SIR, 7.54 [95% CI, 7.17-7.93]; EAR, 168.3 [95% CI, 158.6-178.4] per 100 000 person-years) and cancers of the lung (n=1344; incidence: 173.4 per 100 000 person-years; SIR, 1.97 [95% CI, 1.86-2.08]; EAR, 85.3 [95% CI, 76.2-94.8] per 100 000 person-years), liver (n=930; incidence: 120.0 per 100 000 person-years; SIR, 11.56 [95% CI, 10.83-12.33]; EAR, 109.6 [95% CI, 102.0-117.6] per 100 000 person-years), and kidney (n=752; incidence: 97.0 per 100 000 person-years; SIR, 4.65 [95% CI, 4.32-4.99]; EAR, 76.1 [95% CI, 69.3-83.3] per 100 000 person-years). Lung cancer risk was most elevated in lung recipients (SIR, 6.13 [95% CI, 5.18-7.21]) but also increased among other recipients (kidney: SIR, 1.46 [95% CI, 1.34-1.59]; liver: SIR, 1.95 [95% CI, 1.74-2.19]; and heart: SIR, 2.67 [95% CI, 2.40-2.95]). Liver cancer risk was elevated only among liver recipients (SIR, 43.83 [95% CI, 40.90-46.91]), who manifested exceptional risk in the first 6 months (SIR, 508.97 [95% CI, 474.16-545.66]) and a 2-fold excess risk for 10 to 15 years thereafter (SIR, 2.22 [95% CI, 1.57-3.04]). Among kidney recipients, kidney cancer risk was elevated (SIR, 6.66 [95% CI, 6.12-7.23]) and bimodal in onset time. Kidney cancer risk also was increased in liver recipients (SIR, 1.80 [95% CI, 1.40-2.29]) and heart recipients (SIR, 2.90 [95% CI, 2.32-3.59]).

Conclusion Compared with the general population, recipients of a kidney, liver, heart, or lung transplant have an increased risk for diverse infection-related and unrelated cancers.

JAMA. 2011;306(17):1891-1901 www.jama.com

SOLID ORGAN TRANSPLANTATION provides life-saving therapy for patients with end-stage organ disease. In 2010, a total of 28 664 transplants were performed in the United States, including

Cancers viro-induits

Cancer Site	No. of Cases		SIR (95% CI)	*P* Value	Incidence/100 000 Person-Years[a]		EAR/100 000 Person-Years (95% CI)
	Observed	Expected			Observed	Expected	
Non-Hodgkin lymphoma	1504	199.4	7.54 (7.17 to 7.93)	<.001	194.0	25.7	168.3 (158.6 to 178.4)
Nodal	831	136.6	6.08 (5.68 to 6.51)	<.001	107.2	17.6	89.6 (82.4 to 97.1)
Extranodal	673	62.8	10.72 (9.93 to 11.56)	<.001	86.8	8.1	78.7 (72.3 to 85.5)
Liver	930	80.5	11.56 (10.83 to 12.33)	<.001	120.0	10.4	109.6 (102.0 to 117.6)
Stomach	152	90.9	1.67 (1.42 to 1.96)	<.001	19.6	11.7	7.9 (4.9 to 11.3)
Kaposi sarcoma	120	2.0	61.46 (50.95 to 73.49)	<.001	15.5	0.3	15.2 (12.6 to 18.3)
Oropharynx including tonsil	106	52.8	2.01 (1.64 to 2.43)	<.001	13.7	6.8	6.9 (4.4 to 9.7)
Anus	90	15.4	5.84 (4.70 to 7.18)	<.001	11.6	2.0	9.6 (7.3 to 12.3)
Hodgkin lymphoma	85	23.7	3.58 (2.86 to 4.43)	<.001	11.0	3.1	7.9 (5.7 to 10.5)
Vulva	58	7.6	7.60 (5.77 to 9.83)	<.001	7.5	1.0	6.5 (4.7 to 8.7)
Cervix	45	43.6	1.03 (0.75 to 1.38)	.88	5.8	5.6	0.2 (–1.4 to 2.1)
Penis	22	5.3	4.13 (2.59 to 6.26)	<.001	2.8	0.7	2.2 (1.1 to 3.6)
Nasopharynx	8	8.3	0.96 (0.42 to 1.90)	>.99	1.0	1.1	0 (–0.6 to 1.0)
Vagina	7	3.0	2.35 (0.94 to 4.84)	.07	0.9	0.4	0.5 (0 to 1.5)
Total[b]	10 656	5080.6	2.10 (2.06 to 2.14)	<.001	1374.7	655.4	719.3 (693.3 to 745.6)

Abbreviations: EAR, excess absolute risk; SIR, standardized incidence ratio.

[a] Includes invasive cancers arising during 775 147 person-years. Incidence is presented for the entire cohort, but can be calculated separately for males or females for sex-specific malignancies based on follow-up of 465 521 person-years in males and 309 626 person-years in females. Cancer types are listed in order of decreasing frequency.

[b] Includes non–infection-related malignancies presented in Table 3.

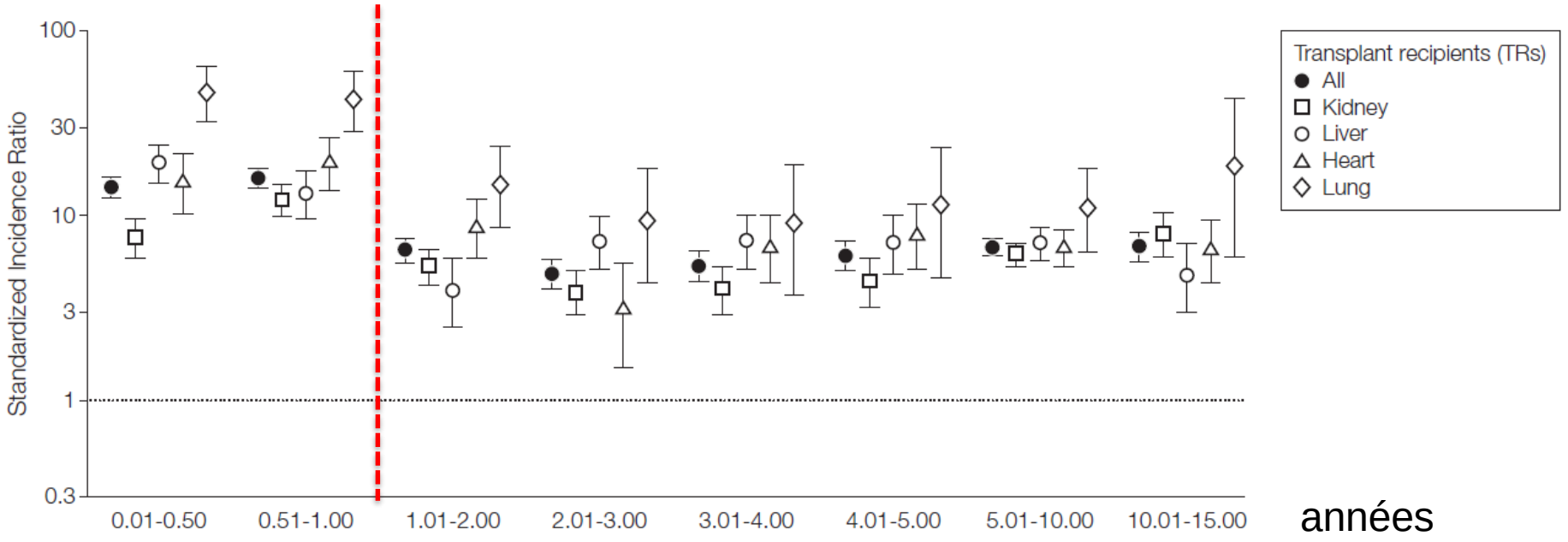
Engels *JAMA* 2011

Standardized Incidence Ration (95% CI)

Transplanted organ	Cancer Site: Non-Hodgkin Lymphoma	Lung Cancer	Liver Cancer	Kidney Cancer
Kidney	6.05 (5.59-6.54)	1.46 (1.34-1.59)	1.08 (0.80-1.43)	6.66 (6.12-7.23)
Liver	7.77 (6.99-8.61)	1.95 (1.74-2.19)	43.83 (40.90-46.91)	1.80 (1.40-2.29)
Heart	7.79 (6.89-8.79)	2.67 (2.40-2.95)	1.02 (0.54-1.74)	2.90 (2.32-3.59)
Lung	18.73 (15.59-22.32)	6.13 (5.18-7.21)	2.04 (0.56-5.22)	1.49 (0.64-2.94)

Engels *JAMA* 2011

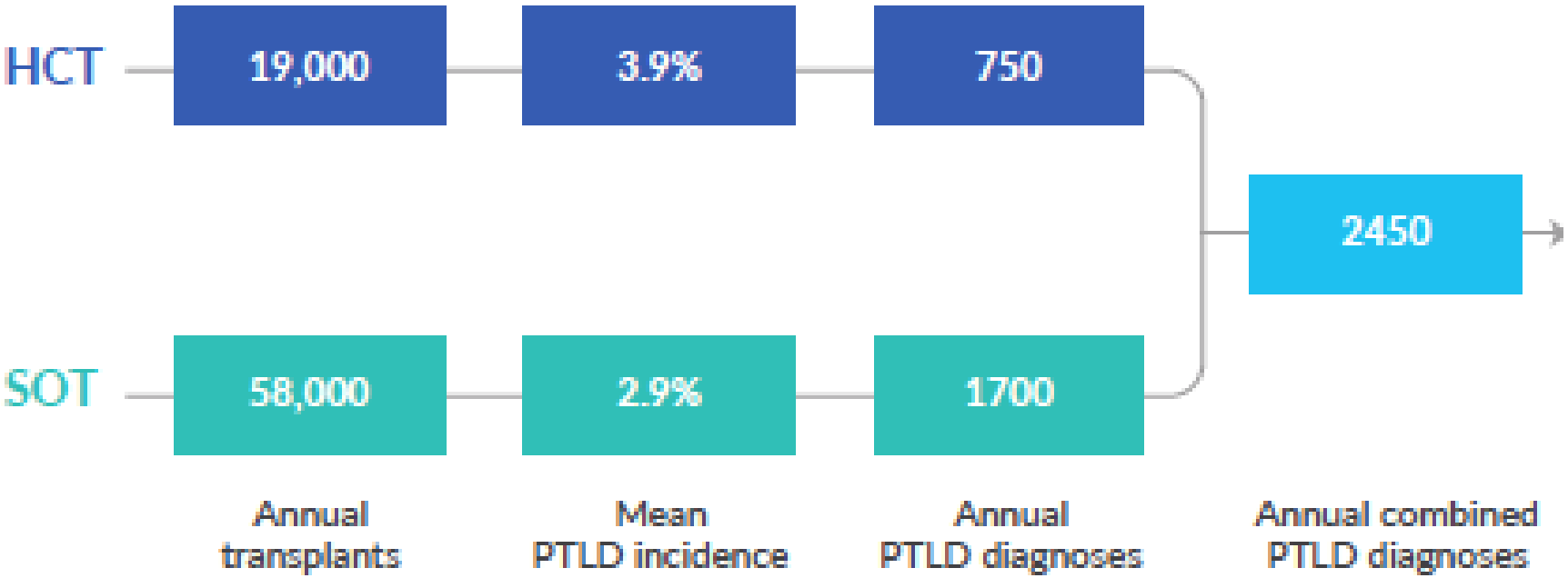
Evolution du risque après greffe



No. of observed cancer events								
All TRs	231	246	179	118	115	113	379	114
Kidney TRs	70	107	83	53	47	44	171	55
Liver TRs	71	45	24	39	35	30	97	23
Heart TRs	30	38	32	11	23	25	77	28
Lung TRs	35	29	17	9	7	7	16	5

Engels *JAMA* 2011

Incidence (Etats-Unis + Europe « 5 »)



126,671 transplant procedures annually worldwide[25]

↑ 5.8% over 1 year[24]

Un traitement spécifique

VOLUME 35 · NUMBER 5 · FEBRUARY 10, 2017

JOURNAL OF CLINICAL ONCOLOGY

ORIGINAL REPORT

Response to Rituximab Induction Is a Predictive Marker in B-Cell Post-Transplant Lymphoproliferative Disorder and Allows Successful Stratification Into Rituximab or R-CHOP Consolidation in an International, Prospective, Multicenter Phase II Trial

Ralf U. Trappe, Daan Dierickx, Heiner Zimmermann, Franck Morschhauser, Peter Mollee, Jan M. Zaucha, Martin H. Dreyling, Ulrich Dührsen, Petra Reinke, Gregor Verhoef, Marion Subklewe, Andreas Hüttmann, Thomas Tousseyn, Gilles Salles, Volker Kliem, Ingeborg A. Hauser, Corrado Tarella, Eric Van Den Neste, Olivier Gheysens, Ioannis Anagnostopoulos, Veronique Leblond, Hanno Riess, and Sylvain Choquet

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ANCERS RARES POST-TRANSPLANTATION

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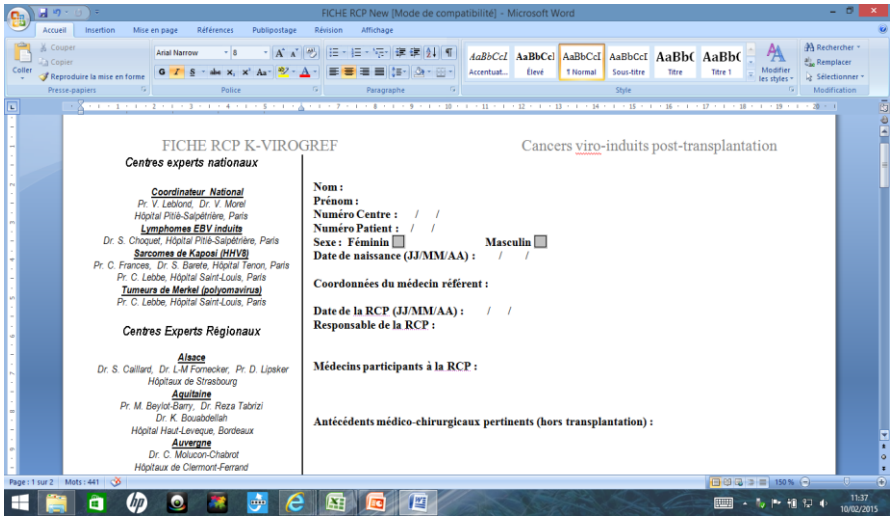
Organisation d’une RCP nationale

R.C.P Nationale

Coordinatrice : Pr V. LEBLOND

PLANNING DES RCP 2018 :

18 / 01	15 / 02	15 / 03	05 / 04	17 / 05	07 / 06	05 /07	13 / 09	18 / 10	08 / 11	20 / 12


FICHE RCP K-VIROGREF

Centres experts nationaux

Coordinateur National
Pr. V. Leblond, Dr. V. Morel
Hôpital Pitié-Salpétrière, Paris

Lymphomes EBV induits
Dr. S. Choquet, Hôpital Pitié-Salpétrière, Paris

Sarcomes de Kaposi (HHV8)
Pr. C. Frances, Dr. S. Barete, Hôpital Tenon, Paris
Pr. C. Lebbe, Hôpital Saint-Louis, Paris

Tumeurs de Merkel (polyomavirus)
Pr. C. Lebbe, Hôpital Saint-Louis, Paris

Centres Experts Régionaux

Alsace
Dr. S. Caillard, Dr. L-M Fornecker, Pr. D. Lipaker
Hôpitaux de Strasbourg

Aquitaine
Pr. M. Beylot-Barry, Dr. Reza Tabrizi
Dr. K. Bouabdellah
Hôpital Haut-Leveque, Bordeaux

Auvergne
Dr. C. Molucon-Chabrot
Hôpitaux de Clermont-Ferrand

Cancers viro-induits post-transplantation

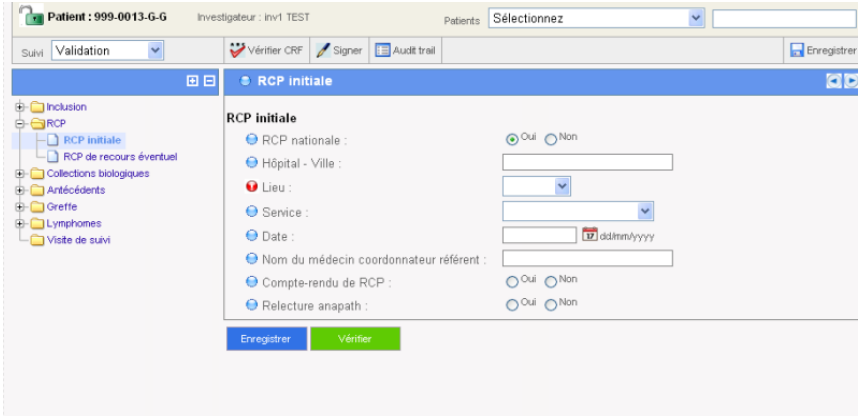
Nom :
Prénom :
Numéro Centre : / /
Numéro Patient : / /
Sexe : Féminin Masculin
Date de naissance (JJ/MM/AA) : / /

Coordonnées du médecin référent :

Date de la RCP (JJ/MM/AA) : / /
Responsable de la RCP :

Médecins participants à la RCP :

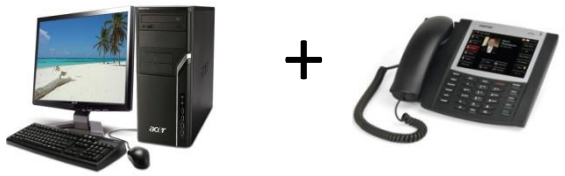
Antécédents médico-chirurgicaux pertinents (hors transplantation) :

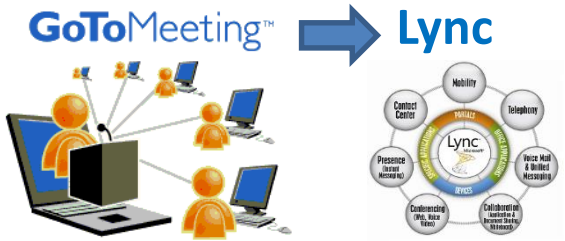


2 eme jeudi du mois à partir de 13h00

Email : sylvain.choquet@aphp.fr

Email : noureddine.balegroune@aphp.fr
Tél : 01 42 16 17 99





R.C.P Nationale

211 FICHES RCP

Centres	Lymphome	Kaposi	Merkel	TOTAL
IDF	65	20	1	86
Province	114	7	4	125
TOTAL	179	27	5	211

Coordinatrice : Pr V. LEBLOND

Inclusions patients (01/2015 au 11/2018)

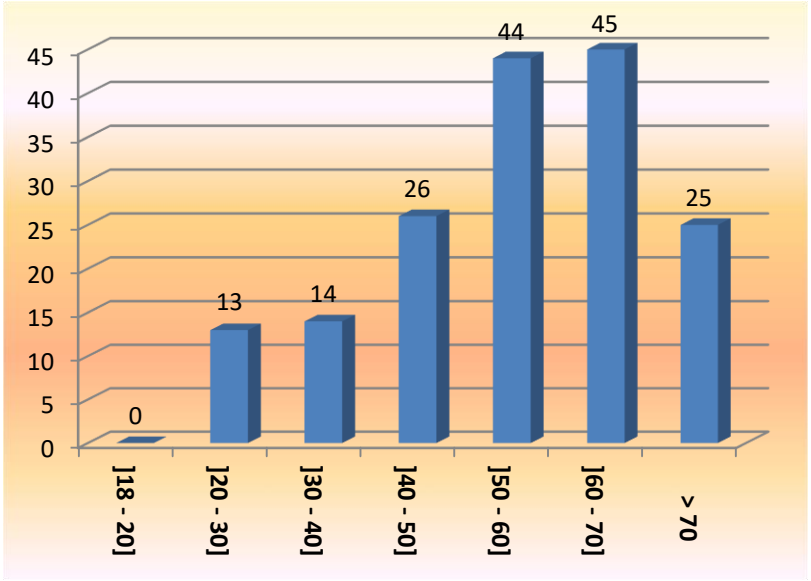
Centre	Nombre d'inclus	Pathologie
IDF (Hémato/Dermato)	80	**51** lymphomes / **28** Kaposi / **1** Merkel
Besançon (Hémato)	5	lymphome
Rouen (Hémato)	4	lymphome
Reims (Hémato)	4	lymphome
Nantes (Néphro)	7	**6** Lymphome, **1** Merkel
Montpellier (Hémato)	6	lymphome
Montpellier (dermato)	3	2 Kaposi / 1 Merkel
Lille (Hépato)	3	lymphome
Amiens (Dermato)	4	3 Kaposi / 1 Merkel
Amiens (Hémato)	1	lymphome
Clermont F (Hémato)	3	lymphome
Strasbourg (Hémato)	8	lymphome
Strasbourg (Néphro)	3	**1** lymphome / **2** Merkel
Bordeaux Bergonié (Hémato)	7	lymphome
Bordeaux (Hémato)	3	lymphome
Rennes (Néphro)	4	**2** lymphome / **2** Kaposi
Nancy (Hémato)	1	lymphome
Lyon (Hémato)	7	lymphome
Tours (Hémato)	5	lymphome
Toulouse (Néphro)	5	lymphome
Limoges (Hémato)	2	lymphome
Chalon (Hémato)	1	lymphome
Caen (Hémato)	1	lymphome
Total	**167**	**126** lymphomes / **35** Kaposi / **6** Merkel

Ile de France
(6 centres)

Province
(19 centres)

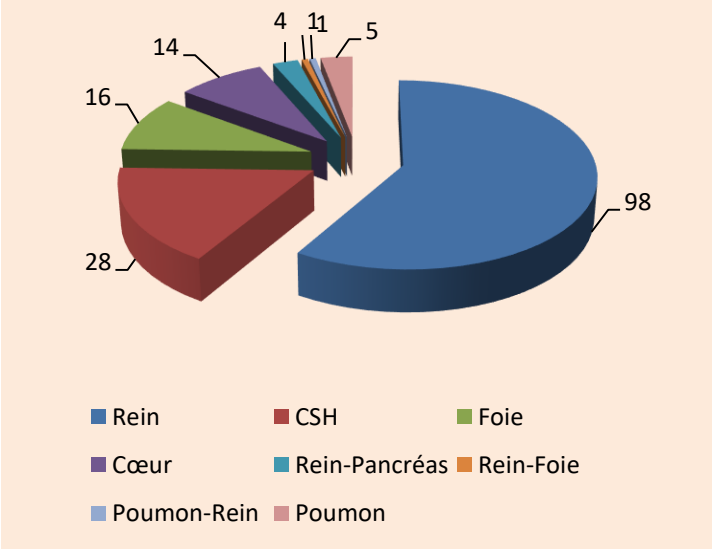
Inclusions patients (au 11/2018)

Total	Sexe	Age	Nbre	Nbre
N = 167	109 ♂ (65 %)	[18 - 20]	0	0
		]20 - 30]	13	6 ♀
				7 ♂
		]30 - 40]	14	9 ♀
				5 ♂
		]40 - 50]	26	8 ♀
				18 ♂
	58 ♀ (35 %)	]50 - 60]	44	13 ♀
				31 ♂
		]60 - 70]	45	13 ♀
				32 ♂
		> 70	25	9 ♀
				16 ♂



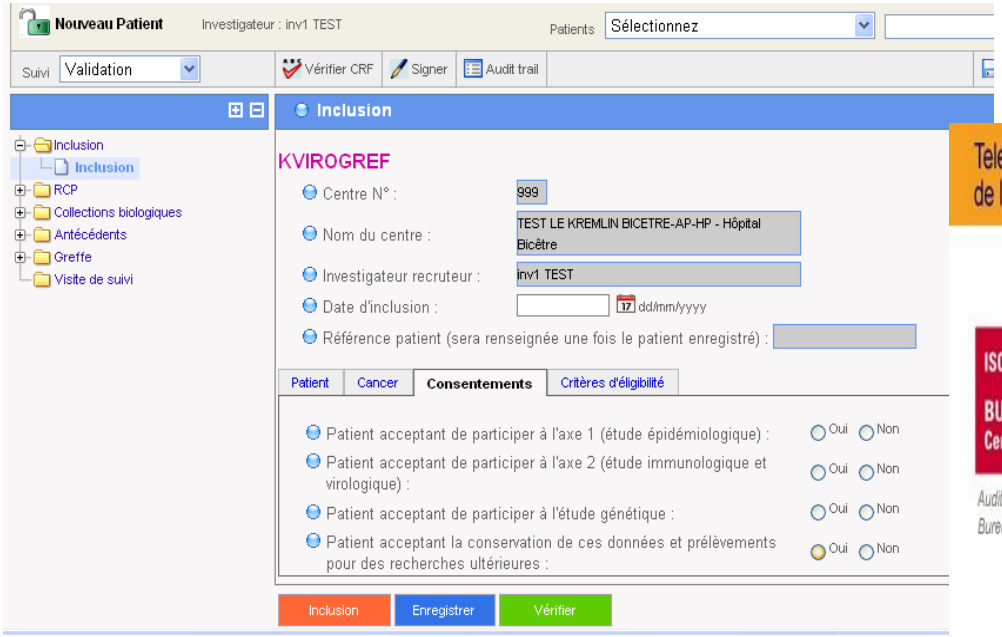
Inclusions patients

Greffe	Lymphome	Kaposi	Merkel	Total
Rein	63	29	6	98
CSH	28	-	-	28
Foie	16	-	-	16
Cœur	8	6	-	14
Rein-Pancréas	4	-	-	4
Poumon	5	-	-	5
Rein-Foie	1	-	-	1
Poumon-Rein	1	-	-	1
Total	126	35	6	167



Constitution d’une base de données

Base de données



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Telemedicine Technologies poursuit sa politique d'investissement dans les domaines de la performance et de la qualité au profit de la satisfaction client

ISO 9001
BUREAU VERITAS
Certification

Audit de certification réalisé par Bureau Veritas Certification (Janvier 2015)

Certification
ISO 9001 : 2008

Domaine d'activité
Développement et mise à disposition de logiciels collaboratifs pour la recherche clinique et épidémiologique

Certificat n°FR020752-1

Validation des systèmes d'information suivant la méthodologie GAMP 5 (Good Automated Manufacturing Practices)

CleanWEB
Solution globale de gestion électronique d'essais cliniques
EDC - IWRS - CTMS - eTMF - CDMS - ePRO

135 dossiers patients complétés (88 %)

102 (hémato) 76 %

33 (dermato) 24 %

Constitution d’une biothèque

Prélèvements sanguins

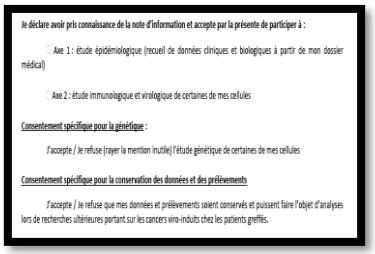
Centralisation des prélèvements au département d'immunologie du Pr B. AUTRAN à la Pitié

Signature consentement

Prélèvement patient

Expédition 24h à T° ambiante

Centralisation prélèvement



Je déclare avoir pris connaissance de la note d'information et accepte par la présente de participer à :

☐ Axe 1 : étude épidémiologique (recueil de données cliniques et biologiques à partir de mon dossier médical)

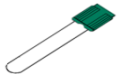
☐ Axe 2 : étude immunologique et virologique de certaines de mes cellules

Consentement spécifique pour la génétique :

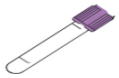
J'accepte / Je refuse (rayer la mention inutile) l'étude génétique de certaines de mes cellules

Consentement spécifique pour la conservation des données et des prélèvements

J'accepte / Je refuse que mes données et prélèvements soient conservés et puissent faire l'objet d'analyses lors de recherches ultérieures portant sur les cancers viro-induits chez les patients greffés.



6 x 7ml
(hep lithium)



1 x 7ml
(EDTA)

TMS

TSE
Express Médical

154 prélèvements sanguins reçus

79 Hémato(51 %)

75 Dermato (49%)

Information

Site Internet K-VIROGREF

Opérationnel depuis le mois d'avril 2016 www.k-virogref.org

Information aux patients et aux professionnels de la santé

Livret d'information aux patients / familles

GUIDE D'INFORMATION À L'USAGE DES PATIENTS ET DE LEURS FAMILLES

Réseau National K-VIROGREF

Prise en charge des cancers viro-induits post transplantation d'organes solides ou de cellules souches hématopoïétiques

Site internet K-VIROGREF

www.k-virogref.org

Lien avec les autres réseaux

- *Réseau CancerVIH*
- *Réseau LOC*
- *Réseau CLIP*

Site Internet K-VIROGREF

Espace professionnel

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- Recommandation de prise en charge
- Newsletter
- Les documents de l'étude

Les documents de l'étude

1 - Le protocole de l'étude

2 - Le résumé du protocole

3 - La notice d'information et de consentement : 3 types selon l'état de santé du patient à l'inclusion

- Formulaire consentement
- Formulaire de consentement proche
- Formulaire de consentement poursuite

4 - Ordonnance de prélèvement

5 - Fiche Demande de transport

6 - Etiquettes anonymat

7 - Procédure gestion des prélèvements

8 - Procédure inclusion patient

9 - N° centre anonymat

10 - Fiche RCP

11 - Planning RCP 2016

12 - Formulaire accord Extraction

13 - Documents réglementaires :

- Avis CPP
- Avis CNIL

Lien avec associations

Lien avec les associations patients

Recommandations


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• Quels déficits immunitaires héréditaires faut-il rechercher lors du diagnostic de lymphome chez un adulte jeune ? – F. Touzot
• Lymphoprolifératons après transplantation S. Choquet
• Prise en charge des lymphoprolifératons du VIH J. Reure, N. Mounier
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Inhibiteurs de tyrosine kinase anti-FLT3 : mécanismes d'action et résultats thérapeutiques
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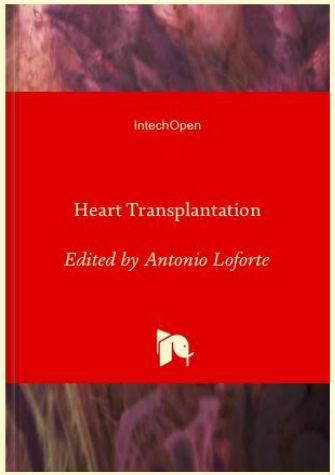

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PRIMER

Post-transplant lymphoproliferative disorders

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Abstract | Post-transplant lymphoproliferative disorders (PTLDs) are a group of conditions that involve uncontrolled proliferation of lymphoid cells as a consequence of extrinsic immunosuppression after organ or haematopoietic stem cell transplant. PTLDs show some similarities to classic lymphomas in the non-immunosuppressed general population. The oncogenic Epstein–Barr virus (EBV) is a key pathogenic driver in many early-onset cases, through multiple mechanisms. The incidence of PTLD varies with the type of transplant; a clear distinction should therefore be made between the conditions after solid organ transplant and after haematopoietic stem cell transplant. Recipient EBV seronegativity and the intensity of immunosuppression are among key risk factors. Symptoms and signs depend on the localization of the lymphoid masses. Diagnosis requires histopathology, although imaging techniques can provide additional supportive evidence. Pre-emptive intervention based on monitoring EBV levels in blood has emerged as the preferred strategy for PTLD prevention. Treatment of established disease includes reduction of immunosuppression and/or administration of rituximab (a B cell-specific antibody against CD20), chemotherapy and EBV-specific cytotoxic T cells. Despite these strategies, the mortality and morbidity remains considerable. Patient outcome is influenced by the severity of presentation, treatment-related complications and risk of allograft loss. New innovative treatment options hold promise for changing the outlook in the future.

PRIMEVIEW

POST-TRANSPLANT LYMPHOPROLIFERATIVE DISORDERS

nature REVIEWS DISEASE PRIMERS

For the Primer, visit doi:10.1038/nrdp.2015.88

Post-transplant lymphoproliferative disorders (PTLDs) are a group of lymphoma-like conditions characterized by an uncontrolled proliferation of lymphoid cells as a consequence of therapeutic immunosuppression following a transplant. PTLDs can develop after a solid organ transplant (SOT-PTLD) or haematopoietic stem cell transplant (HSCT-PTLD); these two transplant settings differ with regard to disease course, prevalence and management.

EPIDEMIOLOGY

The incidence of PTLD depends on the type of transplant. SOT-PTLDs occur in ~10% of SOT recipients; patients who receive intestinal and multi-organ transplants have the highest risk, followed by lung, heart, liver and kidney transplants. Recipients of a HSCT have the lowest risk, with HSCT-PTLD occurring in <2% of patients. Most HSCT-PTLDs are of donor origin, whereas most SOT-PTLDs arise from recipient-derived cells. PTLDs commonly arise in the lymph nodes, gastrointestinal tract, liver, central nervous system and lungs. Incidence of PTLDs, especially early-onset PTLD, is considerably higher in children than adults, whereas the risk of developing late-onset PTLD rises from 60 years of age. The Epstein–Barr virus (EBV) is a key pathogenic driver of many early-onset PTLDs, but other viruses (including cytomegalovirus, hepatitis C virus and herpes virus 8) might also have a role.

EBV-seronegative patients who receive an EBV-positive transplant have a greater than 12-fold increased risk of developing PTLD compared with recipients who are EBV-positive before transplantation

MECHANISMS

PTLDs show some similarities with classic lymphomas in the general population, but only occur in immunosuppressed individuals following transplantation

EBV drives lymphoma development in 50–80% of PTLDs, especially in early-onset disease

The aetiological trigger for EBV-negative PTLDs remains unknown

An impaired immune response, especially the T cell response, can lead to the reactivation of latent EBV or a hampered response to a new infection, leading to hyperproliferation of infected B cells

PREVENTION

EBV viral load in the blood of patients with early-onset PTLD is higher than in transplant recipients who do not develop PTLD, and detectable EBV precedes the development of clinical symptoms. This finding has led to the 'pre-emptive' prevention strategy, which combines viral load measurement with interventions that might lower the risk of developing PTLD, such as reduction of the level of immunosuppression. However, standardization of the viral load measurement and monitoring over time (including at baseline) is required.

DIAGNOSIS

The timing of SOT-PTLD diagnosis follows a bimodal distribution, with a peak in the first 2 years (early onset) and a second peak between 5 and 10 years (late onset) after transplantation. Most HSCT-PTLDs occur weeks to months after transplantation. Symptoms depend on the location of the lymphoid mass. The gold standard for PTLD diagnosis is histopathological examination of biopsy specimens and imaging (FDG-PET/CT). The WHO classification — developed in 2008 — is mainly based on histological features.

PTLD is a heterogeneous condition. Lymphomas arise mainly from B lymphocytes, but originate from T cells or natural killer cells in a minority of patients, can be monomorphic or polymorphic, and monoclonal or polyclonal.

MANAGEMENT

First-line treatment for SOT-PTLDs involves reduction of immunosuppression, which will lead to a complete regression of the PTLD in ~10% of patients within weeks, but increases the risk of graft rejection and graft-versus-host disease. If the response is incomplete, patients can be treated with rituximab (a B cell-specific antibody against CD20), followed, if necessary, by chemotherapy or a combination of chemotherapy and rituximab. In HSCT-PTLD, *ex vivo*-generated cytotoxic T cells or pre-emptive rituximab are additional options. Antiviral therapy, radiotherapy, surgery and adoptive immunotherapy can also be used in specific cases.

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Niveaux d'évidence
Grades de recommandation

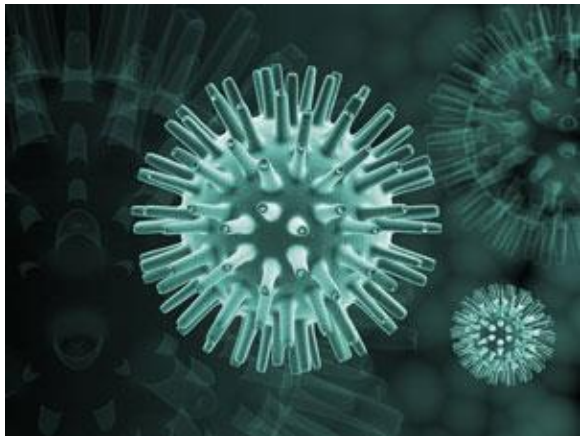
Levels of evidence

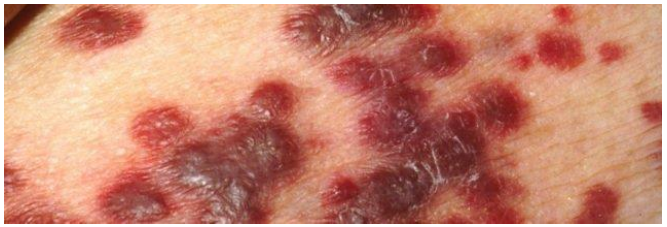
I	Evidence from at least one large randomised, controlled trial of good methodological quality (low potential for bias) or meta-analyses of well-conducted randomised trials without heterogeneity
II	Small randomised trials or large randomised trials with a suspicion of bias (lower methodological quality) or meta-analyses of such trials or of trials demonstrated heterogeneity
III	Prospective cohort studies
IV	Retrospective cohort studies or case–control studies
V	Studies without control group, case reports, expert opinions

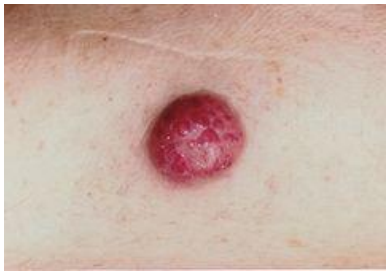
Grades of recommendation

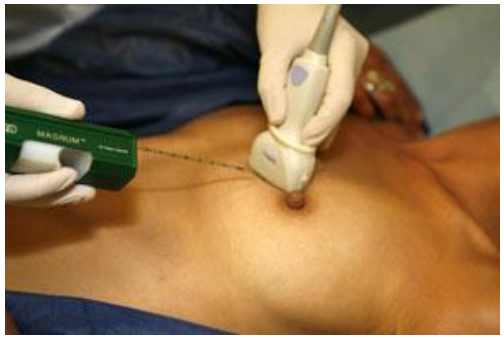
A	Strong evidence for efficacy with a substantial clinical benefit, strongly recommended
B	Strong or moderate evidence for efficacy but with a limited clinical benefit, generally recommended
C	Insufficient evidence for efficacy or benefit does not outweigh the risk or the disadvantages (adverse events, costs, . . .), optional
D	Moderate evidence against efficacy or for adverse outcome, generally not recommended
E	Strong evidence against efficacy or for adverse outcome, never recommended

Groupes référents









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Integrated Discovery of new Immuno-Molecular Actionable Biomarkers for Tumors with immune-suppressed environment

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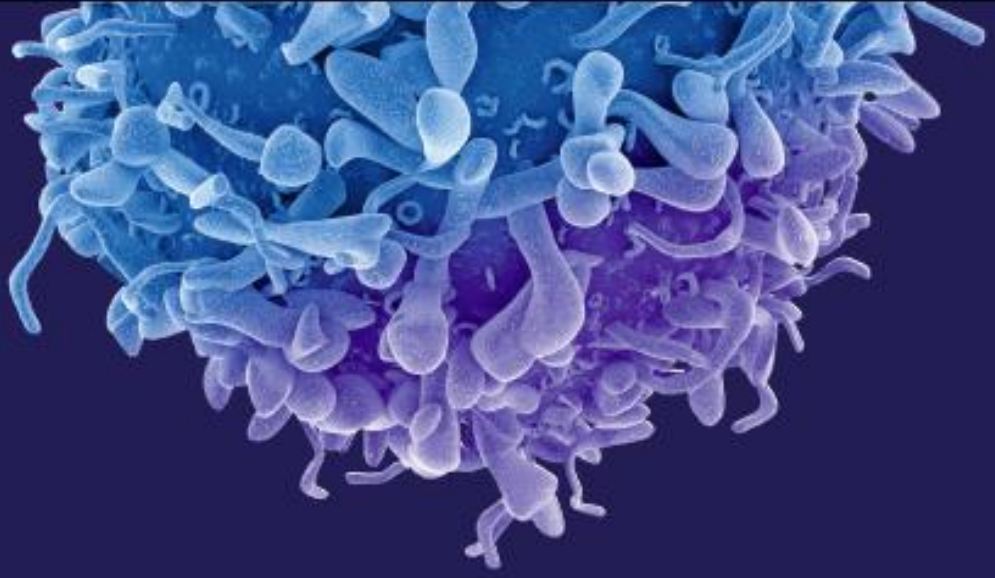

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