SMARCA4 dans le cadre de ces carcinomes semblent plutôt être des mutations « passengers » plutôt que des mutations « drivers » (47).

D'un point de vue clinique, l'âge médian est de 50 ans, plus jeune que les autres CBNPC. Le tabac est un des facteurs de risque retrouvé. La perte de SMARCA4 est retrouvée dans les différents sous-types histologiques (carcinomes épidermoïdes, adénocarcinomes, carcinomes à grandes cellules, carcinomes pléomorphes…) et d'autant plus fréquente que le degré de différenciation est faible. Pour les adénocarcinomes SMARCA4-déficients, le TTF1 est le plus souvent négatif. Le diagnostic de la déficience se fait par IHC ou biologie moléculaire. Sur le plan moléculaire, on ne retrouve a priori pas de mutation d'*EGFR* ou de réarrangement d'*ALK* ou de *ROS1*. Des mutations de *KRAS* en revanche peuvent être associées.

2.2 Principes de traitement

Ces tumeurs ne font actuellement pas l'objet d'un traitement spécifique consensuel. Elles doivent être considérée comme des CBNPC.

Le traitement local, quand il est possible, doit être proposé.

Concernant le traitement systémique, la chimiothérapie à base de platine reste le traitement de référence. La place de l'immunothérapie reste à définir mais plusieurs cas publiés rapportent une efficacité de l'immunothérapie chez ces patients (48). S'agissant de tumeurs le plus souvent indifférenciées et en l'absence de recommandations claires, le schéma carboplatine-paclitaxel-pembrolizumab reste probablement à privilégier.

Des inhibiteurs d'EZH2 sont également en cours d'évaluation.

Il faut bien évidemment si possible privilégier les inclusions dans les essais thérapeutiques.

OPTION : le schéma de première ligne des tumeurs *SMARCA4* déficientes peut être carboplatine-paclitaxel et pembrolizumab (avis d'experts).

3. Carcinomes NUT

3.1 Présentation clinique et diagnostic histologique

Il s'agit de tumeurs se présentant le plus souvent à un stade avancé, sous la forme de carcinomes indifférenciés avec une atteinte médiastinale centrale chez des patients dont l'âge moyen est plus jeune que l'âge des patients atteints de CBNPC. Aucun facteur de risque n'a été clairement identifié, notamment le tabac ne semble pas être associé. Probablement sous-diagnostiqué et de diagnostic anatomo-pathologique difficile, ces tumeurs peuvent exprimer des marqueurs de carcinome épidermoïde comme p40, ainsi que des marqueurs neuro-endocrines ce qui peut être faire porter à tort d'autres diagnostics. De plus, étant souvent diagnostiqués à des stades avancés, le diagnostic est fréquemment porté sur petits prélèvements sur lesquels la morphologie est plus difficilement analysable.

Ce carcinome se caractérise par une fusion impliquant le gène *NUTM1* (NUclear protein in Testis Midline carcinoma family member 1) avec un partenaire de la famille *BRD* (bromodomaincontaining protein). Cette fusion va être impliquée dans le blocage de la différenciation et l'activation de la prolifération cellulaire via *MYC* (activation du promoteur) et via l'inhibition de l'acétylation des histones. En immunohistochimie, l'anticorps C52B1 est très spécifique. Selon l'OMS, le diagnostic peut être porté sur la seule base d'une immunohistochimie anti-NUT positive avec un marquage nucléaire granuleux caractéristique. Néanmoins, compte-tenu de la rareté de ces tumeurs, il peut être intéressant de mieux caractériser le type de fusion et le partenaire notamment par RNAseq, le type de partenaire de fusion pouvant avoir un intérêt pronostique. De plus, ces tumeurs expriment BRG1, ce qui permet d'exclure un diagnostic alternatif de carcinome BRG1 / SMARCA4 déficient (49). En effet, Ces fusions sont retrouvées

dans différents types tumoraux incluant les tumeurs hématologiques. Parmi ces tumeurs, l'origine thoracique initiale et les fusions impliquant BRD4 ont été identifiées comme étant de très mauvais pronostic avec des survies globales inférieures à 6 mois (50).

3.2 Principes de traitement

Le traitement local, quand il est possible, doit être proposé. Il n'y a pas de recommandation précise quant au traitement systémique optimal. La chimiothérapie à base de platine reste le schéma de référence. La place de l'immunothérapie n'a pas été évaluée (51). S'agissant de tumeurs le plus souvent indifférenciées et en l'absence de recommandations claires, le schéma carboplatine-paclitaxel-pembrolizumab reste probablement à privilégier.

En revanche, des inhibiteurs sont actuellement en cours de développement.

Premièrement, des inhibiteurs de BET (protéines à Bromodomain and ExtraTerminal domain incluant BRD2, BRD3, BRD4 et BRDT). Le molibresib (GSK525762), le birabresib ((MK-8628/OTX015) et le RO6870810 sont trois inhibiteurs de BET évalués dans des études de phase I/II avec des taux de réponses allant de 11 à 33% chez les patients avec des carcinomes NUT. Le profil de toxicité est essentiellement hématologique avec des thrombopénies (birabresib) et digestifs (nausées, vomissements, diarrhées) (52–54).

Deuxièmement, des inhibiteurs de la désacétylation des histones qui vont bloquer la prolifération et activer la différenciation cellulaire, développé en association avec des inhibiteurs de PI3K (55).

Il faut bien évidemment, si possible, privilégier les inclusions dans les essais thérapeutiques.

TRAITEMENT

1. Stades cliniques IA à IIIA résécables, patient opérable, *EGFR* WT.

1.1. Chirurgie

Une chirurgie d'exérèse complète anatomique, comportant un curage ganglionnaire complet (2,56) est recommandée.

Pour les stades cIB à IIB, la lobectomie reste le standard. Pour les stades cIA, un essai randomisé contrôlé Japonais de non-infériorité, a montré que la segmentectomie était non-inférieure à la lobectomie en terme de survie globale (survie à 5 ans de 94,3% [IC95% 92,1-96,0] pour le groupe segmentectomie (N=552), et 91,1% [IC95% 88,4-93,2] pour le groupe lobectomie (N=554)) (57). Bien qu'il s'agisse d'un essai de non-infériorité, les auteurs rapportent également une analyse de supériorité avec un hazard-ratio de 0,663 [IC95% 0,474-0,927 ; p pour la supériorité de 0.0082]. Cet effet était constant dans tous les sous-groupes de patients. Bien que le taux d'absence de récidive à 5 ans soit identique dans les deux groupes, le groupe « segmentectomie » présentait plus de récidive locale que le groupe « lobectomie » (10,5% vs. 5,4% respectivement ; p=0.0018). Il n'y avait pas de différence cliniquement pertinente en terme de rententissement sur la fonction respiratoire à 1 an (différence sur le VEMS de 3.5% entre les deux groupes). Les crières d'inclusion de l'essai étaient les patients :

- agés de 20 à 85 ans,
- PS 0-1,
- et avec une tumeur de ≤2cm de diamètre, ET localisé dans le tiers externe du parenchyme (hors lobe moyen) ET avec un ratio consolidation/tumeur >0.5. Ce rapport est défini comme la taille de la lésion consolidée (solide) divisée par la taille totale de la tumeur considérée(58).
- La segmentectomie ne peut être considérée que si les marges attendues sont convenables et que si l'examen extemporané d'un curage ganglionnaire intersegmentaire douteux (inflammatoire) est négatif.

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