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Effectiveness and tolerability of the bictegravir/emtricitabine/tenofovir alafenamide regimen in a cohort of HIV-1 infected treatment experienced adult patients: an observational retrospective single-centre study

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BACKGROUND

Integrase strand-transfer inhibitors (INSTIs) are effective and well-tolerated. Bictegravir (BIC) is also associated with low levels of combined Anti-Retroviral Treatment /cART) resistance and decreased markers of inflammation.

We aimed to evaluate the effects of switching to bictegravir/emtricitabine/tenofovir alafenamide (BIC/FTC/TAF) from different regimens, especially comparing boosted and un-boosted regimens.

MATERIALS AND METHODS

We collected data about lipid metabolism, HIV infection, HIV-RNA plasma viral load, weight, creatinine at four time-points (baseline, 6,12 and 18 months after switch) of experienced people living with HIV (EPLWH) switching from July 1st, 2019, to a fixed-dose single-tablet regimen of BIC/FTC/TAF

Categorical variables are expressed as count (percentages), while continuous variables as mean ± SD when normally distributed or median (IQR) when non-normally distributed.

t-test for paired data and ANOVA test were applied to find any statistically significant difference between normally distributed variables. Wilcoxon test was applied to find any statistically significant difference over time among non-normally distributed variables.

Statistical significance level was set at a p value < 0.05, confidence interval (CI) was set at 95%.

Statistical analysis was performed with SPSS 28.0 for MacOS. Graphs were designed with Graphpad Prism 9.0.

RESULTS

90 EPLWH, 77.8% male, median age 45.8 years (IQR 37.6-56.2), switching from 15 different regimens, were included.

Seventy-two (80.0%) came from an INSTI-based regimen, 61.1% from elvitegravir/cobicistat/emtricitabine/tenofovir alafenamide (EVG/c/FTC/TAF), 10 (11.1%) from a protease inhibitor-based regimen, and 8 (8.9%) from a non-nucleoside reverse-transcriptase inhibitor-based (NNRTI-based) regimen.

Globally, 66 (73.3%) came from a boosted regimen.

Figure 1 shows the distribution of regimens before the switch to BIC/FTC/TAF

Median time on cART before switching to BIC/FTC/TAF was 93.5 months (IQR 42.0 - 211.3), while median time on previous regimen was 24.3 months (IQR 15.0 - 30.1).

We did not find any statistically significant difference between durability of the different anchor drug classes (*p* = 0.186)

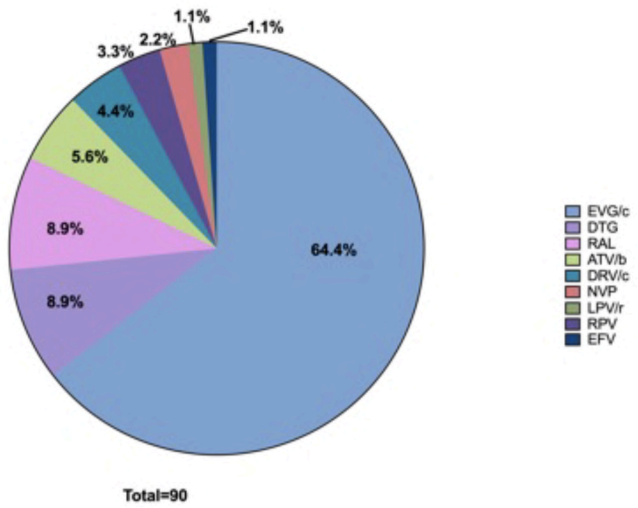

Figure 1. This figure summarizes the percentages of anchor drugs the patients included in the study were taking before the switch to BIC/FTC/TAF. It can be seen that 64.4% of them were on a 3DR containing EVG/c.

We then analyzed the differences at baseline between those who came from a boosted regimen vs. those who came from an unboosted regimen.

Table 1 summarizes our findings.

Baseline	BOOSTER		*p* value
	No	*Yes*	
CD4+ T-cells (cells/µL)	657 ± 278	793 ± 348	0.088
CD8+ T cells (cells/µL)	660 ± 267	912 ± 454	**0.012**
CD4/CD8 ratio	1.16 ± 0.70	1.05 ± 0.59	0.476
Total cholesterol (mg/dL)	198 ± 46	207 ± 43	0.402
HDL (mg/dL)	53 ± 11	47 ± 10	**0.024**
LDL (mg/dL)	118 ± 42	133 ± 33	0.274
Triglycerides (mg/dL)	149 ± 123	152 ± 70	0.899
Weight (kg)	76 ± 15	73 ± 13	0.414
AST (UI/L)	30 ± 12	26 ± 7	**0.020**
ALT (UI/L)	35 ± 26	28 ± 16	0.096
Blood sugar (mg/dL)	91 ± 23	99 ± 49	0.460
Creatinine (mg/dL)	0.87 ± 0.18	0.84 ± 0.15	0.394
eGFR	95 ± 15	98 ± 18	0.454

Table 1. Differences at baseline between those coming from a boosted regimen and those coming from an unboosted one.

Figure 2 shows the CD4+ T-cell count trend in PLWH taking an unboosted (blue) vs. boosted (red) treatment before the switch to BIC/FTC/TAF.

It is possible to notice that, despite the count tends to improve in both the blue and red group, the blue group starts at a lower baseline value (even though it is not statistically significant, p = 0.088) and maintains lower values throughout the observation period.

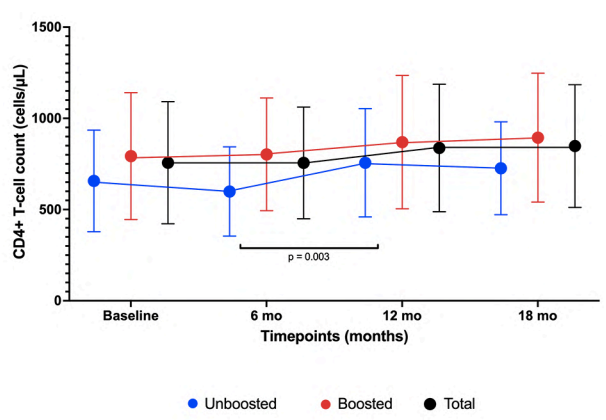

Figure 2. This figure shows the CD4+ T-cell count trend in PLWH switching to BIC/FTC/TAF from either a boosted (red) or an unboosted (blue) regimen. The black line shows the overall trend.

Although the CD4+ T-cell count is higher in boosted PLWH than in unboosted ones, figure 3 shows that CD4+/CD8+ ratio is higher in PLWH coming from an unboosted treatment, although the values are not significantly different.

This seems to suggest that boosted regimens negatively influence the status of the immune system, not being able to completely tame the inflammatory stimulus caused by the HIV infection. This fact is also supported by the significantly higher value of CD8+ T-cells in boosted treatments vs. unboosted treatments at baseline (p = 0.012).

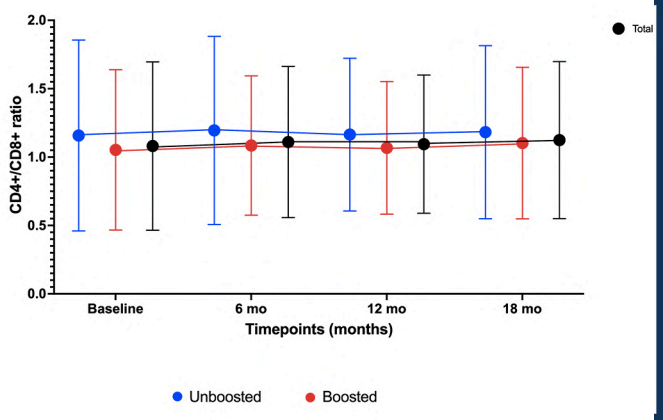

Figure 3. CD4+/CD8+ ratio trend in PLWH switching to BIC/FTC/TAF either from an unboosted regimen (blue) or a boosted one (red). The black line represents the overall trend.

Figure 4 shows that, although the starting values are not significantly different at baseline (p = 0.402), total cholesterol in patients coming from a boosted regimen significantly decrease at 12 months (p < 0.001) compared with baseline.

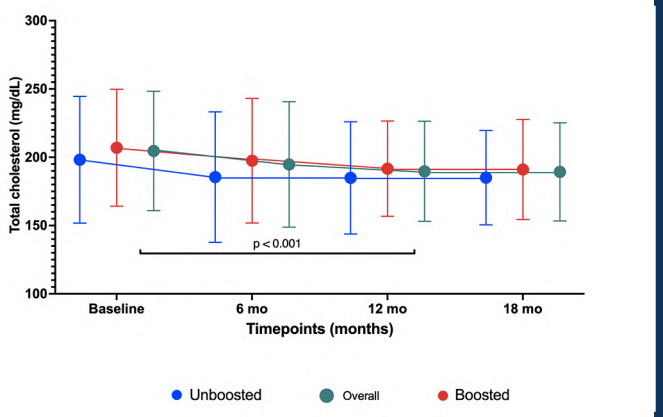

Figure 4. Total cholesterol trend in PLWH switching to BIC/FTC/TAF either from an unboosted (blue) or a boosted (red) regimen. The green line represents total cholesterol trend in the overall study population.

CONCLUSIONS

Our data show that switching from a boosted regimen to bictegravir/emtricitabine/tenofovir alafenamide is safe and advisable, especially for the effects on total cholesterol.

Moreover, our data seem to support the idea that boosted regimens negatively influence the status of the immune system, not being able to completely tame the inflammatory stimulus caused by the HIV infection.

FOR ANY QUESTION, PLEASE WRITE TO: manuela.ceccarelli@unict.it